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188th Meeting

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UNITED STATES NUCLEAR REGULATORY COMMISSION'S
ADVISORY COMMITTEE ON NUCLEAR WASTE & MATERIALS

April 9, 2008

The contents of this transcript of the proceeding of the United States Nuclear Regulatory Commission Advisory Committee on Nuclear Waste & Materials, taken on April 9, 2008, as reported herein, is a record of the discussions recorded at the meeting held on the above date.

This transcript has not been reviewed, corrected and edited and it may contain inaccuracies.

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UNITED STATES OF AMERICA
NUCLEAR REGULATORY COMMISSION

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188TH MEETING

ADVISORY COMMITTEE ON NUCLEAR WASTE AND MATERIALS

(ACNW&M)

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WEDNESDAY

APRIL 9TH, 2008

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ROCKVILLE, MARYLAND

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The Advisory Committee met at the Nuclear
Regulatory Commission, Two White Flint North, Room
T2B3, 11545 Rockville Pike, at 8:30 a.m., Dr. Michael
Ryan, Chairman, presiding.

COMMITTEE MEMBERS:

MICHAEL T. RYAN, Chairman

ALLEN G. CROFT, Vice-Chairman

JAMES H. CLARKE, Member

RUTH F. WEINER, Member

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PANEL MEMBERS PRESENT:

MARY HELEN BARCELLOS-HOFF,

Lawrence Berkeley Laboratory

BERNARD LE GUEN, Electricite de France

JAMES K. HAMMITT,

Harvard School of Public Health

VINCENT HOLAHAN, NRC RES

CHARLES LAND, National Cancer Institute

KENNETH MOSSMAN, AZ State Laboratory

JEROME PUSKIN, EPA

THOMAS TENFORDE, NCRD

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I-N-D-E-X

<u>WITNESSES:</u>	<u>PAGE</u>
JAMES HAMMITT	6
JEROME PUSKIN	43
VINCENT HOLAHAN	76

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

8:38 a.m.

1
2
3 CHAIRMAN RYAN: I'll go ahead and get
4 started, please, so the meeting will come to order.
5 This is the second day of the 188th meeting of the
6 Advisory Committee on Nuclear Waste and Materials.
7 During today's meeting, the Committee will continue
8 with the working group on the effects of low radiation
9 doses. At the end of the day the Committee will
10 consider and discuss ACNNW letter reports on other
11 topics.

12 This meeting is being conducted in
13 accordance with the provisions of the Federal Advisory
14 Committee Act. Neil Coleman is the designated federal
15 official for today's session. We have received no
16 written comments or requests for time to make oral
17 statements from members of the public regarding
18 today's sessions. Should anyone wish to address the
19 Committee, please, make your wishes known to one of
20 the Committee staff.

21 I believe we have the bridge line open,
22 Mr. Brown? So the bridge line is open if callers want
23 to call in. We'll have them announce as they arrive.

24 It's requested that speakers use one of
25 the microphones, identify themselves, and speak with

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1 sufficient clarity and volume so they can be readily
2 heard. It's also requested that if you have cell
3 phones or pagers that you kindly turn them off at this
4 time.

5 Feedback forms are available at the back
6 of the room for anyone who would like to provide us
7 with his or comments about the meeting.

8 Thank you all very much.

9 Our session today will build on the
10 activities that we had yesterday. We have three
11 presentations schedule. One, first, by Professor
12 James Hammitt, from the Harvard School of Public
13 Health, on an economic perspective on regulatory
14 decision making, benefit versus cost on the linear and
15 nonlinear models. We're interested in that topic.

16 Dr. Jerry Puskin, from the United States
17 Environmental Protection Agency, will give the U.S.
18 EPA perspectives. And Dr. Vince Holahan, from the
19 U.S. Nuclear Regulatory Commission staff, will off the
20 NRC staff perspectives. That will be the morning
21 session.

22 We will have a lunch break and then a
23 panel discussion among all participants from both days
24 for a time and then some time is allotted for any
25 stakeholder's views, comments, or perspectives that

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1 will be offered at the end of the day. Then we'll
2 close somewhere around 4:00.

3 So, without further ado, let me turn the
4 microphone over to you, Professor Hammitt. Welcome
5 and thanks for being with us.

6 PROFESSOR HAMMITT: Thank you.

7 CHAIRMAN RYAN: I guess we can get you
8 right up front.

9 PROFESSOR HAMMITT: Up here?

10 CHAIRMAN RYAN: Yes, that's fine.

11 PROFESSOR HAMMITT: I'm glad to be here
12 and disappointed to have missed yesterday's
13 discussions. I was hoping to learn a lot from that.

14 So what I'm going to do today is talk
15 about sort of an introduction, and for many of you a
16 review, of the basic economic perspective on decision
17 making with regard to risks. And then I'm going to
18 illustrate with several contexts for the discussion,
19 building up from the very simple case where we're
20 making decisions for a single individual and we know
21 the exposure response function to the more complicated
22 situations where we're making decisions for a
23 population and we don't know the exposure response
24 function, which is, of course, more realistic, and
25 then illustrate with a simple example involving radon

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1 and drinking water.

2 The objective of economic decision making,
3 our economics assumes the objective of decision making
4 is to maximize well being, and individual well being
5 depends, of course, on health, but on other things we
6 care about, education, housing, food, entertainment,
7 many others. The objective from an economic
8 perspective in setting exposure level, for example to
9 radiation or something else, is both to minimize the
10 harm and/or maximize health benefit and also to
11 minimize control costs.

12 So this requires inherently that we're
13 making tradeoffs between smaller risk of harm and
14 greater control costs so you have to face up to the
15 tradeoff of what incremental control costs justifies
16 what level of reduction in health risks. You have to
17 compare the benefits of better health to lower health
18 risks with the costs of control.

19 And the way this is done is to put a
20 monetary value on risk production or health
21 improvement, and that monetary value is often
22 described as willingness to pay for the improvement
23 and it is defined as if somebody pays money to have a
24 smaller health risk, that's money he could have
25 otherwise used for other purposes so he's foregoing

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1 other things he cares about, that are housing or
2 whatever, and so the maximum value of those foregone
3 alternatives is the willingness to pay for the health
4 improvement.

5 In choosing regulation for population, the
6 general framework is to try and maximize the sum of
7 benefits minus costs where the health benefit can be
8 calculated as the product of the number of people
9 affected by the regulation times their average
10 willingness to pay for the individual risk reduction
11 each faces. And often this is done in a short hand of
12 the expected reduction in the number of cases of
13 cancer or premature fatality multiplied by the value
14 per statistical case.

15 So if willingness to pay is proportional
16 to the reduction in the probability of harm, as it
17 should be under most theories, then you can have
18 either many people paying a small amount for a small
19 risk reduction or you can -- mathematically that's the
20 same thing as a value for each case avoided times a
21 large value for each case.

22 What I'm going to do just to focus ideas
23 is focus mostly on the contrast between a linear
24 no-threshold model and hormetic dose response exposure
25 response function. And the thing that's really

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1 critical here is if, as in the usual case, we have
2 data on exposures at some relatively high level and we
3 measure harm or probability of harm at that relatively
4 high level, over on the right hand side of the screen,
5 and we know that at no exposure there would be no
6 harm. So we have an interpolation problem, but we
7 can't observe harm or probability of harm in the range
8 we care about.

9 And then on the hormetic function, I want
10 to define two points, what I call e_0 . e_0 is the
11 exposure level where there's zero effect or the same
12 health effect as there would be at zero exposure. And
13 then e_M is the exposure level at which the health
14 effect is minimized. And then of course a threshold
15 exposure response function could be very similar to
16 this hormetic line over this range and then simply
17 flat over this interval.

18 But what I wanted to say is, if this is
19 the case where we observe harm at this relatively high
20 exposure level, are interpolating down to 00, then it
21 must be the case for the hormetic exposure response
22 function or a threshold response function the exposure
23 response function is steeper in some range of
24 exposures than the linear, and, of course, flatter
25 than the linear in other ranges of exposure.

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1 So now the optimal exposure for an
2 individual in the very simple case where we know
3 exactly the exposure response functions just to fix
4 ideas. In the linear case, I have in mind e_v as the
5 uncontrolled exposure level. So at this level there
6 is no control costs because we're doing nothing to
7 control exposure, and there is some harm or
8 probability of harm, and I'm measuring this in
9 monetary units.

10 If we think of reducing exposure, the
11 costs of control will rise and typically rise at an
12 increase rate of the convex function of the exposure
13 reduction, and the harm or probability of harm will
14 fall at a linear rate under this linear model. So
15 what we want to do is minimize the sum of control
16 costs and expected harm, that's this line, and the
17 exposure level that does that is what I've called e_L^* ,
18 which is the minimum of this curved line.

19 With the hormetic exposure as Fonda's
20 function, the analysis is the same. It's the same
21 cost function, a different exposure response function.
22 If you sum those and find the minimum cost plus health
23 harm point, it's this level e_H^* . And then if I
24 combine those two graphs just for comparison, you can
25 see the optimal control level is different under the

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1 two exposure response functions, logically enough.

2 In this case, the linear no-threshold
3 model suggests more exposure reduction, a lower
4 optimal exposure level than the hormetic response
5 function, but that doesn't follow necessarily. It's
6 just true in this illustration.

7 Another way to do this analysis is to
8 think in terms of marginal or incremental benefits,
9 meaning incremental reduction in health risk and
10 increment cost. But here, again, now I have this
11 marginal, think of derivative. The comments always
12 say marginal when they mean incremental or derivative
13 or slope, marginal harm, marginal cost and exposure.

14 So starting at the uncontrolled exposure
15 level again, there is zero cost of control, and
16 because the cost function was becoming increasingly
17 steep as we reduced exposure more and more, the
18 incremental cost of more stringent control is rising.
19 And in a linear model, the incremental benefit of
20 reducing exposure is constant. The linear exposure
21 response function has a constant slope.

22 So if you start out here at the
23 uncontrolled level, the incremental benefit from
24 reducing exposure a little bit is much larger than the
25 incremental cost. So it would be a good idea to

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1 reduce exposure until you get to some point where
2 they're about equal or exactly equal. If you go
3 beyond that point, the incremental cost incurred
4 through more stringent control exceeds the incremental
5 benefit in terms of reduced risk, and so that would be
6 excessive control. So, again, the way to identify
7 this optimal exposure level is where the marginal
8 benefit and marginal cost curves intersect.

9 Same analysis for the hormetic response
10 function. And here, you see this is higher than in
11 the linear case because, remember, at the high
12 exposure levels the exposure response function has to
13 be steeper than the linear curve. At some point, I
14 guess this is what I called e_m before, the slope of
15 the hormetic exposure response function is zero. So
16 the marginal benefit of incrementally reducing
17 exposure around this level is about zero. Down in
18 this region, this is where the exposure response is
19 downward sloping. So reductions in exposure would be
20 harmful in a health perspective.

21 And so the optimal exposure levels where
22 marginal benefit and marginal cost intersect here, and
23 put these together on the same graph, and, again, you
24 see e_H at a higher exposure level than e_L^* . For this
25 example, if I keep the exposure response functions

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1 exactly as shown here, but assume the cost and control
2 and the marginal costs and control are higher, this
3 dash line, now the optimal control level under the
4 linear model is here, e_L^* , the optimal exposure level
5 under the hormetic model is here, e_{H1}^* , and so you see
6 the hormetic response function calls for more
7 stringent regulation, larger exposure reductions than
8 the linear model and that is because this is a
9 situation where the incremental costs of control are
10 pretty high so it's only worth controlling a little
11 more when the incremental benefits are pretty.

12 And in this high exposure region the
13 incremental benefits control are steeper under the
14 hormetic than the linear model because the hormetic
15 exposure response function, and similarly a threshold
16 response function, are steeper at these high exposure
17 levels.

18 CHAIRMAN RYAN: Just a second, Dr.
19 Hammitt. My apologizes for interrupting, but we need
20 to announce the caller.

21 Could the caller identify who you are,
22 please?

23 MR. EHRLE: Lynn Howard Ehrle.

24 CHAIRMAN RYAN: I'm sorry. Say again?

25 MR. EHRLE: Lynn Howard Ehrle.

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1 CHAIRMAN RYAN: Good morning. Thanks for
2 joining us. Dr. Hammitt?

3 PROFESSOR HAMMITT: Thank you. So now
4 let's go to a slightly harder and slightly more
5 realistic problem. A decision again for an
6 individual, but we don't know exactly what the
7 exposure response function is. And here the standard
8 economic decision theoretic perspective would be to
9 assign probabilities to the different possible truths
10 about what the exposure response function is, and then
11 use that to calculate expected harm, so the harm
12 conditional -- here, let's assume the exposure
13 response function might be either the linear or the
14 specific hormetic function I showed in the previous
15 graphs, we think there's a probability p that the
16 linear model is most accurate. A complimentary
17 probability, the hormetic model, is most appropriate.

18 The expected harm is just p times the harm
19 if the linear model is right, plus $1 - p$ times the
20 harm fits the hormetic model is right. Obviously,
21 estimating these probabilities is not easy, but,
22 conceptually, this is what one would want to do and
23 there are practical methods for estimating these kinds
24 of probabilities.

25 The expected marginal benefit is just p

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1 times the expected marginal benefit in the linear
2 case, and so forth. And then the optimal exposure
3 level will be between the linear and hormetic
4 solutions. It's going to be some sort of a weighted
5 average of the two. The weight obviously depends on
6 what the probabilities are assigned to the two
7 exposure models and, also, the marginal harms of the
8 alternative models.

9 So here is the graph I already showed with
10 the marginal benefit of exposure reduction under the
11 hormetic and linear models, the marginal costs, and
12 the optimal exposure levels conditional on each model
13 being accurate. This line, now, is the expected
14 marginal harm in the case where we assign probability
15 0.3 to the linear model being correct and probability
16 0.7 to the hormetic model being correct.

17 So this line is always between the two and
18 it'll be roughly twice as far from the linear model as
19 the hormetic model for this value of p . And so the
20 point where the expected marginal benefits are equal
21 to the marginal costs is e^* between the two models,
22 the two exposure levels that are optimal in the case
23 where we know exposure response function for sure.

24 So as that last graph shows, what's really
25 critical is the slope of the exposure response

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1 function, the marginal benefit, the marginal health
2 risk reduction associated with reducing exposure. So
3 the question is, how similar is the slope of either a
4 threshold or a hormetic exposure response function to
5 the linear model?

6 Well, we don't know in general, but one
7 thing we can say is that think of the average slope of
8 the hormetic exposure response function -- I mean
9 threshold function between the uncontrolled level and
10 this level e_0 , which is either the threshold or the
11 level at which there is no harm under the hormetic
12 model. And the average slope of the hormetic function
13 will be equal to the slope of the linear model divided
14 by this number.

15 So think about if e_0 is very, very small
16 compared with the uncontrolled level e_u , this fraction
17 is close to zero, so we're dividing by something close
18 to one, so the average slopes will be roughly equal.
19 And in that situation, uncertainty about whether
20 there's a threshold or not doesn't really matter
21 because it doesn't affect the slope of the exposure
22 response function in the region that may be condition
23 on costs being high enough such that the optimal
24 control level is in this region higher than e_0 .

25 Contrast if e_0 is pretty large, compared

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1 with the uncontrolled level, this fraction can never
2 be bigger than one, but it could approach one. And so
3 we would be dividing by one minus something close to
4 one, and so the average slope of the hormetic response
5 function would be much steeper than of the linear
6 response function. And then it might, uncertain about
7 which exposure response function is accurate, could
8 have a big effect on the implied optimal degree of
9 exposure.

10 And then, of course, if the exposure is
11 smaller than e_0 , then with a threshold case we're on
12 the flat of the curve; with a hormetic case we may be
13 in an area where reducing exposure is even harmful to
14 people. In that region, knowing which exposure
15 response function is accurate is clearly critical to
16 knowing what exposure level is appropriate.

17 So the real problem we have is a
18 population level decision where both the exposure
19 levels and the exposure response functions may differ
20 between individuals. Also, they are uncertain. We
21 don't know exactly the exposure response function. We
22 don't know exactly any individual's exposure.

23 And one implication of this is we can't
24 write a rule that will ensure the optimal exposure for
25 every person. Now, the social choice problem of

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1 balancing benefits to some people against harms are
2 foregone benefits that we could have provided to other
3 people instead.

4 Under a standard economic perspective,
5 economists assume there is no objective way to compare
6 changes in well being between people, so we can't say
7 objectively who suffers more from a certain disease
8 or, you know, who bears more pain. So the kind of
9 minimal idea that's accepted is the idea of Pareto
10 improvement. If we can have a policy change that
11 helps some people and hurts no people, that's defined
12 as a Pareto improvement and we, more or less, all
13 agree that that's a good thing.

14 The caveat there would be it could
15 increase inequality. So something that improves the
16 well being of the very wealthiest, something that
17 improves the well being of Bill Gates had has no
18 effect on anybody else in the country would count as
19 a Pareto improvement even though lots of people in the
20 country might think that's a bad thing socially.

21 (Laughter.)

22 So that doesn't get us far. We're rarely
23 in a situation where we can help some people and at
24 least forego helping others instead.

25 So benefit cost analysis tries to identify

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1 what are called potential Pareto improvements. And a
2 potential Pareto improvement is defined as a situation
3 where the people who benefit benefit enough such that
4 they could, in principle, pay monetary compensation to
5 the people who are harmed. And after the compensation
6 was paid everybody would consider themselves better
7 off with the policy change and the compensation paid
8 or received then without.

9 And so we talk about the Kaldor-Hicks
10 compensation test as just the test for whether a
11 change is a potential Pareto improvement, and the way
12 this is done is you add the monetary value of the
13 benefits across the people who benefit from a change,
14 add the monetary value of the harms across the people
15 who are harmed; if total benefits exceed total costs,
16 then, in principle, compensation could be paid such
17 that everybody would perceive themselves as being better
18 off. So that's the logic behind the benefit cost
19 test.

20 Why is that a reasonable thing to do when
21 this compensation is purely hypothetical; we're not
22 suggesting it be paid? Well, there are two arguments.

23 One argument is that if we make many
24 decisions over time using principles like this, the
25 people who gain in each particular case will not be

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1 the same. And so, in the long run, we, as a society,
2 will all be better off making decisions on this basis
3 rather than some other basis. And there's hand
4 waving here because what is the alternative basis on
5 which we'd make these decisions? It's not clear.

6 One thing to say is benefit cost analysis
7 at least counts the preferences of everybody in the
8 population. So, in that sense, it's more populous and
9 egalitarian than something where just some elite
10 decides or the classic politicians in the smoke-filled
11 room decide in their own interests.

12 A better argument, I think, is that
13 redistribution of resources can be handled more
14 efficiently, more directly through means other than
15 setting health regulations at a non-optimal level,
16 things like tax programs, social transfers, and the
17 like.

18 What I want to say here is, in calculating
19 the population effect of some reduction in exposure,
20 under the linear no-threshold model, we don't have to
21 know anything about anybody's background exposure
22 level because the incremental benefit of reducing
23 exposure is the same regardless of the exposure level
24 at which one starts. We know if we reduce everybody's
25 exposure by x , everybody will get the same incremental

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1 benefit.

2 Under the hormetic model, because the
3 slope depends on your total exposure level, it's much
4 more complicated. People who are at high exposure, if
5 we reduce their exposure a little bit, will benefit.
6 People who are at very low exposure, if we reduce
7 their exposure, will either not benefit; conceivably,
8 they will even be harmed. So we need to know how the
9 exposure reduction correlates with the baseline
10 exposure across the population.

11 Let me illustrate now with an example,
12 just very simplified, doing violence to lots of
13 detail. But I developed this example because there
14 was a regulatory assessment published, a draft
15 regulatory assessment, published by EPA associated
16 with regulating radon in drinking water. And here, as
17 I'm sure probably all of you know, the primary
18 exposure pathway is that radon volatilizes from the
19 water into the air and is then inhaled. That's a more
20 important exposure source than drinking the water
21 apparently. And then this was a good example for me
22 because Ken Bogen had published a couple of articles
23 in which he estimated hormetic exposure response
24 functions for radon and air and the risk of lung
25 cancer.

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1 So here the policy alternatives EPA was
2 considering was to set a maximum contaminant level, or
3 MCL, for community water systems. To estimate the
4 benefits of different MCLs, what they did is estimated
5 the distribution of radon levels in drinking water,
6 calculate the reduction in radon in drinking water as
7 a function of whichever MCL they chose, and then they
8 estimated the change in indoor air concentration as
9 10,000-fold smaller than the change in water
10 concentration based on models and measurements of
11 how, essentially, the effect of drinking water
12 volatilizing into the air and then being breathed in.

13 So in this table, what I'm showing here is
14 potential maximum contaminant levels and pCi/l, 4,000,
15 2,0000, all the way down to zero. The population of
16 people service by water systems with radon levels
17 higher than each threshold, so 77,000 people, have
18 drinking water with higher than 4,0000 pCi/l.

19 The population average concentration of
20 radon is something higher than 4,000. I made up this
21 5,000 actually. But what this table shows you is that
22 average radon concentration for the people above each
23 concentration level. So you see, for the people above
24 the highest concentration level, the average radon
25 concentration is quite high. For the people with any

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1 radon in their water, the average concentration is
2 very low because most of the people have very low
3 radon concentration in their water.

4 And then this shows the incremental
5 reduction on average radon concentration water as a
6 function of the MCL chosen. So for these high MCLs,
7 there's a big reduction in exposure to the small
8 number of people affected. For the lower MCLs,
9 there's an, on average, small reduction, but applying
10 to many, many more people. That is just obviously the
11 distribution of radon drinking water is highly skewed.

12 This illustrates a graph from one of Ken
13 Bogen's papers where this is his estimate of a
14 hormetic exposure response function. I've
15 superimposed his threshold exposure response function
16 on that, and this is linear exposure response function
17 with which he compared.

18 You see here the lowest point on the
19 hormetic function is at a level of about 5 pCi/l.
20 This is indoor air concentration now. It's a relative
21 risk of lung cancer.

22 Now it turns out that only five percent of
23 household levels have radon levels indoor exceeding
24 the EPA action level of 4 pCi/l. Distribution of
25 radon in indoor air, residentially, is roughly

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1 lognormal, geometric mean, geometric standard
2 deviation, 98th percentile six-and-a-half. So almost
3 everybody is in this region where their exposure level
4 is close to 5, maybe even below 5. So under that
5 specific hormetic exposure response function reducing
6 exposure would be reducing a beneficial effect to
7 these people.

8 And under the threshold function, reducing
9 exposure would have no benefits to these people. So
10 that, of course, makes the policy decision very simple
11 if we believe either of those exposure response
12 functions that no regulation would be justified
13 because we're doing essentially no benefit and
14 incurring costs.

15 So to make a more interesting problem I
16 imagined some community with very high background
17 radon in their air and, specifically, I'm assuming 25
18 percent of the people have only 2 pCi/l, 25 percent
19 have 5, 25 percent have 10, 25 percent have 15. And
20 then relative slope of the hormetic exposure response
21 function relative to that for the linear no-threshold
22 model is for people at roughly the 5 exposure level,
23 the hormetic function is flat, zero slope. People at
24 lower exposure have a negative slope, so reducing
25 their exposure would be harmful. And then for people

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1 at high exposure, this function is steeper than the
2 linear model, and the average is about 2. So on
3 average where just seeing exposure to this population
4 would help some people a lot, have no effect on
5 others, hurt some people some, on average the total
6 risk reduction would be twice as large as it would be
7 under the linear model.

8 Then here I'm plotting -- should have
9 reversed the X-axis on this -- but here, going from
10 left to right, is increasing regulatory stringency
11 reducing the MCL and the black curve is the costs.
12 These increase at an increasing rate as expected. It
13 turns out here the benefits under the linear model,
14 the blue, and under the threshold model, the green,
15 are almost exactly equal and that comes about, I guess
16 you can see it here, under the threshold model this
17 -2.8 becomes a zero. So we're averaging 001.8 and 2.4
18 and the average of that is pretty close to 1 it turns
19 out. And so that's why we get the linear no-threshold
20 and the threshold model having roughly equal benefits
21 of exposure reduction in this case.

22 Under the hormetic model, we have lower
23 benefits because reducing exposure helps some people,
24 but is harming others. So on that it's doing less
25 good.

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1 This is here the total benefits under each
2 model and the net benefits, benefits minus costs. And
3 then I've highlighted the optimal control levels under
4 the different models. Under the linear no-threshold
5 model, a 1,000 pCi/l would be the optimal MCL. Under
6 the hormetic function is a little bit less stringent.
7 Under the threshold it happens to be a little more
8 stringent.

9 Obviously, there's some kind of jumpiness
10 in this because I just have different increments of
11 control level. You'd want to do this better by having
12 a more continuous function of the MCL.

13 Now, to deal with uncertainty about which
14 exposure response function is correct, I said before
15 what we want to do is calculate the expected benefits
16 as the sum of the probability that each exposure
17 response function is accurate times the harm if that
18 response function is accurate. So here, for example,
19 I'll put probability 0.6 on the linear model,
20 probability 0.4 on the hormetic model, and probability
21 zero on the threshold.

22 And there, again, we have total benefits,
23 benefits minus costs under each model, so the linear
24 no-threshold and the hormetic are the same as in that
25 pervious charge, and then the expected benefits, the

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1 weighted average of these two, is here, and turns out
2 in this case the optimal regulation would be the same
3 as under the hormetic, the 2,000 pCi/l plus stringent
4 and under the linear.

5 So just to conclude, the first point is
6 economic evaluation can accommodate non-linear
7 exposure response functions. There's no difficulty in
8 principle. It's harder in practice because the
9 incremental benefit of reducing exposure depends on
10 the background exposure level of the people whose
11 exposure is reduced. So you have to know the
12 co-variation of background exposure and exposure
13 reduction due to the regulation. Whereas, under the
14 linear model, you don't need to know that.

15 Uncertainty about exposure response
16 functions can be accommodated in principle by saying
17 any of these might be true, and we assign
18 probabilities which are a numerical statement of
19 degree of belief in the truth of the model in this
20 case to each and calculate the expected benefits.

21 So in a way that's just a generalization.
22 When we say, you know, there's a risk of getting lung
23 cancer from radon or something, in fact, an individual
24 will either get lung cancer from radon or will not.
25 So already we're dealing with that probability. And

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1 at the individual level maybe this is stochastic,
2 maybe this is deterministic, who knows.

3 When we back up a level and say, well,
4 we're not sure exactly what the slope of the exposure
5 response function is or even what the shape of it is,
6 that's just kind of another level of uncertainty that
7 we can assign probabilities to the different potential
8 outcomes and aggregate in that way.

9 And then, finally, the last point is while
10 many people think that threshold and hormetic exposure
11 response functions necessarily imply that less
12 stringent regulation is appropriate than the linear
13 model, if decisions are made on the basis of
14 maximizing benefits minus costs, that is not
15 necessarily true because these alternative anomaly
16 models will tend to be steeper in some parts of the
17 exposure region than a linear model. And in that
18 region it will be appropriate to reduce exposure more
19 than would be appropriate under the linear model.

20 Thank you.

21 CHAIRMAN RYAN: Thank you. Any questions
22 or comments from the panel members?

23 (No response.)

24 DR. TENFORDE: May I ask, do you have any
25 opinions about the Cohen research on radon where he

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1 did county by county modeling of concentrations and
2 concluded there was some apparent hormetic effect?

3 PROFESSOR HAMMITT: Yes. No, I don't want
4 to put myself forward as having any great experience
5 in the epidemiology or the estimation of these
6 exposure response functions. My interest here was in
7 showing if you know or if you thought you knew what
8 the exposure response function was, what you would do
9 with that in terms of decision making.

10 MR. MOSSMAN: Dr. Hammitt, you mentioned
11 with the LNT theory that you really didn't have to
12 know the total background exposure. It was
13 incremental exposure that was important. And I'm
14 assuming that that's based on your assumption that the
15 origin 00 is a measured point and that you were
16 interpolating. But, in fact, we don't know what 00
17 is, and the reason why we don't know 00 is because we
18 don't know the proportion of cancer incidents or
19 cancer mortality that's attributable to natural
20 background and natural background radiation is
21 irreducible.

22 So, in fact, whatever you add, and
23 particularly when you get at very, very small doses
24 where the incremental dose is some significant
25 percentage of the natural background, becomes very

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1 difficult to identify what that value is. I
2 understand what you are doing, but it may be a picky
3 technical point, but the idea is is that 00 is not a
4 truly measured point because you can't eliminate
5 natural background to determine what actually is the
6 cancer rate in the absence of radiation altogether.

7 PROFESSOR HAMMITT: Yes, but can we handle
8 that by defining my axes as the origin is the natural
9 exposure and cancer rate given the natural background
10 exposure? And then I'm just talking about increasing
11 the exposure of both natural background and increases
12 in cancer risks above what it would be at the natural
13 background.

14 MR. MOSSMAN: I suppose you could do that,
15 but it doesn't completely eliminate the fundamental
16 problem of understanding what the cancer rate is in
17 the absence of radiation.

18 PROFESSOR HAMMITT: Right, right.

19 MR. MOSSMAN: I mean when we talk about --
20 you know, frequently LNT is interpreted when I look at
21 zero, I'm looking at the cancer rate in the absence of
22 radiation, when, in fact, you're not. You're looking
23 at cancer rate in the presence of whatever the natural
24 background rate is.

25 PROFESSOR HAMMITT: Right. And also in

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1 the presence of many other things that cause cancer.

2 MR. MOSSMAN: Right. And other things,
3 right. But for smoking and other kinds of things, you
4 can --

5 PROFESSOR HAMMITT: You can eliminate
6 exposure.

7 MR. MOSSMAN: You can account for that.

8 PROFESSOR HAMMITT: Right, right.

9 MR. LE GUEN: This is a question about all
10 compounding factors that you can have.

11 PROFESSOR HAMMITT: Yes. So doing the
12 epidemiology and estimating these things is very
13 difficult, I agree.

14 MR. LE GUEN: Yes.

15 MR. EHRLE: Mr. Chairman, I have a
16 question for the doctor.

17 CHAIRMAN RYAN: Okay.

18 MR. EHRLE: And it is for the whole
19 Committee. Why has this conference omitted a model
20 that has been written about since 1990 and identified
21 in Gofman's impressive book on low dose radiation that
22 was compared favorably with BEIR V, and that is the
23 super linear model. Ken Mossman skipped right over it
24 in his delineation and citing of several models. He
25 omitted it. And now the conference has elevated the

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1 hormesis thesis to the same level as LNT and it's been
2 subjected to numerous --

3 CHAIRMAN RYAN: Mr. Ehrle, that's a
4 comment, not a question. Do you have a question?

5 MR. EHRLE: The question is, is there any
6 way that you can deal with, objectively, the super
7 linear or biphasic model?

8 CHAIRMAN RYAN: Okay. Does anybody want
9 to answer that question?

10 PROFESSOR HAMMITT: I would say that in
11 terms of economic analysis that can certainly be
12 accommodated just like any other non-linear exposure
13 response function. If you have a function and if
14 you're willing to give some probability that it's
15 valid, you would calculate the marginal benefits of
16 exposure reduction under that function just as per all
17 the other non-linear functions I showed.

18 MR. EHRLE: The reason I raise the
19 issue --

20 CHAIRMAN RYAN: Mr. Ehrle --

21 MR. EHRLE: -- an opportunity to hear Tom
22 Hay from Columbia who made this presentation at Mayo
23 Clinic --

24 CHAIRMAN RYAN: Mr. Ehrle?

25 MR. EHRLE: -- to be up there --

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1 CHAIRMAN RYAN: Mr. Ehrle?

2 MR. EHRLE: Yes.

3 CHAIRMAN RYAN: I'm sorry, but I'm going
4 to have to ask you to hold your comments until the
5 comment period later on, if you don't mind?

6 MR. EHRLE: Well, I doubt if I'll be here
7 at that comment that and that's why I appreciate the
8 opportunities to submit this query.

9 CHAIRMAN RYAN: Now is not the best time.
10 If we have some time later in the morning, I'll
11 certainly give you that time to make comments. But we
12 need to press on to other questions.

13 MR. EHRLE: Okay. Thank you.

14 CHAIRMAN RYAN: Dr. Weiner, have you got
15 a question?

16 DR. WEINER: Thank you. First, a comment.
17 I don't know if you're aware of there is a recent
18 paper by Thompson et. al. in I believe it's the next
19 to last issue of *Health Physics* where he actually
20 demonstrates the hormetic effect. It would be
21 interesting to compare your thing.

22 PROFESSOR HAMMITT: Yes.

23 DR. WEINER: But my question is, how does
24 the notion of perceived harm figure into this, and
25 when you have perceived harm, then the effect and the

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1 costs are no longer independent, or could be no longer
2 independent?

3 PROFESSOR HAMMITT: Yes. Well, that is I
4 think the central problem of health and environmental
5 decision making and decision making under uncertainty.
6 So from the economic perspective, well being is
7 defined and assessed by individuals. So you can't
8 tell me that in my preferences over health states and
9 health risks should be determinative in principle.

10 But there is huge amounts of evidence that
11 all of us don't understand probabilities very well,
12 make all kinds of inconsistent decisions in the face
13 of probability and risk. So some of those
14 inconsistencies are clearly just mistakes, and if you
15 point that out to me, I will say, you're right, I'm
16 making a mistake, I was confused, you know, framing
17 effects, things like that.

18 Some of them may not be mistakes, and
19 sorting out which is which is critical. So in terms
20 of -- I didn't really talk about this, but valuing
21 health risk, we talked about value per statistical
22 life and things like that. In principle, there's no
23 reason why I could not have, for myself, a different
24 value of statistical life or a different willingness
25 to pay for a probability reduction associated with

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1 different causes of death, you know, car crash,
2 radiation exposure, terrorist incident, all kinds of
3 things. There's nothing incoherent about that because
4 those ways of dying are different and that might
5 matter to me. I might be willing to spend more money
6 to reduce one risk than another.

7 But, because we're not very good at
8 dealing with probabilities and small probabilities and
9 numbers in general, when you do surveys of willingness
10 to pay and you ask maybe two different sets of people,
11 what would you pay to reduce your chance of dying this
12 year by 1:10,000, in a different group, what would you
13 pay by 2:10,000, in theory you should get numbers that
14 differ by a factor of 2 or very, very close to that.

15 Often you'll get numbers that differ by
16 not at all or by 1.3, or something like that. So if
17 you take those as valid responses, that says people
18 would be willing to pay something for a 1:10,000 risk
19 reduction but much less for another 1:10,000 risk
20 reduction.

21 Do people really believe that? I don't
22 think so. I think that's confusion.

23 Another version of that is we tend to like
24 the idea that we could eliminate a risk, we could
25 eliminate the risk of lung cancer from radiation,

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1 let's say. But given that we have faced many other
2 risks, why is it important to drive this one all the
3 way to zero as opposed to reducing some others more?

4 So I think it's very important to focus on
5 the probability of reduction and harm and reflect on
6 that and help people reflect on that and how much they
7 really care about these other attributes, whether it's
8 radiation or a car crash, or something else.

9 DR. WEINER: How do you extend that to a
10 population? Because if you looked at the Tengs report
11 of some years ago, the differing cost --

12 PROFESSOR HAMMITT: -- life saving?

13 DR. WEINER: Yes.

14 PROFESSOR HAMMITT: So I think, by and
15 large, because we're not good at dealing with numbers,
16 we often don't even know the numbers. We base our
17 judgments much more on the things we can understand,
18 things like perceived control ability and
19 voluntariness, and dread factor large in people's
20 judgments about risks. But if people reflect more, I
21 think those factors become less important and the
22 quantitative probability becomes more important.

23 DR. WEINER: Thank you.

24 CHAIRMAN RYAN: Dr. Clarke?

25 DR. CLARKE: Nothing at this time.

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1 CHAIRMAN RYAN: Dr. Land?

2 DR. LAND: I love this stuff that you're
3 giving. I was just wondering, how does it sell as a
4 way of influencing public opinion, public regulatory
5 behavior, and so forth? Is it accepted?

6 PROFESSOR HAMMITT: Well, yes and no. So
7 often when people learn a little bit about it, I mean
8 it's basically common sense, right? We're making
9 tradeoffs all the time whether we buy something, how
10 much do we think it will give us pleasure, or
11 whatever; what are we giving up by buying this instead
12 of something else? So that's easily accepted.

13 In the U.S. government you probably know
14 when many agencies write regulations they have to have
15 a formal regulatory impact assessment, a regulatory
16 assessment, which is basically doing this stuff.
17 That's required by executive orders going back a
18 couple of decades now.

19 There is certainly a community of
20 activists and of scholars who reject a lot of this,
21 but they don't, in my view, have any very compelling
22 way to tell us what to do, how to make decisions other
23 than this. They tend to talk about, well, let's have
24 more discussion and things like that, which, you know,
25 certainly could be helpful. I think it's pretty

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1 accepted, but, as you know better than I probably,
2 real decisions are based on many, many factors,
3 including some narrower political things. So how much
4 effect this really has is hard to know.

5 MR. MOSSMAN: It would seem to me that,
6 following up on Dr. Land's comment, that an important
7 consideration is this notion that we have the capacity
8 to do something. In other words, if you look at the
9 history of radon regulation, you know the 4 pCi/l,
10 where did that come from? It didn't come from a
11 systematic evaluation of risk. It was before that.

12 And where it really came from was from the
13 Colorado plateau and a determination of what was
14 technically feasible, what could we get down to and it
15 wouldn't cost an arm and a leg to do it. And so we
16 just select 4 pCi/l, and so now we're scrambling
17 around to be able to defend that in a scientific and
18 an epidemiologic sense, which is fine, but it was
19 always curious to me that that seems to be a major
20 driver.

21 Why, in waste management, are we always
22 trying to get down to zero? Because we've got the
23 technical capacity to do it. And, you know, that, to
24 me, is a major issue and it goes to the heart I think
25 of a lot of what you're talking about that sometimes

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1 these decisions are not done with any systematic,
2 rational kind of way that, you know, if we can do it,
3 then we ought to do it.

4 CHAIRMAN RYAN: Ken, there's another good
5 example if I could add to the question, and that is
6 that very often we regulate real dose, obviously, and
7 we also regulate the potential for a dose. Waste
8 management is a real good example where we're
9 regulating and setting requirements based on the
10 possibility of some dose to some people at some
11 distant future time without any realization of that
12 risk.

13 So could you talk a little bit about how
14 do you weight or value future risk versus real risk
15 today? I mean smoking and radon will be a real risk
16 today. Whereas, some of these other things where
17 there's a potential for a dose, a hundred, or a
18 thousand, or ten thousand or more years in the future,
19 we're weighing that as well.

20 PROFESSOR HAMMITT: Let me separate a
21 couple of things. The real risk from the possible
22 risk, to me there's not really any bright line there.
23 Everything can be quantified by probability, and while
24 you take as a real risk means you and the scientific
25 community think there's a very, very high probability

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1 close to one that this exposure may cause cancer under
2 these circumstances, or whatever. Or it's a possible
3 risk like the idea that there is stored waste and it
4 will only harm people conditional on getting out and
5 people getting exposed to it.

6 This is a little bit more complicated to
7 causal pathway. First, there has to be a release or
8 people have to get into the site, or something, and
9 then they might get exposed and then they might be
10 harmed. So there's no real conceptual difference
11 there I think that's important. The timing is -- so
12 the question if it's a current risk is, what will
13 people give up now in terms of foregone other benefits
14 to reduce this risk to them or to people now?

15 In the future risk, what will people give
16 up now in terms of reducing the risk to some future
17 generation maybe far, far off in the future? And that
18 I guess what economics could tell you is that in
19 thinking about that question, you should think of all
20 the things we can do that will affect the well being
21 of these future generations and how effective is
22 controlling radioactive waste relative to many other
23 things and let's weigh the whole portfolio of them.

24 In terms of how much we should care about
25 future people, economics probably doesn't have very

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1 much to say except most economists would sort of say,
2 well, treat people equally. The fact that this is
3 another generation has no real moral content relative
4 to it being the current generation. And so I'll leave
5 it at that.

6 It's sort of apropos Dr. Mossman's point.
7 I think the really critical thing the economic
8 perspective brings that we all know, but often
9 overlook, is that it's tradeoffs. You can always
10 reduce some risks more. Some risks you can even
11 eliminate. It's just by doing that you're spending
12 your time and your resources that you could have used
13 on other things that might have provided a larger
14 total gain in mortality risk reduction or other things
15 we care about.

16 DR. MOSSMAN: On that matter, if you look
17 at countervailing risks, in other words, I apply some
18 risk management strategy to the target risk, but at
19 the same time I'm now introducing some new, perhaps
20 unrelated risk. Is it simply a matter of again
21 probabilities and cost analysis, as you've gone
22 through, to include the possibility of a
23 countervailing risk?

24 PROFESSOR HAMMITT: Yes, I think it is and
25 that's an important point. The way our brains work,

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1 we kind of segment things and we identify some risk as
2 of concern and we forget about all the other risks we
3 could control and the countervailing risks. So if you
4 think of the precautionary principle, the
5 precautionary principle says when we're uncertain
6 about the harm, we should be more cautious about it.
7 So that's fine.

8 But what if actions to reduce this one
9 harm increase the risk of other harms? Being
10 precautionary against one entails, by necessity, being
11 less precautionary against the countervailing risk.
12 So which one do we take the precaution against?

13 CHAIRMAN RYAN: Anything else?

14 PROFESSOR HAMMITT: I think the only
15 answer to that is kind of tradeoffs. How much do you
16 think you're gaining in reducing one risk, increasing
17 another? Is it worth it?

18 CHAIRMAN RYAN: And I think the judgment
19 ultimately ends up on the certainty or uncertainty of
20 what you know, what you're think you know.

21 PROFESSOR HAMMITT: Right. Just caution,
22 I agree, but certainty and uncertainty are more of a
23 continuous variable than a discrete one to me.

24 CHAIRMAN RYAN: Sure. With that, we're on
25 the schedule for a short break until 9:45. So, Dr.

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1 Hammitt, thank you for being with us today and we'll
2 look forward to your participation for the rest of the
3 day. We'll take a short break and reconvene at 9:45.

4 (Whereupon, the foregoing matter
5 went off the record at 9:35 a.m.
6 and went back on the record at
7 9:50 a.m.)

8 CHAIRMAN RYAN: All right. If we could
9 come to order, please, we'll begin our next
10 presentation. Dr. Jerry Puskin from the Environmental
11 Protection Agency. Good morning.

12 DR. PUSKIN: My talk is entitled EPA
13 Perspective, but some of it of it's going to be my
14 perspective I guess based on the work I do, which
15 is --

16 MR. COCHRAN: This is Tom Cochran phoning
17 in. Thank you.

18 CHAIRMAN RYAN: Good morning, Tom.

19 DR. PUSKIN: -- assessing health risk from
20 ionizing radiation and I try to track all the
21 literature and epidemiology and the radiation biology
22 that bear on this. Let's go to the next.

23 The first slide is definitely EPA point of
24 view though, why we use LNTs.

25 (Laughter.)

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1 DR. PUSKIN: First very good reason is
2 it's the default assumption for EPA and for the
3 federal government generally that this is something
4 that is a carcinogen, that's clear, and it is also a
5 mutagen. So it's guidance for the agency, for I
6 believe IARC. It is OSTP guidance going back to the
7 Reagan administration. It says when something's a
8 mutagen and it's a carcinogen through that type of
9 mechanism, that use in linear no-threshold. Also,
10 that we have guidance from NCRP and ICRP and National
11 Academy that specifically ionizing radiation to use
12 LNT.

13 Well, right now, of course, we have to
14 have some sort of model for extrapolating because the
15 epidemiological studies have insufficient statistical
16 power to test LNT down at the low doses we're
17 interested in, which for EPA it's really usually your
18 near background levels. And so far the biological
19 research has not filled this gap, so we need to have
20 some sort of model for extrapolating, and, as I said,
21 we have this advice.

22 Now, I would particularly highlight the
23 last one that the National Academy has said that the
24 scientific weight of evidence still favors LNT.
25 Previous National Academy reports and NCRP reports,

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1 they always kind of hedge; they say, well, if LNT is
2 not inconsistent with the data or something like that.
3 In this report, since we're spending so much money on
4 it, we decided, well, we want more information now.
5 We're want to say, given all the -- we know how far
6 the epidemiology can take you, how far down it can
7 take you.

8 What we want to know is, in light of the
9 scientific evidence, what is the best way of
10 extrapolating risk? Not from a policy standpoint,
11 just, scientifically, in the judgment of this expert
12 committee, what is the best scientific evidence? And
13 they said, unequivocally, LNT. Now, that's a very
14 powerful reason to use it at this point until that
15 changes.

16 Scientific basis. First of all, both
17 animal and human data on cancer generally is
18 consistent with LNT. That is, as you reduce the dose,
19 the incidence of cancer goes down linearly, whether
20 you do animal studies or human studies, as far down as
21 you can go until the statistical power is gone. So
22 that's one reason.

23 Another is there is a scientific basis in
24 the idea that there's a mechanism that electrons cause
25 ionizations in the cell leading to damage of the DNA

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1 and that there's a good chance that some of this DNA
2 will not be repaired properly. And we also, knowing
3 the monoclonal origin of cancer, that a single
4 mutation in a cell will increase the probability the
5 cell will become malignant. Not that a single
6 mutation is sufficient, but that it's one step in a
7 process, but you increase the number of cells that can
8 be transformed.

9 Now, this is a picture from Dudley
10 Goodhead showing the pattern of ionizations. I'm
11 going to talk mostly about low LET radiation because
12 I think that's where the main interest is here and
13 there's even more evidence I think for LNT for high
14 LET.

15 But for low LET, while there's a -- on
16 average the ionizations are further apart. When you
17 get down to the ends of the electrons, as the
18 electrons slow down, they produce clusters of
19 ionization, and this is shown on a scale here, with
20 where you see it, the distant, how they're distributed
21 typically at the end of these tracks and with the same
22 scale the DNA molecule. And you can see that this can
23 produce rather complex damage: double strand breaks,
24 which you see there in red, or green will be single
25 strand breaks; then you can get base damages, that

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1 sort of thing. So it's often possible to get a double
2 strand break and -- or, two double strand breaks close
3 together, a double strand break and a base change,
4 this is not something that easily can happen with
5 chemicals.

6 So there's the fact that this damage can
7 be clustered creates much more complex damages, more
8 difficult to repair, and that's why a threshold is
9 very much less likely for ionized radiation. I know
10 Dr. Le Guen said yesterday that this type of damage
11 won't be repaired, cells just die, and I think in many
12 cases that would occur. But I think this is generally
13 thought to be the mechanism and I would say that for
14 low LET radiation a substantial fraction of the energy
15 is deposited at the ends of tracks like this.

16 What do we mean by a threshold? Normally,
17 I guess strictly speaking, a threshold's defined as
18 the radiation dose or dose rate below which you have
19 no harm to anybody, even the most sensitive individual
20 and the risk would be absolutely zero to everybody.

21 That's perhaps very unlikely. I'm going
22 to relax the definition here and talk about a
23 practical threshold, which means, really, just that
24 LNT -- below some level of dose LNT greatly
25 overestimates risk, that maybe there are just some

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1 sensitive people or maybe it's linear, but with a much
2 lower slope than what we would extrapolate based on
3 epidemiology. That might affect our regulations.

4 Or you might imagine that we could have
5 something like hormesis, that below some dose,
6 beneficial effects, you might still get some cancers
7 caused by radiation, but maybe the radiation prevents
8 more cancers than it causes or it prevents many more
9 heart attacks than it does cancers, or whatever, but
10 that the net health benefit might be beneficial.

11 Is there a low dose threshold?
12 Epidemiology is generally, generally sensitive down to
13 about 100 mGy low LET. People could argue a factor of
14 2 up or down from that based on the A-bomb survivor
15 data. You can't really get much lower than that
16 because the risk is just too small and you don't ever
17 have enough people.

18 Well, you can recognize that from natural
19 background radiation you get, over a life time, about
20 75 mGy of low LET radiation, and we get additional
21 exposures from medical and so for. So in terms of
22 life time dose, there's really not much of an
23 extrapolation. It's just 100 mGy that -- if we get
24 75 mGy from natural background and we know there's a
25 risk at 75 plus 100, since the A-bomb survivors got 75

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1 plus 100, you know there's a risk at 175, we're
2 interested in is there a risk at 75. That's not much
3 of an extrapolation.

4 If that were the case I guess we'd be
5 done. The fact is there is a big extrapolation
6 because the difference is that in life span study, the
7 A-bomb survivors received all their dose, essentially,
8 instantaneously, or at least over a few minutes. So
9 they got about 100 tracks per cell nucleus in a very
10 short period of time. And we're interested in natural
11 background rates, which is one or two tracks per cell
12 nucleus per year. So in that sense there is a huge
13 extrapolation.

14 If there is a threshold, it's most likely
15 one dose rate, or the way I'd like to think about it
16 more is some dose increment over some critical time
17 period. So it might be, let's say, the time for DNA
18 repair is typically a few hours. So what matters is
19 how can you, as long as it's there, you get more than
20 a certain amount of dose in that time period there
21 could be a threshold let's say. Maybe that's the
22 wrong time period. Maybe what matters is time for
23 cell division, which would be weeks maybe, depending
24 on the type of cells.

25 Right now we know that there's these

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1 various low dose phenomena which might modulate the
2 risk at low doses and normally you -- these were
3 already described by Dr. Le Guen and Dr. Barcellos-
4 Hoff. Some of these could be beneficial. Some of
5 them could be harmful. I guess I would even -- I've
6 indicated that by with a plus that this is potentially
7 protective. Normally you would think of the adaptive
8 response that way as being protective, but it's not --
9 some of these aren't too clear.

10 Let's take the bystander effect. There's
11 a case where we -- presumably, when you get up to
12 doses where all the cells are hit, the bystander
13 effect is going to be less important than those direct
14 hits. That's at least the theory. Below that, the
15 bystander effects might be dominate. But the
16 bystander effect would be either harmful by causing a
17 mutation in a nearby cell, or it could be protective
18 either by inducing the adaptive responses in a
19 neighboring cell or killing off transformed cells as
20 there is some data to suggest.

21 Genomic instability, I said, is harmful.
22 Actually, I'm not even sure that's the case
23 necessarily. It could be -- it's really more a matter
24 of which of these mechanisms are operative at very low
25 doses as compared to higher doses. So, in fact, I

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1 guess genomic instability, while it's a bad thing to
2 happen, I guess if it happens at high does, not low
3 does you could think of it as protective -- not
4 protective, but it would give you a hormetic dose
5 response.

6 The same for low dose hypersensitivity, we
7 know that at very low doses cells are more readily
8 killed. That could be a good thing if it kills off
9 cells that are transformed. It could be a bad thing
10 if it leads to mutations.

11 Another thing, though, is there are types
12 of hormesis that aren't even covered here, like just
13 kind of a general effect, you know. I think of
14 exercise. If you exercise, you know, you go out and
15 you use all kinds of free radicals, tear down your
16 macromolecules and all this kind of thing, and, yet,
17 the general effect on the body is beneficial.

18 Now, you might think, well, maybe
19 radiation works that way too, you know, kind of just
20 an overall stimulus to your system? I think I would
21 argue that's unlikely, but I think some people are
22 thinking in those terms. Or it could stimulate an
23 immune response let's say again, perhaps unlikely, but
24 possible.

25 Well, one thing I would say, which

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1 radiation biologists maybe don't like the sound of
2 this, epidemiology trumps radiobiology. Where we
3 actually have the epidemiology data, I mean you've got
4 to think, well, no matter what the experiments on
5 cells show, if increased radiation leads to increased
6 rates of cancer, you've got to think that takes
7 precedence.

8 Or putting it another way is that if we
9 show that there's these kind of protective effects in
10 tissues, and so forth, before we would want to apply
11 it to human risk estimation, I think we'd want to show
12 that these mechanisms would operate in humans in a way
13 that would actually modulate the risk. So, yes, you
14 might not be able to -- as I say, you probably can
15 never get down to -- you can never do an
16 epidemiological study at natural background levels and
17 see an excess risk I don't think, or it's going to be
18 very, very hard.

19 However, you might be able to, if you
20 understood the mechanism well enough based on cells,
21 you might be able to look for some kinds of changes in
22 the cells of people to say, yes, we can see all the
23 damage is repaired or we can actually see these
24 beneficial changes in the tissues, so we can really
25 have confidence that radiation risks are lower than

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1 would be projected from epidemiology. So I think we
2 would need that step before we could make the changes.

3 Well, contrary to a lot of assertions you
4 see, there is epidemiological evidence for risks below
5 100 mSv or 100 mGy low LET. And Dr. Mossman nicely
6 summarized the first one yesterday that prenatal
7 x-rays at about 5 or 10 mGy led to increases in
8 childhood cancer. Now, I had some of the same
9 problems with it as Ken does. I mean this is one very
10 small part of the population, so, even if it's true,
11 it doesn't really affect the population risks very
12 much.

13 Secondly, it's not seen in the atomic bomb
14 survivors where you might have expected to see it, and
15 it's a rather small effect. But I would point out
16 that the dose -- but you do see a positive dose
17 response, which is one of the very strongest evidence
18 that it's a real effect, and the other thing I'd say
19 is these are x-rays rather than gamma rays.

20 What's the difference? Well, for gamma
21 rays, as I said before, at 100 mGy, we were seeing
22 around 100 tracks per cell nucleus. Here, because
23 they're x-rays, they're actually fewer electron tracks
24 for a given dose. So it turns out that 5 mGy of
25 x-rays, you're really getting down to very close to

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1 about one or two tracks per cell, and so we really
2 have evidence here for a finite risk down to nearly
3 one track per cell.

4 If you believe this resolve, if you're
5 going to look for a threshold, we're only going to
6 have to look between natural background and one track
7 per cell. So that's going to be a very special
8 mechanism. It doesn't work -- it doesn't occur one
9 track per cell, but it's occurring below that.

10 Two other examples, though, are ones where
11 -- by the way, why is that you can see this? I just
12 said that you couldn't get down below 100 mGy. The
13 reason you can here is this is a very large
14 population, and the other thing is that you're looking
15 at childhood cancers, which are very rare. So you
16 have a lot of more statistical power than you could
17 for just whole body radiation of the population.

18 For two other populations, we have data
19 where the individual doses are very small. As I said,
20 I thought what really matters is probably the dose
21 over a short time period. We have two groups of
22 patients who were followed in their treatments,
23 tuberculosis patients. They were fluoroscoped
24 periodically every couple of weeks or so. Scoliosis
25 patients, their treatment was being monitored to see

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1 the changes in their spine.

2 And we're particularly interested in
3 hearing the female patients who received fractionated
4 doses, that is, so at any one time they only received
5 a few mGy, less than 10 mGy, but they received
6 repeated, up to 100 or so fractions. So the total
7 dose was large enough to cause a measurable increase
8 in cancer even though the individual doses were very
9 small.

10 In both these groups they saw an increase
11 in breast cancer. Now, again, breast cancer rate is
12 a special case. It's possible, but it's certainly a
13 very important one since we have a lot of young women
14 who might be susceptible. It appears that, again,
15 just a few tracks per cell nucleus could -- this
16 provides evidence that that can cause breast cancer.

17 And then still in other cases, tinea
18 capitis group who were irradiated for ring worm in
19 Israel and they got slightly higher dose, 17 mGy,
20 which is still pretty low, and that saw an increase in
21 thyroid cancer in that group. So that's another type
22 of cancer.

23 But, again, both these cancers are
24 hormonal. We can't say that it applies to everything,
25 but this is pretty strong evidence that -- one other

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1 thing, not only did these tuberculosis patients get
2 breast cancer, they got it at about -- the risk per
3 unit dose about the same as in the A-bomb survivors.
4 So that would say that LNT, even, goes down to the --
5 not only is there not a threshold, but LNT works
6 pretty well down to this type dose.

7 DR. BARCELLOS-HOFF: But that's
8 cumulative, right?

9 DR. PUSKIN: What?

10 DR. BARCELLOS-HOFF: You required a
11 cumulative dose?

12 DR. PUSKIN: Yes, right. But these
13 individual tracks somehow caused cancer.

14 DR. BARCELLOS-HOFF: Were added --

15 DR. PUSKIN: Yes.

16 Well, can we go lower still? And I think
17 there's some chance by looking at epidemiological
18 studies of chronically exposed individuals where,
19 again, you have to have enough total dose to see a
20 cancer, but the dose over a day, a week, can be even
21 smaller than what we saw in the fractionated dose.

22 Here are some populations that are
23 chronically exposed. The nuclear workers is the one
24 that immediately comes to mind and it's questionable
25 whether this does have the statistical power because

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1 the doses are pretty low and there's potential
2 confounding.

3 I would say what we really have out of the
4 nuclear workers' study so far is that the risks that
5 we're estimating for chronic radiation are not way
6 low. We know that the LNT is not greatly under
7 predicting the risk. You know, if the risks were ten
8 times higher than what we project, I think you would
9 have seen something, nuclear workers or some other
10 studies. You'd probably also see increases of
11 leukemia in Colorado and the rest of the country and
12 things like that.

13 Some of these studies may not be useful.
14 They all have problems. So far the first population
15 hasn't really shown any clear indication of increased
16 risk. The Mayak workers probably are not going to be
17 very informative just because their doses are so high
18 that even one day they get what those TB patients got,
19 and they've got additional doses from medical, so
20 their doses are extremely high of the order of 10 mGy
21 a day.

22 The Semipalatinsk gives another one that's
23 -- I don't want to discuss that one. But the two of
24 them that are probably the most promising I think are
25 the Techa River cohort and the occupants of the cobalt

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1 60 contaminated buildings in Taiwan, but both are --
2 you know, they're still working out the dosimetry.
3 The cobalt 60 population, the epidemiological followup
4 is very short.

5 Interestingly, both of these studies show
6 a statistically -- at this point, at least based on
7 the current followup and the current dosimetry, both
8 these studies show a statistically significant
9 increase in both solid tumor cancer and leukemia.
10 Again, this is probably down well below 1 mGy per day
11 perhaps. I don't know. It's not too clear because
12 the Taiwanese, for example, there's a big range of
13 doses and they really haven't broken it down, dose
14 rates.

15 And Techa River, there is also quite a
16 range of doses, so more needs to be done. But the
17 preliminary results suggest about the same risk per
18 unit dose as the A-bomb survivors, suggesting the
19 DDREF is not very super high, not ten or more, or
20 something like that, and that there's not a threshold.
21 Now, that's sort of to the side.

22 But the risk principles I'd like to talk
23 a little bit about how we apply these to standards.
24 I don't know, from the introduction I got yesterday,
25 maybe this is less interesting than policy here. I

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1 think risk protection standards need to account for
2 uncertainty, and, particularly as Dr. Land talked
3 about yesterday, we have to ensure that we are not
4 greatly underestimating the risk.

5 So if there is a reasonable probability,
6 even if we think there is likely to be a threshold,
7 even if there is a substantial probability there is
8 not and that LNT is correct, even it were to say one
9 chance in three, we would probably not be able to
10 change our regulations. We would have to -- in order
11 to protect, to make sure that everyone is -- that the
12 bulk of the population is at a low risk level, we
13 would still have to regulate radiation fairly
14 stringently.

15 If we did get new signs and were really
16 convinced -- or there was pretty good evidence that
17 there was a threshold or hormesis, or something like
18 that, at these very low dose levels, would we change
19 our regulations?

20 Well, one thing, is suppose the risks went
21 up substantially, a super linear dose response, based
22 on past history, regulations are likely to get
23 tightened if that were a very significant increase.
24 If the opposite were true, if let's say we had strong
25 evidence that there was a practical threshold, not a

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1 strict threshold, but let's say we've said, oh, risks
2 are really ten times lower, 50 times lower at least,
3 would we change the regulations?

4 The answer is maybe, maybe not. It would
5 depend. It depends whether the statute would permit
6 it and you would also have to say that there's a need.
7 Some people would say, oh, let's take the drinking
8 water rates. Somebody might say, well, these are too
9 stringent; the risks are really 50 times lower. Well,
10 people would say, but everybody's meeting them; what's
11 the compelling need to change them? So that would be
12 the --

13 Before rejecting LNT I would say that EPA
14 would want a scientific consensus as reflected in
15 these kind of reports from National Academy, UNSCEAR,
16 NCRP, and so forth, that we want a concurrence from
17 our science advisory board. In fact, right now we are
18 revising our risk estimates based on BEIR VII
19 primarily, and our changes are subject to science
20 advisory board review. And they've already talked,
21 weighed in a little bit on this issue. They wanted us
22 to go beyond BEIR VII to some extent and acknowledge
23 more of the uncertainty about the risk at low doses.
24 Tony Brooks was on our advisory committee.

25 We'd want acceptance from the other

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1 federal agencies I think, you know, try to -- I think
2 we want as much consistency across the federal
3 government as we can have. And we would also want a
4 transparent, public process that people from the
5 public would have a chance to criticize what we're
6 doing and that we would have to consider and we would
7 want our advisory board to consider any evidence that
8 people would want to, at least make it clear that we
9 do consider all the evidence from everywhere.

10 Well, if we did think there was a
11 threshold, let's say, how might that affect
12 regulations?

13 First of all, if the threshold is below
14 natural background, it's not going to have any effect.
15 I mean nobody really cares if, okay, we get as I say
16 1 mGy per year. If there's a threshold of 0.1 mGy per
17 year, it doesn't really matter. That's not going to
18 have any -- and remember, in case of radon, we're
19 actually in this situation that for radon we already
20 know that levels that people get from natural -- in
21 their homes, indoor levels of radon that a lot of
22 people get, has been shown with epidemiological
23 studies that there's a increase in lung cancer.

24 Now, if there was a practical threshold
25 above background, they could perhaps change some

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1 regulations that are based directly on risk. One is
2 the soil clean-up levels potentially. Another is the
3 drinking water MCLs. I talked about the MCLS, about
4 the compelling need.

5 But even more so, there's also a provision
6 in the Safe Drinking Water Act amendments that says
7 what they call no backsliding, that if you have a
8 regulation and it's working and you now -- you cannot
9 make the regulation more stringent -- less stringent,
10 sorry, you cannot relax it unless, let's say you said,
11 oh, it's really a strict threshold and there's no
12 risk, in that case you could.

13 If it was a practical threshold, I think
14 it's a gray area. I think if the risks were below
15 1:1,000,000, which is where EPA normally doesn't
16 regular below 1:1,000,000 maybe, but if the risk went
17 from 10^{-4} to 10^{-5} , no backsliding regulation would say
18 you really can't do anything about it. Now it might
19 be that at that point Congress would say change that
20 no backsliding regulation.

21 This is important because a lot of
22 clean-up levels and things relating to waste disposal
23 are tied to the Safe Drinking Water Act in terms of
24 the MCLs for drinking water.

25 Well, issues in setting a threshold based

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1 standard, well, obviously, would be magnitude of the
2 threshold dose or dose rate. The uncertainty and
3 where that dose is, the uncertainty and how big the
4 risk is below that level would have to be considered.

5 You would have to consider sensitive
6 subpopulations. It's a threshold for most people, but
7 what about people with let's say they're missing some
8 repair enzyme or something or they have less of it.
9 And you have consider multiple sources. Say, for
10 example, and there's no epidemiology that rules this
11 out, let's say that that there's a threshold for
12 chronic radiation at 10 mSv/y, 10 mGy/y has no risk,
13 okay, so no one would be harmed by this dose.

14 Well, you still, for an individual source,
15 you would still want to set the level lower than that
16 because people are exposed to radiation from multiple
17 sources. So it might be that if there were a
18 threshold of 10 mSv/y you might still have an
19 individual source limit that was 1 or 2 mSv/y. This
20 is along the same lines where, for example, ICRP
21 recommends that, from all sources combined, you can
22 receive 1 mSv/y. Then they have individual source
23 constraints I guess they call them that are 25 or 30
24 percent I think of that.

25 Well, what are the down sides of LNT? I

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1 think we've heard a lot about that already the last
2 couple of days. You've spent too much money
3 obviously. That actions taken to reduce these very
4 low risks may not be warranted from a cost benefit
5 standpoint. We're spending more money than we'd like
6 to.

7 The other is probably more important. I
8 think more people are disturbed by this. That this
9 perception of the risk of low doses cause people to
10 either oppose beneficial nuclear technologies or to
11 potentially shun advisable medical procedures like
12 mammograms. I don't think actually think the latter
13 occurs so much, but those people trust their doctors
14 so much. But it could and I think this is a problem,
15 and I can't say that I've got the solution to it.

16 How do we live with this? The obvious
17 answer is education and I think a lot of people are
18 frustrated. We've tried hard at this and had very
19 limited success. I suggest you try to help the public
20 put the risk into perspective and to balance the risks
21 and benefits and to make clear to them that you cannot
22 -- life has risks and some risk is unavoidable.

23 The thing about LNT though is it says that
24 low dose's risks are very low. That's what LNT,
25 that's the message is that risks decrease as the dose

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1 decreases. I guess I've spoke up several times
2 already. I think that if we try to say that, well,
3 we're not really sure there is a risk, so let's just
4 not say there is one. I think it's going to damage
5 the credibility and work against the trusted
6 scientific community and the radiation protection
7 community in particular.

8 So to summarize, radiation protection is
9 based on LNT and that's consistent with current
10 science, and the recent Academy recommendations. We
11 would really need a consensus of these kind of
12 scientific bodies before we would adopt a threshold.
13 If you could show there's a threshold, yes, it could
14 change regulations conceivably. However, you'd have
15 to worry about things like safety factors, sensitive
16 subgroups, and multiple sources.

17 That's all I have.

18 CHAIRMAN RYAN: Thank you very much.
19 Questions? Dr. Mossman, then Dr. Tenforde, then
20 Dr. Le Guen.

21 DR. MOSSMAN: On your last slide, what do
22 you mean by a change in standards? To me the whole
23 problem about thresholds and the like is not about the
24 dose limit, it's about how you apply ALARA. In other
25 words, I don't think any of this discussion has

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1 anything to do with dose limits because what radiation
2 protection is all about is a top down approach in
3 which the dose limit is the ceiling, you use ALARA to
4 reduce the dose as low as reasonably achievable.

5 The question about a threshold then
6 becomes how far do you take the ALARA down? Because
7 once you reduce the dose, if you're down below
8 threshold, then, of course, you're not getting any
9 more incremental benefit for additional costs of dose
10 reduction. So, to me, the whole issue is not so much
11 the dose limit, it's how you apply ALARA. Could you
12 comment on that?

13 DR. PUSKIN: Well, I would say this, that,
14 first of all, you can think of regulation -- I don't
15 know that it always works this way, but I think this
16 is the way it was envisioned and to some extent, great
17 environmental regulations work this way, but,
18 unfortunately, they don't entirely. It's to set a
19 level of acceptable risk, okay -- or, unacceptable,
20 and above that we're going to regulate, and that might
21 be a 10^{-4} risk or something like that. And then below
22 that we look at cost benefit and we try to reduce it
23 further as if it's cost effective.

24 As far as I know, it's almost always
25 decided by the first, that it's almost never cost

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1 effective to go lower than what you're already doing
2 with this risk. Now that may be not true in the
3 occupational setting. I don't know. But
4 environmental, you set the standard.

5 Let's say it's 15 millirem per year,
6 whatever. They never say, oh, wow, let's calculate
7 whether we can go down to 1 millirem and it's still
8 effective. It won't be. Probably the 15 wasn't
9 effective in terms of if you put a reasonable value on
10 human life, are risks avoided is a better way to say
11 that. You probably wouldn't have reduced it to 15.
12 But we've decided that 15 was -- that above that was
13 unacceptable, or 15 and lower was acceptable. So
14 that's usually the driving point.

15 I know when we set the standards for the
16 Clean Air Act, it was more looking at how many people
17 were in different risk ranges and it was decided that
18 taking the overall picture, again, that roughly
19 10 millirems, which is about 10^{-4} risk, was about as --
20 -- didn't want to go lower than that, but there was --
21 in fact there was a court case which kind of said that
22 the risks should be not much above the 10^{-4} ,
23 something 10^{-4} range, and at times EPA has said 10^{-4}
24 ranges means three times 10^{-4} or two times 10^{-4} . It's
25 sometimes higher than one times 10^{-4} .

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1 So things are generally thought -- usually
2 staying in that -- not going above that is usually the
3 driving thing. Now, the exception could be in the
4 Safe Drinking Water Act where sometimes there are
5 carcinogens out there that, you know, can easily be
6 regulated down to 1:1,000,000, you know, they're not
7 there and so it's possible to do.

8 I hope that answers your question.

9 DR. MOSSMAN: Managing chemical risks is
10 an entirely different game than radiation risks. I
11 mean chemical risks, you're quite right, it's a bottom
12 up approach. With ionizing radiation, it's a top down
13 approach. So there's a different philosophy. Now I
14 can't tell you whether one's better than the other.
15 It's just from historical --

16 DR. PUSKIN: Also, I'd say that, for
17 example -- maybe Mike could speak to this. The NRC
18 operates more on this top down approach, that here's
19 a limit and we really try to go lower than that. EPA
20 sets the limit pretty low and say, if you can meet
21 that, you're done, you know, kind of thing.

22 CHAIRMAN RYAN: Anybody else? Tom, you
23 had a question.

24 DR. TENFORDE: I just wanted to make a
25 comment. You were talking about the ICRP 1 mSv/y

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1 public dose limit, which is the same as NCRPs, and you
2 mentioned that for a single source under the control
3 of a single operator or group of operators, they
4 recommend three-tenths of a mSV. But I wanted to
5 remind you in 1984 NCRP wrote a statement at the
6 request of EPA when they were beginning to develop the
7 CERCLA regulations recommending 0.25 mSv/y --

8 DR. PUSKIN: That's where I got confused.

9 DR. TENFORDE: --for any single source
10 given that the other exposures of an individual
11 exposed that source may be unknown. And, therefore,
12 the idea was you might have as many as four such
13 sources contributing up to 1 mSv/y.

14 But 0.25 was conservative and there was
15 huge debate about that in terms of shielding for
16 medical facilities and so for. And, in fact, in 2004,
17 NCRP published statement 10 reaffirming the public
18 dose limits and the applications of public dose
19 limits, and reconfirmed that this was, you know, not
20 an unrealistic or unreasonable limit, and in a 70 year
21 life span will get you a risk of more than 10^{-4} of
22 cancer, more like 10^{-3} . But it's still a very low
23 risk compared to natively occurring natural cancers,
24 or cancer caused by other sources associated with life
25 style, you know, smoking, whatever, or, for that

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1 matter, radon at a higher level anyway.

2 I just wanted to reconfirm that that
3 single source limit is still in place.

4 CHAIRMAN RYAN: Dr. Le Guen was next.

5 DR. LE GUEN: Well, I would like to come
6 back on two sides. First side is on the
7 epidemiological studies of chronically exposed
8 cohorts. From my point of view you forgot to mention
9 another study. For example, you remember women
10 workers who painted with radium, watches, and has
11 developed radium osteosarcoma. And in this kind of
12 study they showed also a threshold.

13 And also about Mayak workers and internal
14 contamination, I think the publication has shown
15 curvilinear. So you remember what I said yesterday,
16 from my point of view there is not only one, but
17 perhaps more than one and perhaps several curves
18 between dose and effects.

19 And my question about the slide, why
20 didn't you take into account people exposed to all
21 natural background, natural radiation for a risk
22 assessment? Because it is chronic exposure and I
23 think that it would be very good to have
24 epidemiological studies on this population.

25 DR. PUSKIN: I know Charles could speak to

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1 that latter one.

2 DR. LAND: Well, you know, there are such
3 studies. There's the high background area in China.
4 Personally, I think these studies tend to be
5 disastrous because -- well, if you look at the reports
6 from the Chinese study, every time there's something
7 you see in excess, well, it's because these women have
8 few children, or so forth, and it's just -- we just
9 don't get anything, any good information out of it
10 because it's so difficult to control that the sort of
11 things that might have the same level of effect as the
12 exposure you're studying. I mean maybe in a more
13 regulated world it might be possible.

14 DR. LE GUEN: Because in China and India,
15 we have begun to have these kind of studies in France
16 and also to associate it with molecular biology
17 because we simply say it's a different dose. From our
18 point of view, if you receive ionized radiation, if
19 you receive from natural background or from external
20 sources, if we assess the dosages, it's the same dose.
21 So from our point of view it would be very interesting
22 to estimate the risk.

23 DR. PUSKIN: The problem is like if you
24 have -- an example in the case Charles gave, let's say
25 the level, let's say it's even five times normal, I

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1 mean we think that natural background radiation causes
2 roughly three percent of the cancer. So in this other
3 area it might cause 12 percent, 15 percent. So that's
4 12 percent higher.

5 I mean the difference between Connecticut
6 and Louisiana is more than that, and here's two
7 separate areas of China, which we don't know that much
8 about, so they could easily differ by that amount.
9 It's hard to -- the potential for confounding is too
10 great.

11 DR. LE GUEN: Yes, but perhaps what's so
12 interesting about life styles if we have a good
13 control group, because one of the problems that we
14 have at low dose, say, is not only one genetic
15 connection, but there is a lot of them, and perhaps
16 we'll see factors due to life styles. And I think t
17 his kind of study, which can -- of course, I'm sure
18 that it's not because you will have only one study
19 that you will change everything.

20 But I think we must be open minded and we
21 must continue to work on this field to a lot of
22 different experiment. Because, of course, I said
23 yesterday from my point of view, if we have Hiroshima
24 and Nagasaki just one case, one exposure, we've
25 neutron and gamma ray and very short exposure, and you

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1 can see and you mentioned different studies and
2 cohorts, and we have different sources. We have
3 internal contamination with plutonium. We have
4 external exposure and so on, so different case.
5 Yesterday we mentioned the problem of dose rate, and
6 that's why that it's very difficult. Of course,
7 that's why, today, we are here. It's because it's so
8 sophisticated. Because we have different kind of
9 source, different kind of exposure, and we must take
10 into account all of this. Okay?

11 DR. PUSKIN: Yes. I would say the radium
12 dial painters, I don't get into that much because
13 that's a high LET situation, but there is -- not
14 everyone thinks that that is convincing the threshold.

15 For example, there risk study where they
16 have injected radium in patients where -- radium-
17 induced bone cancer where it's certainly consistent
18 with linear no-threshold. And the radium dial
19 painters is very high dose. What's clear is it takes
20 a lot of dose to see an excess of bone cancer and it's
21 a very high dose. The damage to the bone tissue is
22 very high, so we're not really looking at the kind of
23 low dose kind of a phenomenon.

24 CHAIRMAN RYAN: Jerry, just a follow-up
25 question if I may. In some of the other studies that

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1 are in your list, the Techa River cohort, it's
2 reconstructed doses, and you commented on some of the
3 issues that that's tough. That's real tough, I mean,
4 you know, the fuel cycles, and how they processed
5 fuel, and when they processed fuel all contribute to
6 the short lived component.

7 I guess I'm not picking on that so much as
8 saying that I think -- I don't know whether it's a
9 background study or high background study, or a real
10 exposure case, or a mixed exposure case with alpha and
11 gamma. Every study has good points and bad points in
12 how you can extract the data.

13 DR. PUSKIN: It's a question of how well
14 you can do that. I mean it's whether -- I don't know
15 what you'll end up with.

16 CHAIRMAN RYAN: Well, what my point is I
17 think -- the point I would offer is that all of them
18 probably have some value and all of them probably have
19 some flaws. So try to pull all the evidence together
20 rather than just setting one aside for whatever
21 reason.

22 DR. PUSKIN: I guess I would maybe retreat
23 a little bit. When I was saying that the epidemiology
24 takes is trumps, if you have an epidemiological study
25 which is positive and you have a strong radiobiology

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1 indicating that it can't be positive, you should look
2 at the weaknesses of that epidemiology study and see
3 whether you can reconcile it. I mean that's part of -
4 - I mean it's not --

5 DR. LE GUEN: But you know just an example
6 about nuclear workers, you know that for a different
7 study we observed an LC effect, and the LC effect,
8 there are two reasons. Perhaps we have a natural
9 selection about workers and we follow those works.
10 That's one of the reasons, also, for the moment if I
11 take into a French cohort, I say yesterday, because
12 this cohort is too young. And we need time, also, and
13 that's why for this kind of epidemiological studies,
14 I say it's not only one research that changes
15 something. We need to be very serious, but we must
16 take everything into account, not only one point.

17 CHAIRMAN RYAN: If I may, I think we want
18 to make sure we get Dr. Holahan's presentation in this
19 morning, and we can certainly continue this discussion
20 after lunch in our roundtable. So, with that, let's
21 hear our second presentation and we'll go from there.

22 DR. HOLAHAN: Good morning. I'm Vince
23 Holahan. I'm a senior level advisor for health
24 effects research programs in our office of Nuclear
25 Regulatory Research.

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1 I'd like to, first, apologize or account
2 for Dr. Cool. Dr. Cool would loved to have been here.
3 He's my counterpart in our materials office.
4 Unfortunately, he's part of a drafting session in
5 Vienna, and I guess Vienna in April versus Washington
6 in April, he made the decision to do some traveling.

7 I'd also like to express the thanks of the
8 health effects group, as well as our environmental and
9 rad transport group. We appreciate the guidance that
10 you've provided to our groups up on the ninth floor
11 over the past years, and I hope even in an advisory
12 status with the ACRS that you'll be able to give us
13 very valuable input.

14 With that said, what I would like to do
15 today is provide what we would call a staff
16 perspective on the low dose work and some of the
17 changes that have gone on in the literature for the
18 past 15, 17 years. This is a staff perspective,
19 because as we've previously briefed the ACNW, the
20 staff is looking at some of the materials that have
21 been produced. We're looking at our regulations, part
22 20, part 30, part 50, part 62, to see whether or not
23 we should make a wholesale change to part or all of
24 this.

25 My role is to look at the technical basis

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1 for those reviews. Options will be prepared and we're
2 hoping to send to the commission a staff requirements
3 memorandum at the end of this year. Therefore, if
4 this meeting were held approximately a year from now,
5 I'd love to tell you exactly what the Agency was going
6 to do.

7 It's a staff perspective because I've been
8 specifically told, try not to get ahead of our
9 commission on what we think might happen because we
10 really don't know what's going to happen. So with
11 that in mind, what I'd like to do is discuss some of
12 the biology through the rose-colored glasses that I
13 wear as a regulator.

14 I'm appreciative to Dr. Puskin for
15 providing the science, but I'm not going to get into
16 the damage of the DNA double strand break, and I hope
17 not to get into too much detail on the epidemiological
18 studies. But how does this information affect our
19 regulations and where we should change? I'll talk
20 about some of the technical basis information that we
21 look at, where we think the science might be today,
22 and how it's going to impact our regulations.

23 First off, you have to understand we've
24 got three basic fundamentals in our radiation
25 protection system. (1) You must have justification

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1 from any exposure to radiation. We don't permit
2 licensees to have unauthorized or frivolous exposures
3 to radiation. (2) We have a limitation on the
4 exposure, whether it be occupational or public. And,
5 (3), optimization, and our regulations would call that
6 ALARA.

7 For all intents and purposes, it's a dose
8 based system. We've heard a little of the differences
9 between EPA and NRC, that is to say it's
10 observationally based. We look at effects in human,
11 animal systems and we start setting dose limits below
12 that. And then we use a series of constraints, if you
13 will, in some cases to worry about source specific
14 items.

15 There are a number of assumptions. We
16 assume in our regulations that there's a linear
17 no-threshold response for stochastic effects,
18 primarily cancer hereditary effects. Our regulations
19 are gender averaged and age averaged. And right now
20 we protect the most exposed individual. EPA is
21 looking at differences such as looking at the most
22 sensitive individual, but that's a discussion that's
23 going to probably go on with their science advisory
24 board for at least a number of months.

25 Dr. Cool wanted me to put in that our

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1 system of protection in the U.S., at least with the
2 NRC, is supposed to be coherent as well as
3 predictable. That doesn't necessarily mean it's
4 comprehensive or consistent. The reason I say that is
5 many of our regulations are based on regulations from
6 the ICRP, 2, 26. We actually have 60 involved. And
7 there are many things that we're doing today that are
8 consistent with the recommendations in report 103.

9 But it's been a period of time since we've
10 done a major revision. That was some 17 years ago.
11 That revision was the product of many years of work by
12 the staff. I guess the question is, and this is a
13 question that will come up next week at the NCRP
14 meeting on the low dose radiation as a topic that Dr.
15 Lipoti as specifically asked on the second day, what
16 would it take to prompt a change in the NRC
17 regulations?

18 First and foremost, we'll have to go back
19 to 10 CFR Part 50. That's our backfit rule. That is
20 to say a revision would have to prompt a substantial
21 increase in the overall protection of public health
22 and safety, and that increase is going to have to keep
23 in mind both the direct and indirect costs associated
24 with that change.

25 In 1991 we had great difficulty

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1 demonstrating that significant increase in public
2 health and safety, with ICRP 60 and many of the
3 changes that proceeded that. Even though we had DS86
4 changes in the risk coefficients, that wasn't
5 sufficient to prompt a change because of backfit. But
6 the Commission has the ability to waive that.

7 What other things might we consider?
8 Well, clearly, updated scientific information.
9 Obviously, there have been many changes that we'll
10 talk about in a couple of minutes. Possibly reduction
11 in burden, risk informed regulation, and the last item
12 here that Jerry also eluded to that would be new for
13 the Commission is inner agency alignment. Clearly,
14 none of our federal agencies are on the same page.
15 This might be a reason to prompt a change in our
16 regulations.

17 So what do we do? Obviously we look at
18 the basic research. This includes the DOE low dose
19 radiation program. That's a 10-year, \$17.5 million
20 program. For all intents and purposes it dwarfs much
21 of what NIH is doing. We also look at much of the
22 work that's done in the EC with Neil Kelly. That
23 program is on the order of about \$30 million euros,
24 and given the difference between the euro and the
25 dollar, it's a very significant program.

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1 We take a look at peer-reviewed
2 publications, as well as unreviewed publications. We
3 find that many of the states will do epidemiological
4 studies for cohorts around various facilities. Those
5 aren't necessarily in the journals, but we'll take a
6 look at those. There was a recent report in Germany
7 about childhood leukemia I believe it was in proximity
8 to their power plants. That has not necessarily been
9 peer reviewed and published per se. I think it's more
10 of an agency report, although it's got their own
11 internal procedures.

12 Literature reviews, this is one of the
13 areas that we, as an Agency, get very much involved
14 in. We were one of four sponsors of the BEIR VII
15 report where we looked to established, balanced
16 technical review committees to survey the literature,
17 put together a review and recommendations on future
18 research. I'd have put up here the French National
19 Academy review, but I didn't have a copy of the page
20 to insert in.

21 (Laughter.)

22 DR. HOLAHAN: The other item here is
23 UNSCEAR, the United Nations Scientific Committee on
24 the Effects of Atomic Research. They actively are
25 engaged in looking at both radiation sources, looking

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1 exposures, and evaluating the impact of those
2 exposures.

3 We have a number of bodies that will look
4 at all of this information, both the summary reports
5 and the individual reports, generally, again, focusing
6 on the peer review publications, make some summary
7 recommendations in terms of radiation protection,
8 whether it be the ICRP or the NCRP. We fund both
9 organizations to provide their guidance. And all of
10 this, again, all of it impacts both the regulations
11 here in the U.S., in one case it's our 10 CFR series,
12 as well as the international series, that's the basic
13 safety standards.

14 Well, needless to say, in 17 years there
15 has been a substantial amount of work that's gone on.
16 We were and continue to be participant at the DOE
17 workshops. We were at workshop I, and, quite frankly,
18 myself and some of the other regulators tried to
19 articulate to the investigators what low dose is,
20 trying to explain to them in regulatory space we're
21 interested in mSv exposures or several mSv exposures
22 and we're talking to investigators that have been
23 working in gray type of exposures.

24 I know that when we worked with Dr. Upton
25 we defined for LNT. We were interested in low dose.

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1 That low dose was 20 rads. And at that junction the
2 question was, is, well, there's no information there.
3 Well, the reason there wasn't a lot of information
4 there is we didn't have the tools. And, fortunately,
5 by articulating to a low dose program that we were
6 interested in exposure of 10 rad, 10 centigrade or
7 less, it's prompted a lot of research to develop tools
8 so we can examine some of the effects of the very low
9 doses.

10 JCCRER has been a program that this Agency
11 has been very much involved with for over 10 years.
12 Now, Dr. Puskin mentioned he was little concerned
13 about the doses that the workers are receiving, but we
14 view those as intermediate doses that are between the
15 atomic bomb survivors and some of the very low dose
16 studies. But, more importantly, there is a huge
17 cohort of female workers that were exposed either
18 externally or internally to help us ferret out some of
19 the gender differences, and we're hoping to see some
20 of that come out of that data.

21 Just in the last year or two we have had
22 some significant information out of the RERF. A
23 revision of the dosimetry system, DS02, a re-analysis
24 of the mortality data, which basically reaffirmed that
25 the estimates that were in ICRP 60 are very relevant

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1 and valid today. But, more importantly, last year we
2 got some information on cancer incidents, and that is
3 going to be of more value to us than probably the
4 mortality data because of the advances in various
5 countries on treatment of cancer. The mortality data
6 tells very little if we're dealing with exposure to
7 radioiodine.

8 UNSCEAR, the last major compilation of
9 data was put out in 2000; inheritable effects in 2001.
10 There are at least five reports that should have been
11 out last month. These reports are going to be dealing
12 with the epigenetic work. We've got non-cancer data
13 that's going to be presented in a separate annex.
14 We're looking at a review of the Chernobyl. So we're
15 hoping in the next couple of months we'll have a
16 series of reports out of UNSCEAR. Not only coming out
17 this year, but we have at least four more annexes that
18 we're looking at this year for finalization for next
19 year.

20 BEIR V, BEIR VII, the French National
21 Academy report's come out, again, it will be very
22 interesting to get a group of folks together to find
23 out why two groups can look at virtually the same data
24 and come up with diametrically opposed conclusions.
25 In ICRP over the last 17 years has come out with some

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1 43 reports, possibly 44 reports, and the question
2 we'll have to ask is, as an agency, do we want to look
3 at ICRP 60 recommendations or do we want to make a
4 jump all the way to 103 and see if we can entice our
5 sister agencies to make the same type of change.

6 I put this up here very briefly. I think
7 we've got pretty much consensus if we're looking at
8 epidemiology, and if we're looking at excess relative
9 risk, it can be fit with a linear curve, maybe a
10 linear quadratic curve. Maybe the limit of the data
11 is down to about 100 mSv. We had a sponsors' briefing
12 in 2005. I asked the epidemiologist on the group, Dr.
13 Gilbert, what the lower limit of their sensitivity
14 was, and she was 100 mSv, that's it. I asked the same
15 thing of Dr. Bill Dewey, the molecular biologist on
16 the group. He said 1 centigrade.

17 Dr. Puskin indicated that there are a
18 number of studies that seem to be pushing these limits
19 a bit. I could be the recent mortality morbidity
20 study from RERF. With the trends analysis they think
21 they might be able to go down to about 10 mSv. But
22 there's some question there. You can force the fit of
23 that curve to actually show that you could possibly
24 have a practical threshold of maybe 60 mSv.

25 The Techa River data is down into the 10s

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1 of mSv. But, again, as we've discussed over the last
2 day or so, there's a lot of question, and not only the
3 cancer incidence, but the certainty we have on the
4 dose estimates.

5 And the workers' studies. Dr. Cardice is
6 indicating that there's an increase at very low doses
7 to radiation exposure occupationally. Much of that
8 was driven by the Canadian data. However, there was
9 a problem with the Canadian data. They underestimated
10 the exposures to the workers.

11 In the 1970s they set up a national
12 database for radiation exposure. At Chalk River they
13 zeroed all the workers out, so any of the prior
14 exposures to those workers prior to 1974/1975 was not
15 included. When you include that data, there shouldn't
16 be an excess increase in the Canadian workers.

17 That information is prepublication, but
18 the Ministry of Health up in Canada is working to get
19 that out. Therefore, when I extrapolate from 10 rem
20 to 1 rem, 100 mSv to 10 mSv, I'll put that in as a
21 dash line. The cellular data, depending on the
22 source, is primarily out of BEIR VII, would take this
23 down to about 1 rem, again, showing dicentrics,
24 acentrics, increased mutation frequencies at these low
25 doses.

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1 But still, even with this information, we
2 have to put a dash line in assuming LNT. Because,
3 quite frankly, we don't know what's going on here.
4 And our concern, whether it be DOE, EPA, or the NRC,
5 is it's this very low dose region right down here that
6 we're concerned for regulatory purposes.

7 We've seen these phenomena over the last
8 day, day-and-a-half now. The question is, what impact
9 may, could, should, will that have on our regulations?
10 With bystander effects, this was considered by the
11 BEIR VII committee; temporarily discounted. This has
12 got a huge impact on LNT and target theory.

13 What is the size of target when we talk
14 about radiation exposure? Is it the nucleus? Is it
15 the whole cell? Is it a group of cells? What impact
16 does that have on the surrounding tissue? What impact
17 does that have on the organ? Keeping in mind that
18 type of information might help us understand what's
19 going on, but it doesn't necessarily change the
20 epidemiology.

21 Genomic instability, is this real? Can we
22 actually induce damage in cells that will perpetuate
23 to the daughter cells, to future daughter cells, to
24 future daughter cells? We heard that there might be
25 some information for that. Maybe apoptosis takes care

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1 of that.

2 In either case, as an agency, what I'm
3 interested in is to see if this type of information
4 can be repeated from laboratory to laboratory. One of
5 the problems that they've had with investigators in
6 the DOE program is getting results to repeat between
7 different laboratories.

8 Adaptive response, priming dose required
9 to some reduced sensitivity to a following challenge
10 dose. Those priming doses are greater than our public
11 dose lines. We're not going to use that for public
12 protection.

13 What about emergency responders? We're
14 not going to allow our emergency responders to receive
15 more than 25 to 50 rem, 250 to 500 mSv. Chances are
16 we're not going to do an adaptive response. We're
17 going to control the exposure of those individuals.

18 Hyper-radiation sensitivity, I've actually
19 seen it in the tissue culture. Haven't reported on
20 it. I thought it was an artifact where at very low
21 doses, for some reason, you'll see a dip from let's
22 say 95 to 90 percent surviving fraction.

23 Now, the question is, does that incur in
24 organs and tissues? Have we observed this in the
25 clinic? Have we observed with conventional

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1 radiotherapy a 10 percent breakdown in surrounding
2 tissue to where we've got a targeted region? So I
3 guess the question would be with hyper-radio
4 sensitivity, do we see this in vivo?

5 What issues might prompt a change? Well,
6 here are several of them. What is the real threshold
7 for lens opacification? ICRP 60 say 5 Sv. Dr. Wortle
8 last year, prior to his passing away in February, in
9 *Radiation Research* published an article on lens
10 opacification for the Chernobyl liquidators suggesting
11 that it might be on the order of about 700 mSv for a
12 threshold, not 5 Sv.

13 Can that be reduced in other studies?
14 That might be important because that might prompt a
15 change on our regulations ocular exposures.

16 Non-cancer diseases, RERF is starting to
17 report that there might be an occurrence of
18 cardiovascular diseases, possibly the same type of
19 thing in some of the Chernobyl workers. The problem
20 we have with non-cancer diseases is the induction of
21 those type of diseases is about one-tenth the excess
22 risk than radiation, very low levels.

23 The second problem that you run into is,
24 what is the impact of socio-economic effects on those
25 individuals? And I'll cite the Russian liquidators as

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1 an example. You have a group of individuals that
2 smoke, high alcohol consumption, diet is very fatty.
3 They've had a decrease in the life span of the Russian
4 males. It's currently about 57 years of age compared
5 to surrounding populations where we're talking late
6 60s, early 70s.

7 How do you account for all of those
8 confounding factors and then make judgments about
9 non-cancer diseases? It appears to be a deterministic
10 effect. But if it is, what's the threshold?

11 Gender sensitivity, our regulations are
12 gender averaged. Is there a real difference between
13 males and females to 1 Gy exposures? We don't know.
14 Should it be something that we need to tease out? It
15 would be something that would be after consideration.
16 Age sensitivity, children, with children, should they
17 be protected because they might be three to five times
18 more sensitive than adults? Should we take that into
19 consideration in our regulations? And, finally,
20 should our regulations reflect us protecting the most
21 sensitive individual as opposed to the most exposed?

22 Dr. Puskin mentioned we've got statutes
23 that limit what we can do, and this is a big one right
24 here, Johnson Controls Act. In this particular
25 situation, Johnson Controls prevented women from

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1 working in areas where they could be exposed to lead,
2 and the rationale was is that if they became pregnant,
3 the embryo fetus might incorporate the lead, would
4 have developmental problems. You know, the are
5 workers sued basically contending that the woman had
6 the right to choose whether she wanted to work in that
7 environment and accept the economic benefits of
8 working there or protect the fetus, and the Supreme
9 Court sided with the woman's right to choose based on
10 Title VII of the Civil Rights Act.

11 So what impact does that have now if
12 there's a gender difference? Most likely none because
13 we're limited from doing anything.

14 Would we be able to also discriminate
15 based on age? Are older workers more sensitive than
16 younger workers? Steve Wing has expressed some
17 concerns about that. We may not have anything we can
18 do. That would be discrimination based on age now.
19 So there are going to be certain limitations that we
20 as an agency, we as a federal government can do
21 without changes in the statutes and court decisions.

22 So let's go back to our curve here where
23 we've nominally expressed some biological effect as
24 dose. On the solid line I've got what we believe are
25 the actual effects. We'll call it linear. And we've

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1 got this postulated linear extrapolation.

2 And just for exercise, let's say there's
3 a practical threshold. Now I've set this, if that's
4 a logscale, probably around 20 or 30 mSv. We heard
5 yesterday, Dr. Le Guen said that if there was a
6 practical threshold it might be between 10 and 60. So
7 for purposes of illustration, this could be fairly
8 close. What does that mean to NRC from a regulatory
9 standpoint?

10 Well, a practical threshold might say,
11 well, we've got efficient repair below that level.
12 Either efficient or maybe there are mechanisms, like
13 apoptosis, that can take care of air prone type of
14 situations, and above it we saturated the repair
15 processes or we've induced some sort of air prone
16 repair process.

17 What impact might that have on our
18 regulations? Well, as it was expressed earlier this
19 morning, we're going to have to consider what
20 exposures now do we have to monitor and record?

21 Right now we monitor and record the
22 occupational exposures. But what about differences in
23 background radiation? Clearly, if there's a practical
24 threshold, we're going to be concerned with monitoring
25 medical exposures for each of those workers.

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1 What about a frequent flyer? Should be
2 put the additional cosmic radiation exposure into that
3 to see whether or not we are below or above a
4 practical threshold? Is there a single threshold or
5 are there multiple practice thresholds? Do men and
6 women have the same practical threshold? Do children
7 have a different practical threshold? Are there other
8 groups in the population that could have a different
9 threshold? If there are different thresholds, now
10 which one do we regulate to?

11 Dr. Weiner, you were asking about the
12 fourth point there, that history exposure. Does it
13 fade? Is that an annual practical threshold or is a
14 lifetime practical threshold? If I receive a mrem
15 today, and a mrem next year, and mrem the third year,
16 is that a total of three years or a total of one? We
17 don't know.

18 Then the last point would be is, how do I
19 deal with different workers that have different
20 exposure histories? That is to say I have two
21 workers, one's above the practical threshold, one's
22 below. Do I try to not give any additional exposure
23 to the worker that's above the threshold and assign a
24 task to one below it, or not? Can I do that? How do
25 I regulate that?

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1 Well, let's go back, maybe do some case
2 study if you will with our practical threshold here.
3 First thing to keep in mind, and this comes out of a
4 case study back in 1975, that just because there's a
5 practical threshold or we have a lack of adverse
6 effects of any substance, it does not generally mean
7 that being below that threshold is safe.

8 Because of that, we're going to have to
9 regulate our non-threshold, or deterministic effect if
10 you will, with a series of safety factors. We see
11 this in ocular hazards, acoustic hazards, exposures to
12 heavy metals, exposures to organophosphates.

13 Safety factors, well, they can be a number
14 of things. First and foremost, what's the type of
15 data that we have in animals? Do we have consistent
16 information on rats, mice, dogs? If not, we have to
17 throw a safety factor in, anywhere from three to ten.
18 What about variation between humans? Again, in some
19 cases that'll be a variation of three to ten. How
20 confident were in the exposure? How confident were
21 you with the duration of exposure? Each of those
22 could have safety factors of ten. EPA, in fact, has
23 something on the order of I think six different
24 classes of safety factors to consider.

25 Note that when Dr. Puskin mentioned

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1 something about statutory authority to look at
2 practical thresholds, carcinogens are explicitly
3 excluded from consideration in the system. FDA, when
4 they're looking at food and drug, typically their
5 safety factors are anywhere from 200 to 2,000. In
6 1996 the Food Quality Protection Act set even tougher
7 standards for children. They said another safety
8 factor of ten would have to be put into this.

9 So what's that do with our curves? Well,
10 we could have a series of safety factors for just
11 illustrative purposes that might reduce our observable
12 concerns from let's say 100 mSv down to 1 mSv, or a
13 factor of maybe 20 or 30 below that practical
14 threshold.

15 Do we have sensitive groups we have to
16 deal with? And, finally, what about constraints?
17 We're talking about multiple sources now. We're not
18 talking about a single source of exposure.

19 The point I bring here is a practical
20 threshold may not necessarily give us any regulatory
21 relief. We're basically back in the same system where
22 we have right now.

23 This was a toxicity profile that was
24 conducted by the Agency for Toxic Substance and
25 Disease Registry. It was done in September of '99.

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1 This is required as part of CERCLA. And in that
2 assessment for ionizing radiation, they tried to
3 derive an estimate of what the minimum risk level
4 should be for ionizing radiation. The minimum risk
5 level would be is what type of radiation can you
6 receive on a daily basis so you won't have an adverse
7 effect.

8 The no-observable adverse effect level
9 they selected was 360 mrem/y, background radiation.
10 Now, why did they select it?

11 (1) It represents the U.S. population.
12 It's representative.

13 (2) It considers radon. This particular
14 level is not associated with an adverse effect. I
15 think everybody's pretty much in agreement there that
16 we don't think we have any adverse effects there, and
17 it is below some of the levels where we might see some
18 deterministic effects in the embryo fetus. They
19 corrected this value for an uncertainty factor of
20 three because of variability between individuals, and
21 with that they came up with an MRL of 100 mrem/y, or
22 in today's parlance 1 mSv, which is our public dose
23 limit.

24 Things they didn't consider, however, back
25 in '99 is, could the human variability be higher where

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1 we factor in gender differences? There is no
2 uncertainty factor considered for children, which has
3 been an issue, and it doesn't consider source
4 constraints. But what we might find is, is we've got
5 an MRL something less than 1 mSv/y potentially.

6 With all this in mind, what I'd like to
7 sum up with is a couple of statements. (1) Without a
8 doubt, it's my firm belief, it's a staff belief, our
9 regulations, our standards are adequately protecting
10 public health and safety. That does not necessarily
11 mean that we wouldn't be convinced that we need to
12 take a look at our regulations for consistency
13 purposes if nothing else.

14 Adoption of the new biokinetic models,
15 risk coefficients, and weighting factors will not
16 significantly improve public health and safety. We
17 mentioned this committee when we were looking through
18 the ICRP recommendations that was a bottom line, we're
19 adequately protected. Does that mean we would still
20 not do it? No.

21 For some of the other considerations I
22 mentioned earlier, the better science, we know that
23 we'll probably get some burden relief by just adopting
24 the ICRP 66 lung model. And on a case by case basis
25 we allow many of our nuclear fuel cycle licensees to

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1 do just that. So it's possible, especially if we want
2 to talk about consistency, getting EPA, OSHA who's
3 back in ICRP 2, DOE that's not going to ICRP 60, and
4 our Agency on the same thing, we'd consider that.

5 And for my standpoint, based on some of
6 the things that we've seen and where we're concerned,
7 we right now don't see any radical developments in the
8 science that are going to have a significant impact,
9 at least in the near future, on our regulations.

10 With that said, does that mean DOE should
11 not continue their program? No. We're firm advocates
12 of that, firm advocates of the EC program because this
13 is our basic research program that, even though they
14 might not have a near term practical application in
15 the regulatory community, there are other things that
16 might come out of these programs, a better
17 understanding of the cell and molecular biology that
18 might have applications in the clinic, and, as such,
19 I would firmly endorse continuing those programs.

20 Thank you.

21 CHAIRMAN RYAN: Thank you, Vince. Just a
22 quick question. Could you back up to your slide?
23 Let's see, one more. You know, I kind of focused on
24 360 because that number's been around for a long time,
25 and I recall last year's NCRP meeting when 360 may not

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1 be the best number to represent the background or
2 distribution of the various components of the
3 background. What would happen if it were 600, the
4 medical and radon and everything else being
5 considered? What do we do then?

6 DR. HOLAHAN: Well, keep in mind 360 is
7 the 1999 ATSDR number.

8 CHAIRMAN RYAN: Sure.

9 DR. HOLAHAN: So keeping that in mind.
10 Let's say we adjust it and we say that the background
11 is something higher because, obviously, 360, it
12 includes radon, it's industrial sources, other
13 commercial sources, and medical. And let us assume
14 that the medical goes to something on the order of 3,
15 3.2, 3.5 mSv, whatever the final number is going to
16 come out. So, yes, it's going to go up to 600 or
17 6 mSv a year. Fine.

18 Now the question I would have is, is they
19 used an uncertainty factor of three. Typically they
20 use ten. When we look at inner human variation in EPA
21 and FDA space, that's going to wipe out --

22 CHAIRMAN RYAN: But you could actually
23 argue the other way, that because of the NCRP report,
24 the uncertainty has perhaps been at least the same or
25 reduced by further update. I just throw that in to

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1 think that these numbers aren't necessarily fixed in
2 stone and they have a two-way impact. One is, what do
3 you with a different number, higher or lower, either
4 way? And then, you know, how does that factor into
5 any kind of derived standard or requirement that falls
6 out of that? So it can be a complicated question.

7 DR. HOLAHAN: The other issue that you're
8 going to run into is there are deterministic end
9 points, and one of the concerns in another analysis
10 would be reduction IQ. And if you look at a single
11 acute exposure of reduction IQ, we're down into the
12 several Sv level. So it's not going to be a whole lot
13 difference.

14 And, really, the point I have is I
15 wouldn't chase decimals on any of these discussions
16 here. It's just illustrative that our system of
17 radiological protection that we have right now, that
18 those limits that we've established, the optimization
19 in the ALARA programs that we've done, the constraints
20 that we have on some sources are protected, and if we
21 were to have a practical threshold, quite frankly, I
22 think we're going to end up in the same place we're
23 already at now.

24 CHAIRMAN RYAN: One other practical thing
25 I think in your next slide on harmonization that's

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1 important to think about is just within the NRC that's
2 everything from ICRP 2 to support reactor --

3 DR. HOLAHAN: Exactly.

4 CHAIRMAN RYAN: -- calculations right on
5 up to the ability to use the most recent
6 recommendations for models and those calculations, and
7 so forth, and then you mentioned a broader issue that
8 across other agencies is a wide variation of what
9 underpins various regulations, so that's a bigger
10 issue than just the NRC's.

11 Have you talked to other agencies at this
12 point? Do you have any insights about the inner
13 agency task force on what their thinking is?

14 DR. HOLAHAN: We actually brought this
15 topic up two weeks ago. We have an inner agency
16 steering committee subpanel report federal guidance
17 subcommittee and this is one of the topics that we
18 brought up. The question is is what is each agency
19 going to do, and, of course, I was specifically said
20 we are going to put NRC on the hot seat, and they
21 directed the question to me, and my response was
22 pretty much what I said about 30 minutes ago, pass me
23 an ear because we're going to have to bring this up to
24 the Commission and get Commission direction.

25 But, quite frankly, across the board, the

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1 other agencies are just starting to think about it.
2 Impacts as simple as we're not going to anything this
3 year because it's an election year. That was one of
4 the responses and that's just, welcome to D.C.

5 Unfortunately, the rule making processes,
6 they take time. We need for our agency to get
7 guidance from the Commission because, quite frankly,
8 we're talking about a huge investment financially in
9 technical basis. We're looking at Fed guidance 11,
10 Fed guidance 12, Fed guidance 13. Updating and
11 changing all of the annual limits on intake; derive
12 air concentrations, that's in appendix B; that's a lot
13 of work that has to be done and it's going to take
14 some contract dollars.

15 That, plus any time you manage that
16 program or get into rule making space, we're talking
17 full time equivalents and staff time. And, quite
18 frankly, none of this is budgeted in even our 2010
19 budget. And if we have a flat budget, the
20 Commission's going to have to make a decision, if we
21 put resources there, where are going to take resources
22 away from.

23 CHAIRMAN RYAN: If I could impose one more
24 second on your plan? You're actually going to produce
25 a Commission paper at the end of 2008?

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1 DR. HOLAHAN: At the end of 2008 a paper
2 will be prepared laying out a series of options with
3 resource requirements, costs if you will, for the
4 Commission to consider.

5 CHAIRMAN RYAN: And just for the folks
6 that might be interested, what would be the public
7 part of the process on reacting to anything you might
8 do or what the Commission might do? What does the
9 public have input?

10 DR. HOLAHAN: Well, the public will have
11 input on the actual rule making process because we'll
12 solicit information before an advanced proposal is
13 prepared. Public comments will be solicited. There
14 will be public meetings on the topic. Obviously,
15 we'll be going to the advisory committees looking for
16 their input, working with the other federal agencies.
17 Annually, they have a public meeting. I'm sure that
18 will be a topic of discussion there as well.

19 All of the proposals are put in the
20 Federal Register. Comments are solicited.
21 Undoubtedly, we will receive thousands of comments.
22 And, quite frankly, every one of those comments has to
23 be considered and reconciled.

24 CHAIRMAN RYAN: Right. I just wanted have
25 that kind of requirement and everybody here hear it as

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1 well. Thank you.

2 Other questions? Dr. Tenforde first.

3 DR. TENFORDE: Is ISCORS directly involved
4 in the inner agency dialogue or is that separate?

5 DR. HOLAHAN: ISCORS is the Interagency
6 Steering Committee on Radiation Standards --

7 DR. TENFORDE: Right.

8 DR. HOLAHAN: -- and it's membership
9 includes all of the federal agencies --

10 DR. TENFORDE: Right.

11 DR. HOLAHAN: -- to include OSTP, and we
12 have representatives on the federal guidance
13 subcommittee for all of those agencies that have
14 representation with radiation regulations.

15 DR. TENFORDE: So the inner agency
16 committee reflects the ISCORS composition was my
17 question. That wasn't so clear.

18 DR. HOLAHAN: Yes.

19 DR. TENFORDE: I think that's good, and,
20 at the same time, I've been a little discouraged and
21 I think others around the table have written on this
22 that there doesn't seem to be a constructive end point
23 to some of the inner agency dialogues, and I mentioned
24 yesterday one of our reports, which you didn't
25 mention, 146. I'm looking at the final

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1 decommissioning goals of EPA and NRC and I think
2 that's just one example of a number where a little
3 more harmony and constructive dialogue would really be
4 helpful because I do think things need to be looked
5 at, at least periodically, even if no changes are made
6 and I'm glad this is happening.

7 But I hope that the end goal will be to
8 make whatever changes seem appropriate in view of the
9 exposure to the public, as well as, of course, the
10 occupational setting. So I'd like your sense on that
11 subject.

12 CHAIRMAN RYAN: Allen?

13 DR. CROFF: Can you go back to your slide
14 8, please? If my math is correct, natural background
15 is on the order of 15, 20 rem, and you're showing the
16 region of regulatory interest being well less than
17 one. Maybe I don't understand the scale or something
18 about this graph.

19 DR. HOLAHAN: Here we're just talking
20 single exposures for all intents and purposes. I'm
21 not talking about cumulative background. I mean if
22 you want to think about it as such, this is the
23 discussion that was earlier this morning. That
24 biological effect isn't zero if you're talking about
25 a cumulative effect. You've got a mortality rate of

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1 20 to 21 percent. And, clearly, the dose here we're
2 talking about is that addition to background.

3 Background, if we're talking about the
4 lower LET is what, 1 mSv/y times 70 years. That
5 would, what, 7 mSv. Not 7 mSv, 70 mSv, 7 rem.

6 (Laughter.)

7 DR. HOLAHAN: Keep in mind, our
8 regulations, we have rem first and parenthetically we
9 have mSv. Thank you.

10 DR. CROFF: I guess I understand your
11 response. Let me just let it go at this point.

12 CHAIRMAN RYAN: Ken?

13 DR. MOSSMAN: Could you go to your slide
14 11? I've been interested for a little while on the
15 question of, do we need additional protections for
16 sensitive subpopulations? And it's really interesting
17 that the Commission has been at the forefront of this.

18 In fact, the Commission essentially
19 preempted the Supreme Court on this decision because
20 we are quite right that in the Johnsons Controls
21 decisions, essentially what the Supreme Court said was
22 it's up to the woman, and that's exactly what the
23 Commission says with regard to pregnancy. You know,
24 in other words, a pregnant woman can declare her
25 pregnancy; under those circumstances, the employer is

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1 obligated to provide additional engineering controls
2 or other kinds of controls, and there's a new dose
3 limit that's established for that person temporarily.

4 My question is this: if we think of the
5 pregnancy situation as just being a broad model for
6 sensitivity, then if we identify sensitive
7 subpopulations, and there have been estimates anywhere
8 between one percent and ten percent of the U.S.
9 population might be sensitive, that's a very, very
10 rough estimate, then could we adopt a pregnancy-type
11 model and allow workers to say to the employer, yes,
12 I am sensitive, and by doing so, then the employer
13 either educates the worker, assigns new positions,
14 establishes new engineering controls, whatever it is,
15 and just like we have for pregnancy, the worker could
16 also undeclare the sensitivity if they don't happen to
17 like what the employer is going to do for them, or
18 whatever? Are you looking at that, at the sensitivity
19 question that subpopulations in the pregnancy model at
20 all?

21 DR. HOLAHAN: It hasn't been discussed.
22 It's something I guess we could look at. But I guess
23 the question would be, from a simplicity purpose or
24 point of view, how many different standards do I want
25 to set for a worker?

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1 DR. MOSSMAN: Well, you already have the
2 pregnancy standard that you've set.

3 DR. HOLAHAN: But, again, that's
4 voluntary.

5 DR. MOSSMAN: Right.

6 DR. HOLAHAN: It's not required. That's
7 in this country. Now, if you go over to the European
8 Union, the fetus has the right of an individual --

9 DR. MOSSMAN: Right, right.

10 DR. HOLAHAN: -- and that fetus basically
11 is limited to 1 mSv during the term of the pregnancy
12 and there is no choice about voluntary, involuntary
13 declaration.

14 DR. MOSSMAN: I'm talking the U.S.

15 DR. HOLAHAN: And that's one of the
16 concerns or one of the problems we have with adopting
17 the BSS because of those type of considerations.

18 CHAIRMAN RYAN: Dr. Le Guen, do you want
19 to make a comment on that?

20 DR. LE GUEN: Well, we have an AEN meeting
21 on this topic and I sat during this topics, but it's
22 not my point of view. It's much more a Europe point
23 of view. No one should be discriminated by gender
24 characters. And when you have a good radiation
25 protection process, you must process the most

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1 sensitive. And as a second point, if you look, if you
2 remember yesterday what I mentioned about the dose
3 received by the nuclear workers, but also true about
4 ideologies. In fact, about nuclear war, the average
5 dose was 1.5 mSv. And for the moment we don't have
6 describe population. We are very sensitive for 1.1
7 mSv. But, you know, sometimes this is a rule. But
8 sometimes much more complex, the real life is much
9 more complex.

10 I have a story, as a physician, I remember
11 a few years ago one woman, she had breast cancer and
12 after five years she survived. And she asked me
13 because she wanted to work again, and she was in the
14 hospital and she was a technician for radiography, and
15 she said, well, I would like to work again. And the
16 occupational physician also called me and said, well,
17 I have trouble because I know about radiation, there's
18 a link between radiation and breast cancer. And so
19 what is the solution? And I told him, you know, she
20 survived after first cancer. If you said to her, you
21 cannot work, you will die again, so be careful about
22 that. And I say, well, can we have a work place
23 study? He said, yes, of course. So where the risk
24 is? In fact the risk is when she need to go in
25 emergency service close to the patient and you must

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1 make some radiography. But if she stay in the
2 department behind all protection, she receive no
3 radiation. So I say okay. So she can work, but she
4 will work only in the department and that's all.
5 That's why it's sometimes not so easy.

6 DR. MOSSMAN: No, no, I certainly didn't
7 mean to imply that it was easy. But, you know, in the
8 case of subpopulations, you may want to consider
9 alternative work environments simply because there is
10 some enhanced sensitivity. There's two ways you can
11 do that. (1) You can have different administrative
12 levels or you can just use some kind of average limit
13 as we are currently doing. There's any number of ways
14 of doing it, but it's an issue that's important. I
15 know that the Nuclear Energy Agency, I was on the
16 committee that Henri Metivier had shared and one of
17 the questions that surfaced was this whole notion of
18 how you deal with sensitive populations, and is it
19 something that we in the international radiation
20 protection community should be concerned about? Is
21 the current system protective of everybody?

22 And, again, it's a utilitarian philosophy
23 versus one in which, well, no we need to be very
24 specific about how we're going to deal with sensitive
25 subpopulations. So it's an ongoing debate, but it's

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1 very important.

2 DR. LE GUEN: Yes. About sensitive
3 population, I can one point. In France we are looking
4 for people who have cancer at the moment of treatment.
5 And, of course, we try to have different tests because
6 if they are sensitive to radiation, we will try to
7 have another kind of treatment, chemotherapy for
8 example, much more than the radiotherapy because we
9 are looking for the certain malignant cancer in case
10 of radiotherapy.

11 But so, all the time it's a problem of
12 dose and, of course, in case of sensitive population,
13 it exists but at very high dose. So you remember what
14 you say yesterday, you believe much more in ALARA
15 process, me too. In this case I think we need to
16 protect everybody and I think this is a most important
17 thing.

18 DR. MOSSMAN: I agree.

19 DR. HOLAHAN: What I would suggest that
20 you do is, if you're interested, we have a radiation
21 exposure information reporting system report that the
22 agency puts out on an annual basis. All of the NRC
23 licensees that report into this system we publish
24 exposures for each of several groups of individuals
25 and break out the ranges where we have the exposures.

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1 We find that with our ALARA system, on average, most
2 workers receive zero exposure.

3 Now, in that type of situation, if you're
4 using ALARA, you're optimizing the exposures, I'm not
5 sure what benefit in an occupational setting a
6 differential, multi-tiered system is going to have
7 because the exposures are so low. We're saying on
8 average most of these workers are received a mSv or
9 less, and that's the average. There are a few that
10 might exceed 2 mSv, but generally that's a fraction of
11 one percent; 99.some percent are below that. And
12 that's the value of, again, the optimization, the
13 ALARA programs that our licensees have because, quite
14 frankly, they want to keep, if nothing else for
15 litigious purposes, exposures as low as possible.

16 CHAIRMAN RYAN: That's a great way to
17 finish up, Vince. Thank you for a very informative
18 presentation, and Dr. Puskin, and all our presenters
19 today and yesterday.

20 I hope that after our lunch break, when we
21 reconvene at 1:00, we can have a rich panel
22 discussion. We'll start with that some question from
23 the members and we'll continue on from there.

24 Again, thank you all for participating in
25 what has been a real rich meeting today. Hopefully

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1 this afternoon will be even better. Thank you. See
2 you at 1:00.

3 (Whereupon, the foregoing matter
4 went off the record for lunch at
5 11:35 a.m. and went back on the
6 record at 1:06 p.m.)

7 CHAIRMAN RYAN: If I could get everybody
8 to take their seats, please, we'll come to order for
9 our afternoon sessions. We are scheduled for a panel
10 discussion and individual summaries by all of our
11 participants and questions from the committee members
12 and any other questions that might arise and that's
13 going to go on from 1:00 to 3:00.

14 I've had one request from Mr. Dennis
15 Nelson of the organization SERV to speak for about
16 five minutes and he will be --

17 (OTR comments)

18 CHAIRMAN RYAN: As others join the
19 conference call line, we'll have them announce
20 themselves when they do that, so please forgive any
21 interruptions. Dr. Mossman, you started us off
22 yesterday morning. How about starting us off now?
23 And let me set the stage, if I may. We started off
24 yesterday with Commissioner Lyons giving us his
25 interesting perspectives on an interest in this topic

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1 and I guess I'd ask all of you to think about what
2 advice or insights would you share with the Committee
3 as we think about what sort of a letter and what kind
4 of information we might want to convey to the
5 Commission and the Commissioners in particular.

6 DR. MOSSMAN: Thank you, Mr. Chairman. I
7 sort of summarized my comments yesterday and so I'll
8 just spend just a couple of minutes conveying my
9 thoughts about today. I was particularly grateful to
10 Professor Hammitt for taking time out from his busy
11 schedule to come join us and talk a little bit about
12 some of the economic perspectives which is a
13 perspective that I, for one, don't fully appreciate
14 but realize how very important it is in the grand
15 scheme about how we deal with the science.

16 You know, we'll be making some decisions
17 or perhaps, in the future there will be some decisions
18 about the nature of the dose response and whether we
19 should continue to use LNT as policy and part of that
20 is going to include the economic considerations and I
21 think Professor Hammitt's overview of some of the
22 basic principles on costs and benefits and the issues
23 about threshold and whether that's really relevant in
24 the end, I think, was very important, so I'm
25 particularly grateful for Professor Hammitt's

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1 perspective on that and I think that whatever we do we
2 need to consider that.

3 I was also very interested in the comments
4 by Dr. Puskin and Dr. Holahan, differing agencies, but
5 I think we all have the same kinds of issues in mind
6 about linear no-threshold theory and the underlying
7 radiobiology and what this particularly means.

8 At lunch today, I -- we had a very
9 interesting discussion on future directions and one of
10 the issues that we brought up that we might want to
11 explore later was, would it be useful for the
12 Commission to revisit the Below Regulatory Concern
13 policy, the BRC policy, that was, for lack of a better
14 word, a disaster back in 1988 and '89, primarily
15 because of a -- because it was not -- the concept
16 wasn't marketed well. And I think a lot of people in
17 the public had -- the general public had some concerns
18 about whether safety was being compromised by a BRC
19 kind of proposal.

20 The interesting thing is from my
21 perspective as a scientist, BRC is really on very
22 solid ground, the notion that there may be risks even
23 though they're non-zero risks nonetheless, they're so
24 low that they don't cause us any heartburn. They're
25 not anything that we should be concerned about with

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1 regard to public health protection and should we be
2 concerned about ratcheting -- or should we be
3 concerned about expending resources to very, very
4 small doses that, in fact, the incremental benefits
5 that you would be expected really aren't very real at
6 all.

7 So one of the things I'd like to see is a
8 revisit of that and maybe that's something that might
9 be considered for this letter that you want to write.

10 CHAIRMAN RYAN: That might actually be a
11 little bit beyond the scope of our information
12 gathering --

13 DR. MOSSMAN: Okay.

14 CHAIRMAN RYAN: -- for this session. So
15 that certainly could be something that could be
16 considered by somebody down the line but it would be
17 a little bit out of the wheelhouse of gathering
18 information on that topic for this letter.

19 DR. MOSSMAN: Okay.

20 CHAIRMAN RYAN: But I can clearly see it's
21 a logical extension of --

22 DR. MOSSMAN: My -- the reason why it's
23 brought up is the idea of risk communication, how you
24 frame risks, become very important and that was the
25 failure point, if you will, in the whole evolution of

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1 the BRC initiative.

2 CHAIRMAN RYAN: I understand that and I
3 think what our letter is going to focus on is the
4 appropriate and best way to communicate risk and to
5 characterize risk and to analyze risk.

6 DR. MOSSMAN: Right.

7 CHAIRMAN RYAN: And whether it's applied
8 to any one regulatory effort or another, I think that
9 our focus ought to be on the risk aspects that we've
10 heard this time but I appreciate your point.

11 DR. MOSSMAN: I understand. That's really
12 all I wanted to say.

13 CHAIRMAN RYAN: Okay, anybody else? Mary
14 Helen.

15 DR. BARCELLOS-HOFF: Well, I wanted to add
16 -- I thought it was very useful for me as a basic
17 scientist to hear how regulatory decisions are made
18 and the complexity for each agency. It leaves me a
19 little bit to wonder how relevant basic biology is,
20 but I think there is an underlying assumption that I'd
21 like to just bring out and that is essentially that we
22 know the basis for radiation's action as a carcinogen.

23 I think that's one of the underlying
24 assumptions and thus, you know, radiation is a
25 mutagen, a poor mutagen. I think that one of the

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1 considerations that the basic biology brings to the
2 table is not only the complexity of biological
3 responses but given that that complexity may well be
4 very much dependent upon dose, and that there may be
5 contributing factors at high dose that really augment
6 the carcinogenic potential of that mutagenic effect
7 and that's what we're really trying to bring to the
8 table, is that the non-targeted effects that we have
9 this kind of question, well, these are very
10 interesting biology but what does it mean to us, is
11 that that non-targeted -- those non-targeted processes
12 are the ones that more and more basic biology is
13 focusing on as really the drivers in carcinogenesis
14 and understanding then the dose dependence of those
15 non-target effects become critical to actually saying
16 not only do we have a regulatory model to evaluate
17 risk in a population but we have a good biological
18 understanding what that risk is due to.

19 I think that allows us to do something
20 that we haven't been able to do before and that is
21 actually think about susceptibility in a different
22 fashion and I can go on about that but I'm not going
23 to.

24 CHAIRMAN RYAN: Please do. I find this
25 part of our meeting fascinating because you know, as

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1 a physical scientist based person, you know, ergs per
2 gram is just fine and has been for a long time but you
3 know, I'm re-educated over the course of these two
4 days by the details that are so important, well,
5 that's not fine. I mean, it really is energy
6 deposited in what, where, how, when and next to what.

7 DR. BARCELLOS-HOFF: And the consequences
8 of the --

9 CHAIRMAN RYAN: And the consequences. So
10 I would appreciate you expanding on that a bit.

11 DR. BARCELLOS-HOFF: So I guess my -- the
12 thought I'm trying to convey here is that we have, for
13 example, in the presentation -- I'm sorry, I can't
14 read your name from this far away. Vince, and I'm
15 terrible with names as I demonstrated yesterday. It
16 was Peter O'Neal whose name I was trying to remember
17 yesterday.

18 So in one of your slides you had dose and
19 effect. It was one slide we went back to later and
20 there was the epidemiology and then there was cellular
21 molecular biology and then there was this line and one
22 of the things that the cellular molecular biology you
23 referred to was cytogenetic and clearly we can see
24 cytogenetic effect. But effects, like cytogenetics is
25 really an assay or really reflects dose and therefore,

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1 we think the effect is also associated to the risk and
2 the only -- the main point I'm trying to convey in the
3 biology is that risk is multi-faceted. The process of
4 carcinogenesis is multi-faceted and that what we're
5 really looking at is in cancer incidents is the
6 culmination of this. And that while it's true we see
7 very early effects and that we can track them linearly
8 with dose and there's absolutely no question that
9 there is a linear consequence of radiation exposure at
10 one level, which is generally DNA damage, and that it
11 does have a probability of causing mutation and that
12 mutation has a probability of contributing to
13 carcinogenic process, that it's really a more
14 complicated process and one of the things that allows
15 a tissue to develop a clinical cancer is perturbation
16 in all the other cell types that are not mediated by
17 mechanisms dealing with mutations.

18 And that's -- but it's a two-part problem.

19 I believe you have to have the genetic change in a
20 cell and that radiation is good at doing that, but I
21 also believe you have to have this perturbation of the
22 system that we referred to and that actually high dose
23 radiation is good at perturbing that system and that's
24 why it's good carcinogen at high doses.

25 But the question that remains is whether

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1 it perturbs the system at low doses and whether it
2 does it in a deleterious fashion. And that's my
3 assessment of the biology and so of cancer as a
4 process. And that's the part I don't see represented
5 when we talk about radiation effects being a damage
6 and then leap to carcinogenesis. There's a big leap
7 there and we see it over and over when we draw these
8 models and I know everybody -- I just wanted to bring
9 that up.

10 DR. MOSSMAN: Is this a merchant's
11 problem, I mean, you know, where you're looking at
12 individual cells and then extrapolating over to the
13 grosser pathology.

14 DR. LAND: Is there anything radiation
15 specific about the non-targeted effects?

16 DR. BARCELLOS-HOFF: Is there anything
17 radiation specific about the non-targeted effects?
18 No. Well, I'm afraid that my -- I don't know anything
19 but radiation. No, so I couldn't compare and contrast
20 it to like a chemical carcinogen. The experiment that
21 I showed you yesterday -- here I can, I can. Okay, so
22 here's a non-targeted effect, right?

23 The experiment I showed you yesterday,
24 where you have your mouse and you take out the
25 epithelium from the mammary gland and then you

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1 irradiate the mouse, right? This is the experiment I
2 did and then I transplanted in unirradiated epithelial
3 cells and showed that they readily went to cancer even
4 though the host had only been irradiated, right?

5 And that actually that was a very strong
6 effect, because I could see an increase in cancer, a
7 30 percent increase in cancer at 10 centigrade. Okay,
8 so that's acting on all of those other processes not
9 on mutagenic load in the target cells. So that
10 experiment has been done with two other chemical
11 carcinogens by colleagues of mine, one with NMU and
12 one with DNBA.

13 In the case of DNBA, in rats, DNBA in rats
14 or NMU in mice or vice versa, but anyway they're both
15 carcinogens of mammary gland. In the case of NMU,
16 there was no effect via the host. If you treated the
17 host, you didn't change NMU's carcinogenic potential
18 but in DNBA if you treated the host you almost -- it
19 was almost 100 percent of the cytogenetic potential.

20 So are there other agents that act through
21 additional processes than mutation? Yes. And there
22 are actually a lot of carcinogens that aren't very
23 good mutagens, asbestos. Asbestos actually acts
24 indirectly through the production of reactive oxygen
25 to generate mutations but not a direct mutagen.

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1 DR. MOSSMAN: Mary Helen, do you want to
2 comment on the school of thought that this
3 guesstimates derived from epidemiologic studies
4 already include consideration of non-targeted effects?
5 I mean, it would have to. Simply, is there anything
6 more -- I mean, so in terms of our understanding of
7 risk, if in fact, linearity holds and it is true, then
8 the risk estimates that we get primarily from studying
9 effects at high doses, say above 200 mSv, 20 rad, then
10 whatever influences, positive or negative, that
11 bystander effects would have and things like that are
12 already accounted for in the risk.

13 DR. BARCELLOS-HOFF: Well, that's true but
14 that's only true as far as the epidemiology shows an
15 effect.

16 DR. MOSSMAN: Right, right.

17 DR. BARCELLOS-HOFF: After that, you're
18 extrapolating based on underlying assumption that you
19 understand the mechanism and that the mechanism isn't
20 linear. And I have a slide. I don't know that we
21 have an AV person, and we don't have a chalkboard.
22 And actually, I'll talk about and try to present this
23 idea next week at the NCRP but if you think about that
24 linear component, and we say it's a two-compartment
25 problem that you have to have both compartments or

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1 both modes of actions, right, I talked about modes of
2 action yesterday. Both modes of action have to occur
3 in order to actually get that effect, that consequence
4 cancer.

5 So you're linear no-threshold, right,
6 that's targeted effect. So remember one of the
7 things about non-targeted effects is they tend to have
8 a step-function dose response. A very small dose will
9 elicit the response, a larger does doesn't increase it
10 considerably. It's not proportional to dose. It's
11 more like it's a biological process that turns on and
12 once it's on, it's on.

13 And so then it becomes a question, well,
14 at what dose does those other processes occur? And yo
15 could put your linear no-threshold. You could say,
16 okay, at 10 centigrade, see, I use a completely
17 different set of -- 10 rem, right, that's where it
18 turns on and anything below that all you're going to
19 have is that linear component and it's therefore, not
20 going to be as efficient as a carcinogen because all
21 you've got is the mutagenic potential.

22 And I think if you go to the chemical
23 toxicology literature, there's a lot of discussion
24 about modes of actions and how they intersect with
25 each other and how they change as a function of dose.

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1 But in radiation biology for some reason we kind of
2 left off that whole other effect that radiation really
3 has and that may well be acting in concert with the
4 mutagenic effect and we don't understand it.

5 So that's the -- I started off trying to
6 say what we tried to bring to the table is from the
7 science side is what we understand about the
8 biological processes and clearly we understand a lot
9 more about DNA damage than we did 25 years ago and we
10 have an exhaustive amount of information about the
11 mechanisms of damage repair and resolution and cell
12 type specificity and now I think we'd like to have
13 that equal depth of knowledge about these non-target
14 effects, changes in phenotype that have persist on
15 genomic instability. It's really a phenotype. It's
16 not a mutational -- it's not a train mechanism
17 frequencies consistent with a mechanism mutation.
18 It's a phenotype.

19 CHAIRMAN RYAN: So the next leg of this
20 chair is to kind of gather that all up at the cellular
21 and now we're going to talk about you know, groups of
22 cells and tissues and organs and organ systems and the
23 whole --

24 DR. BARCELLOS-HOFF: The systems biology
25 where we try then to compile all that information in

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1 that useful fashion that we begin to predict how -- so
2 when you get back to sensitivities of populations,
3 what I begin to -- what I find very interesting about
4 radiation cancers is that it's not -- there's nothing
5 unique about it. It's no different. You don't induce
6 a particular set of cancers. There's a susceptibility
7 inherent in the population. We seem to be augmenting
8 that susceptibility and whether that susceptibility is
9 lifestyle, in the case of the gastric cancers somebody
10 mentioned yesterday or is it a case of genetic
11 predisposition, it could be that you're actually
12 dealing with an accelerated -- well, you know, I don't
13 want to say that because it gets into very -- but in
14 breast cancer right now, there's a large effort in
15 understanding not only those very strong genotypes
16 that drive familial breast cancers like BfCR1 and 2
17 which only contribute to -- only account for what is
18 it, five percent of all breast cancers is familial; is
19 that right, Charles, something like that. But the
20 preponderance of breast cancers are actually due to
21 interactions between very weak polymorphisms so
22 there's -- but they're high frequencies so the BrCA1s
23 are very strong but they're very infrequent.

24 And then you have the genetic component
25 where you have a lot of weak high frequency

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1 polymorphisms and there's an argument right now that
2 a large proportion of those cancers that we distribute
3 across the population actually only occur in a very
4 small portion of the population. This is Bruce
5 Ponder's analysis of polymorphisms in the breast
6 cancer populations.

7 And I think that's an interesting idea
8 that we should consider in radiation protection is a
9 sensitive population, whether those cancers are really
10 occurring randomly throughout the population or really
11 in a very discrete set of individuals.

12 CHAIRMAN RYAN: Interesting. Thank you
13 very much. Jerry?

14 DR. PUSKIN: May I respond to that? Maybe
15 my take on it and you can respond to this. If it's
16 correct let's say that radiation causes mutations but
17 then it also causes other things and these other
18 things are necessary in order to get a cancer from
19 this mutation, it would seem like a threshold, a real
20 threshold you're in likely because we already know
21 that whatever processes convert a mutation into a
22 cancer are already occurring in the body without any
23 extra radiation, people get cancer. So if all these
24 cancers kind of rise out of these mutations. So
25 wouldn't that argue that yes, the dose response could

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1 be non-linear below where we can see the epidemiology,
2 sort of the question that Dr. Mossman asked but it
3 could be very non-linear because the relative
4 importance of these different processes, the effect of
5 radiation on these processes could be very different
6 low doses than they are at higher doses. So you might
7 get something that doesn't look like a linear dose
8 response but you still -- radiation should still be
9 able to cause some cancers.

10 Now, you would say --

11 CHAIRMAN RYAN: I think the secret there
12 is some, you know, but not all.

13 DR. PUSKIN: That's right, that's right.

14 CHAIRMAN RYAN: So that's a little bit of
15 a confounder there.

16 DR. BARCELLOS-HOFF: And so you could have
17 two parallel curves with a drop in between, right?
18 And so then my question is, yes, there's -- the linear
19 component will always give some kind -- we did talk
20 about this concept of negligible and at some point it
21 does become negligible in a body of, you know, 14
22 cells, that one mutation and one randomly hit cell.

23 DR. PUSKIN: Or you can prevent some
24 cancers, you know, and that sort of thing.

25 DR. BARCELLOS-HOFF: And one of the ideas

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1 that comes out of thinking about it, is that you can
2 actually begin to think of ways of reducing the
3 carcinogenic potential of radiation which you can't do
4 with mutations because you don't know what your
5 mutation is. You can't come in and target your p53 or
6 your, you know, whatever, ETFR. It's hit and you
7 don't know what it's going to be but these other
8 processes actually do lead you to other strategies for
9 thinking about carcinogenic risk and it's
10 inevitability.

11 DR. LE GUEN: We must keep in mind that if
12 we observe cancer due to the edge, it's do to an
13 accumulation of mutation due to the edge and in fact,
14 at high dose we accelerate the process and that's why
15 you know, of course, that after high exposure you have
16 a risk of cancer not next year after the exposure but
17 15 -- an average of 15 years after high exposure.

18 It's only time -- the need, time to need -
19 - no, the need to have a second mutation and to have
20 a process and in fact, for us to -- the first exposure
21 is the beginning of the process, this is a first step
22 but you need to have other steps before to have the
23 cancer, the tumor and for sort of tumor it's between
24 10 and 15 years.

25 And that's why in fact, I wanted to say

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1 this morning we talked about the different non-
2 targeted effect but from my point of view, it's not
3 good to try to compare one non-targeted with another
4 and say this one is good for the body, this one is not
5 good. This is a war reaction of the body and we must
6 take this reaction as a war and particularly, you
7 know, today we know that cells react at very low dose.
8 This is a reaction and it's not a problem. And for
9 people to say, "Well, of we observe a reaction, it's
10 bad".

11 No. We live under stress and if we are
12 not a reaction of a cell we die. And in fact, this is
13 a reaction and this is normal reaction. Yesterday I
14 said about the evolution and probably because now at
15 this dose we have a lot of different stress. Today we
16 talk about raising radiation, but we must take into
17 account also the other stress. That's why about
18 education on the seven point, I full agree with Ken
19 and also Vincent who says this morning that we must
20 think about which kind of communication we must have
21 with the population.

22 And if we are talking about risk, we must
23 talk about all the genetic toxic agent because if we
24 want to focus only on one, it's not fair because we
25 live with other stress and to -- the body is a

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1 marvelous device because if we can live under stress
2 it's because we have different mechanisms. The
3 problem is and when you begin to have trouble when we
4 begin to be on the way of the cancer and that's the
5 difficulties that we have. But to have a -- to
6 observe a reaction at low dose, I think it's not bad.
7 It's normal.

8 CHAIRMAN RYAN: One of the things that,
9 you know, in this whole issue of, you know,
10 accumulating dose and thinking about the natural
11 background and then workplace exposure, there's one
12 part we really haven't talked about and I'd be happy
13 to have any insights, and that's medical exposure.
14 Medical exposure is usually given compared to the
15 workplace or compared to the natural environment, a
16 very high dose rates relatively speaking in very short
17 bursts. So I'm not so sure, you know, fluoroscopy can
18 be 10s or even 100 centigrade over, you know,
19 typically, you know, major portions of the body.

20 How do we account for what is -- what NCRP
21 has reported last year and hopefully will publish soon
22 an increasing population of folks, now I know not
23 everybody gets, you know, the same level of medical
24 care. Certainly nuclear workers get a level of
25 medical care that's appropriate for good health and

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1 all of that, but how do we deal with that now
2 significant component of what is typically ignored as
3 part of their background exposure?

4 DR. BARCELLOS-HOFF: I was actually very
5 struck by that comment. Essentially, isn't it doubled
6 almost.

7 CHAIRMAN RYAN: Yeah, it's more than that
8 actually.

9 DR. BARCELLOS-HOFF: Yeah, I mean and so
10 I'd characterize it as a schizophrenia, right, because
11 on the one hand we regulate to incredibly small doses.
12 On the other hand there's no regulatory checks other
13 than, you know advisor decision --

14 CHAIRMAN RYAN: And again, I'm asking this
15 question about the radiation biology and how that
16 would flow into the epidemiology. I realize people
17 judge medical exposure differently than they would
18 workplace and background. I'd just like to leave that
19 on the side.

20 DR. BARCELLOS-HOFF: Well, how can you
21 treat it differently?

22 CHAIRMAN RYAN: Well, I mean, very often
23 it's not recorded or known and yet it's double the
24 background if not more in some cases. Some folks have
25 lots and lots of exposure. Some have very little and

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1 some are in this kind of average condition but there's
2 a large fraction of folks who get up into the 50s that
3 have cardiac scans and all the rest. You know, those
4 could be up in the near 100 rad.

5 DR. BARCELLOS-HOFF: Well, my colleague at
6 DOE always asked the question about the RERF data set
7 and how the population there has been very carefully
8 monitored with radiation and how that doesn't -- that
9 piece of information isn't part of the dose exposures
10 or the cumulative dose is not included in that.

11 CHAIRMAN RYAN: Can you, Tom, talk a
12 little bit about what the NCRP is finding in this area
13 in terms of the numbers?

14 DR. TENFORDE: Yes, actually we will be
15 soon putting the draft of the Committee report on our
16 website and that will be publicly available at that
17 point and it will undergo then formal council review.
18 It's about to undergo expert panel review, which we do
19 before the council review but in brief, the average
20 medical exposure per annum for an individual in the
21 United States has increased from about 50 millirem in
22 the early 1980s to a little more than 300 millirems in
23 2006, a six-fold increase, which is very substantial.

24 So now in looking at the total exposure
25 with average values for terrestrial, cosmic, internal

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1 body, radon, and minor contributions from
2 occupational, et cetera, adding medical you're up to
3 about something like 6.2 mSv per year. About twice
4 what it was at the time Report 65 was published in
5 1987.

6 Now, this introduces in my mind a lot of
7 very interesting questions and complications. When we
8 were talking about average exposures, let's say 20
9 years ago, we were talking about roughly 300 millirem
10 of which nearly all was chronic exposure, very low
11 rates, like a millirem a day. Now, we're suddenly
12 looking at a background exposure including medical,
13 where about half of the exposure consists of acute
14 exposures to fairly significant, non-trivial doses at
15 higher dose rate, much higher dose rate.

16 So given the fact that a lot of
17 regulations are built around the idea that exposures
18 are chronic at low dose rates, how do you now compare
19 those regulatory guidelines with the current, if you
20 will, total average amount received by US -- a member
21 of the US population? This is true, by the way, in
22 Europe, Japan and a number of other countries, having
23 looked at this --

24 CHAIRMAN RYAN: And if we pick up on
25 Vince's point that the large fraction of the workforce

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1 has, you know, low cumulative doses, you know, it
2 really boils down to even in the nuclear workforce,
3 it's really the medical exposure is in excess and at
4 the higher dose rates than the work exposure.

5 DR. TENFORDE: Right, and the issue, where
6 I was headed on that is that you now have the
7 complexity of comparing low chronic doses delivered at
8 low dose rates with a much higher average annual, if
9 you will, background, including medical --

10 CHAIRMAN RYAN: Right.

11 DR. TENFORDE: -- for the population and
12 half of which is delivered at a much higher dose rate.

13 CHAIRMAN RYAN: And in small bits or in
14 bits across --

15 DR. TENFORDE: Yeah. And I don't -- this
16 is a very complex issue. In regulatory circles
17 typically, in the past, medical has been set aside,
18 the idea being that this is a beneficial use of
19 radiation and you really need to look at health
20 benefits versus the risk of having radiation
21 administered for medical uses and you know, we've
22 tended to ignore that but the level of medical
23 exposure now is reaching a point where I'm not sure it
24 should be ignored in terms of public or occupational
25 exposures.

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1 CHAIRMAN RYAN: And just to take the point
2 -- and I don't disagree that that premise is a valid
3 one to think through but the fact that there's now
4 these episodic exposures that are significant compared
5 to the chronic exposure from what we've learned about,
6 you know, these more sophisticated ways to think about
7 the biology, it would seem that the biology could be
8 confounded by these short higher dose rate exposures
9 as well as you know, the question of is there a
10 question of appropriate, you know, requirements for
11 control, et cetera. So am I right there, that that
12 could be a confounder?

13 DR. BARCELLOS-HOFF: But it would also be
14 compounded by, except in the whole body CT scans, you
15 have very localized radiation and one of the things,
16 I just don't know how to extrapolate is, is whether --
17 we were talking about this over lunch, your colomated
18 (phonetic) tumor would elicit an immune response,
19 right, even though it was a local volume that was
20 irradiated, but, you know, volumes irradiated also
21 might impact this.

22 CHAIRMAN RYAN: Oh, sure.

23 DR. MOSSMAN: Mike, if I could add --

24 CHAIRMAN RYAN: Yes.

25 DR. MOSSMAN: -- you know, this problem

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1 with medical exposures and the high doses isn't
2 anything new. You can go back better than 20 years
3 and the American College of Radiology and other groups
4 fully recognized even back then that doses were very
5 high for many of these procedures. The problem became
6 very acute within the last four or five years when it
7 was recognized that you had this tremendous increase
8 in number of examinations that were done from three
9 million CT scans in the early 1980s to over 60 million
10 today and so that's the fundamental problem.

11 It might behoove the Advisory Committee to
12 look at the paper that Amos, et al., published in the
13 Journal of the American College of Radiology back in
14 May or June of last year in which they set up a whole
15 structure of dose reduction, the kinds of issues that
16 they needed to look at that included unnecessary
17 repeat examinations, partnerships between patients,
18 physicians, insurance companies, that were major
19 drivers in elevating the dose.

20 I mean, there are all sorts of stories
21 about a patient going to his primary care physician.
22 The primary care physician orders a CT exam of the
23 abdomen. That study is done. The patient is then
24 triaged to a gastroenterologist specialist. The
25 gastroenterologist specialist within two weeks does

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1 the exact same exam all over again and why are they
2 doing it, well, in the name of ligation, in the name
3 of whatever philosophy of patient care that they have
4 but it's those kinds of problems.

5 From a personal standpoint, I think the
6 driver in all of this is not so much the public health
7 impact of the increased radiation dose, but the
8 medical costs. I mean, I think that the major issue
9 is the tremendous costs of doing these CT
10 examinations, but if you look at the ACR White Paper,
11 they have a well-thought out strategy about how to
12 deal with what is ultimately a dose reduction problem.
13 How do you eliminate unnecessary x-rays things of that
14 nature.

15 CHAIRMAN RYAN: And I appreciate those
16 additional, you know, areas of interest and concern,
17 but again, I'm trying to narrow our --

18 DR. MOSSMAN: No, no, but in terms of
19 where we're going in terms of it's a dose problem from
20 a radiation protection standpoint, it's how you
21 eliminate the dose and there's all sorts of reasons
22 why you have the high dose.

23 CHAIRMAN RYAN: Right. No, I appreciate
24 that and not all just because it's more. I mean, I
25 understand. Thank you.

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1 DR. LE GUEN: I agree, just to moderate
2 but all is true and I agree with everything but one of
3 the increase is also due to the aging of the US
4 population and because in the modern democracy in
5 Europe and in US we have trouble that we have an
6 aging, an important aging of the population. And of
7 course, if you increase the aging, you increase the
8 number of medical examinations and that's why if we
9 are talking about -- as the problem yesterday I
10 mentioned that from my point of view, it's very
11 important to focus on the most sensitive population,
12 so children, pregnant women and so on, much more than
13 other all population because if you are 80 years old
14 or 75 years old, it's not a problem if you have two CT
15 scans but if you are younger, yes, of course, it's
16 much more interesting to take into account.

17 CHAIRMAN RYAN: It would be interesting to
18 try and figure out how many nuclear workers or
19 radiation workers have medical exposure that exceeds
20 their workplace exposure.

21 DR. LE GUEN: Yeah, yeah, you're right.

22 DR. HOLAHAN: Well, I think that
23 information might be available. One of the things hat
24 we haven't seen yet because the report is not out, is
25 with CT demographics. And it's pretty much equal

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1 across the board. The children under age 10 are
2 getting as many CT scans as the geriatric cases in
3 their 70s and 80s.

4 DR. TENFORDE: Yeah, actually, I have a
5 slide that shows the distribution. It does -- it's
6 sort of bell-shaped with a peak in the 50s, age-wise
7 but it's not a, you know, it's not a huge drop-off
8 between the very young and the very old. It's a very
9 understandable peak because people begin to develop
10 health problems that require nuclear cardiology and CT
11 exams in their late middle age and as they get older,
12 either the problem is cured or they die, you know, or
13 their judged not to be curable. So they don't get
14 more and more exams.

15 So that's the explanation of the curve, I
16 think.

17 DR. HOLAHAN: But the issue that I'd go to
18 is those children are also the most sensitive. All
19 you have to do is look at the life span study and the
20 children under three and five are much more sensitive
21 than somebody radiated in their 30s or their 50s and
22 what's going to be interesting to see what happens to
23 those kids 50, 60, 70 years from now, because if you
24 look at the life span study, when did most of the
25 solid cancer start showing up? It's only been that

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1 last 10 or 20 years.

2 That is to say, it was the folks that were
3 exposed under age 20 at Hiroshima and Nagasaki, so
4 that age dependence is going to be very important.

5 DR. MOSSMAN: But you know, in that
6 regard, though, the Oxford Childhood Cancer Survey is
7 very -- is very instructive because one of the issues
8 in trying to understand the nature of causality was
9 asking the question, what was the medical reason for
10 the woman to have the exam to begin with. And did
11 that medical status or risk of disease have any impact
12 on the risk calculations?

13 We can ask the same questions here with
14 regard to CT exposure of children. Why are they
15 having the examinations?

16 DR. HOLAHAN: Traumatic injury. I mean,
17 traumatic injury won't necessarily be disease.

18 DR. MOSSMAN: And -- it may not be, but we
19 don't know. I mean, we just -- we don't know whether
20 it's some kind of chronic illness. We don't know if
21 it's, you know, and appendicitis or something like
22 that. Sure you might say that it's an isolated
23 disease, we don't have a problem but we just don't
24 know and all I'm saying is that it's -- that kind of
25 concern complicates the interpretation of the data in

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1 trying to attach some kind of public health impact to
2 CT exams of children and you know, that's the only
3 reason I bring it up is that those kind of issues have
4 been brought up before and it makes the interpretation
5 difficult.

6 CHAIRMAN RYAN: Jerry.

7 DR. PUSKIN: Along those lines, another
8 concern is CT scans of infants and that often happens
9 if there's problems, spinal fluid and so forth.
10 There's -- there was a study done by a Swedish group
11 Herr Hall and others that showed that infants who are
12 radiated for birthmarks on their face that years
13 later, it turned out they had lower cognitive ability
14 than controls and the doses weren't that much higher
15 than typical head CT scans.

16 You know, the total dose was around six
17 rad. You know, if you get a series of three CT scans
18 to the head, you're in that same range. So that's
19 certainly another concern.

20 DR. LAND: Also true of the tinea Capitis
21 patients.

22 DR. MOSSMAN: Reduced?

23 CHAIRMAN RYAN: Well, it's a dimension I
24 think we've kind of heard a number of, you know,
25 examples of the studies that address this idea that

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1 medical exposure is certainly increasing and certainly
2 there's some evidence that say that's part of the
3 overall radiation risk profile for workers or others
4 and as well as background and workplace exposure.
5 That's an interesting observation.

6 DR. PUSKIN: This is off of medical.

7 CHAIRMAN RYAN: Please change the subject.
8 That's fine.

9 DR. PUSKIN: I just wanted to sort of make
10 a final few points along the lines that I made. First
11 of all, I would second what Dr. Holahan said, that you
12 know, that aside even from the question of radiation
13 risk, that we certainly second the support for the low
14 dose program at DOE. I think there are very
15 interesting things coming out of there that I think
16 will have wide implications in terms of understanding
17 carcinogenesis and biology in general. And also we're
18 interested, very glad that DOE and NCI are supporting
19 the Techa River study and other studies of chronically
20 radiated cohorts.

21 What I've seen here though is that we have
22 these effects, these low dose effects and undoubtedly
23 they are real in some systems at some doses and so
24 forth but what we don't really know is do they have
25 any significant effect on the US and I think that's

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1 really what drove BEIR VII. In Committee they said,
2 yeah, these effects occur but any effect on the reask
3 at this point is highly speculative.

4 You know, in looking at it, we don't see
5 why it would necessarily have a big effect and given
6 also that as far down as we can look, there's no
7 indication of a strong deviation from LNT. And as I
8 tried to bring out, I think we're going down pretty
9 far. You know, it's true, it's not as far as we need
10 to go, but and we don't see that.

11 So right now, I think the effect on risk
12 is at least highly speculative and given that, I don't
13 think there's really an alternative to LNT either for
14 risk assessment and especially, I think Dr. Holahan
15 made the point stronger than I did but on regulation.
16 That we're really not going to be able to relax the
17 risk estimates in the -- or relax regulations based on
18 these kinds of studies any time really soon.

19 And I guess that's really what I was --

20 DR. BARCELLOS-HOFF: And good I just add
21 as the biologist here --

22 CHAIRMAN RYAN: Yes, please.

23 DR. BARCELLOS-HOFF: -- that as a citizen,
24 I hope you don't. The precautionary appearance of
25 ALARA all those things hold. What we're trying on the

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1 basic biology side is really to understand radiation's
2 action as a carcinogen. It is the only known human
3 carcinogen that we have to understand this process
4 better. I often speak to cancer biologists who go in
5 and mutate that and then make a mouse that's all
6 mutated or you know, and say this oncogene drives all
7 of carcinogenesis and I say, "But does that tell you
8 anything about spontaneous cancer or does it tell you
9 about exposures in terms of how we think about human
10 populations". And it's very hard to get them to come
11 to that, you know, "Oh, well, radiation is spontaneous
12 DNA damage, it would cause this mutation one out of
13 10^{14} times, you know.

14 And you could do those calculations. So
15 it's really important just to understand that
16 radiation is very interesting as a biological -- in
17 terms of the biology it elicits. And what we're
18 trying to understand better is, is that biology and
19 you're absolutely right, some of these effects may be
20 just that, effects, transient. And one of the goals
21 if the DOE program is to make sure that people try to
22 take that biology and link -- make the next linkage
23 which is does that effect have a consequence that fits
24 into this model of cancer?

25 And it's easy to do with DNA damage. But

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1 it's going to be much easier to do in the next 10
2 years with the biology that's coming out now to link
3 all these so-called non-targeted effects. I just want
4 the radiation biology community to be aware of them
5 and to be thinking about how that might impact the way
6 they consider radiation's action as a carcinogen. But
7 it's actually true, we're not done.

8 DR. MOSSMAN: Mary Helen, do you see in
9 the future moving away from cellular radiobiology
10 studies all together and focusing on tissue and organ
11 effects in a system biology approach recognizing as --
12 we see that cellular effects are fine but they are
13 very limited in terms of what it is that they can tell
14 us about cancer as a tissue and as a multi-cellular
15 organism phenomenon. Do you see a general shift in
16 the kinds of models that you will be using that --

17 DR. BARCELLOS-HOFF: That's one of the big
18 emphasis in the DOE program against a fair amount of
19 resistance if a portion of the radiobiology community
20 because it is easier to look at things that you can
21 have a flat on a dish, you know. There's a lot of
22 technical advantages to that when you're trying to
23 control variables.

24 As we get into more complicated models
25 it's more difficult to control variables and to

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1 attribute. And actually, you know, it's very hard in
2 the United States right now and I think even worse in
3 Europe to do an animal study.

4 DR. MOSSMAN: Yeah, that's true.

5 DR. BARCELLOS-HOFF: But it's -- you know,
6 to put all those pieces together, I think requires a
7 slightly different framework that we brought up
8 earlier.

9 DR. MOSSMAN: Right, right.

10 DR. LE GUEN: If we have a -- just to
11 complete because that's an interesting point. I
12 believe in that. You know, if you have a look on the
13 story, during the '60s I was too young but a research
14 was -- worked on the protein and after the discovery
15 of the molecular biology and we begin to work on the
16 genome, and after the genome, perhaps it's interesting
17 to look on the function of the genome, so we have the
18 transfetom (phonetic).

19 Now, we are talking about proteinic so
20 about the protein again, because it's only a part of
21 the answer, the gene. After it's very important to
22 have the function into the cell and after into the
23 cell, into the tissue and into the body. And we have
24 a lot of disease about that just -- I don't know who
25 I was talking to yesterday about that to say, when we

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1 have a higher radiation, we don't --

2 DR. BARCELLOS-HOFF: We were talking about
3 that, multi-organ failure.

4 DR. LE GUEN: Yeah, absolutely. In fact,
5 this is a reaction, this is a reaction of the body.
6 We die at the end due to an important inflammation.
7 And the reaction is too strong and we know that.
8 That's very important after we observe physical
9 evidence but yesterday I say it's important to know
10 what will be the outcome, what will be the
11 consequence. And as a consequence we must take into
12 account the tissue reaction and the body reaction.

13 So that's very important to all of this.
14 And one of the problem, and I full agree with you Mary
15 Helen, it's that today it's very hard to work on
16 animals, that's true. And you remember yesterday I
17 mentioned that it's very hard to extrapolate from a
18 model to the body because we miss something and of
19 course, it's very important to have this link between
20 the observation and the consequences as label, in 3-D
21 in the body, not only in vitro experiment.

22 DR. TENFORDE: Let me add one thing, I
23 don't know whether this has been said yet or not but
24 in my own mind, the very important research that's
25 being done with low dose radiation effects to me it is

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1 important for more than one reason, more than just
2 understanding low dose effects in the context of
3 policy, practices and regulations. To me it's basic
4 science that will undoubtedly eventually pay off in
5 terms of medicine. I think there's no question about
6 that.

7 We know that localized insults to tissue
8 propagate. I mean, this has been known for many
9 years, I mean, in terms like abscopal effects, you
10 know, and that the more we understand about response
11 of integrated tissues to localized radiation effects,
12 the more we will be able to put that knowledge to work
13 in terms of treating disease not only at the tissue
14 level but you know, a major issue that's still being
15 dealt with, we deal with it at NCRP and ICRU as well,
16 is what happens outside the treatment volume because
17 we know there is scattered radiation and there are
18 certain norms for how much that can be for various
19 types of radiation and we know that this is an
20 appreciable amount of radiation compared to the amount
21 that people are getting from natural background or
22 other sources.

23 So I think that a lot of this basic
24 knowledge will ultimately translate into the medical
25 arena and lead to some enlightened decisions on either

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1 proactively or retrospectively treating secondary
2 effects of disease or treatment of disease.

3 DR. MOSSMAN: I wanted to add that --

4 CHAIRMAN RYAN: Vince, did we skip over
5 you, Vince? Did you have --

6 DR. MOSSMAN: Yeah, I need to leave and I
7 just wanted to make one comment --

8 CHAIRMAN RYAN: Oh, please, okay, all
9 right, sure.

10 DR. MOSSMAN: -- on Dr. Tenforde's
11 comment. I agree with you 100 percent. I think that
12 the more we get to know about a system or systems and
13 understand their behaviors, the better off we are in
14 managing it. But on the flip side of the coin, it's
15 interesting to note that historically all of the major
16 treatment strategies for radiotherapy in cancer back
17 in the 1910s, 1920s, 1930s were done and understood
18 and in place before we ever understood the concept of
19 radiation repair or anything like that.

20 We learned about fractionation and all of
21 that stuff and the benefits of doing that before we
22 ever understood one single thing about cellular basis
23 of ionizing radiation repair and the like. So the
24 flip side is interesting but I concur with you 100
25 percent that we need to learn more about these things

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1 in order to be able to develop new therapies like
2 clevat (phonetic) for the treatment of CML that only
3 came about because of findings in molecular biology in
4 the nature of the ABL oncogene and things like that.
5 I mean, I think that that was absolutely critical and
6 is a perfect example. And with that, I excuse -- I
7 need to excuse myself, Mr. Chairman. Thank you.

8 CHAIRMAN RYAN: Thank you very much.

9 DR. MOSSMAN: Good to see everyone, thank
10 you.

11 CHAIRMAN RYAN: Vince?

12 DR. HOLAHAN: I guess my thought might be
13 to Mary Helen and actually Dr. Mossman is we have to
14 be very careful with the information technology and
15 availability of information. That is to say many of
16 the young investigators know the internet and nothing
17 else. And here's my point; back in the '60s and '70s
18 Al Klein (phonetic) was doing experiments in sub-
19 lethal damage repair and potentially lethal damage
20 repair.

21 That's not a new phenomenon. I mean, we
22 knew going back to your four R's of radiotherapy,
23 there is going to be repair, repopulation,
24 reoxygenation, redistribution. Much of this is where
25 we got our tissue, much of this is where we get our

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1 DDRF. And I would go back to the French National
2 Report. That was all discounted. It's there. It's
3 nothing new. The BEIR VII report acknowledged that,
4 yet the French Academy Report pounded them on that
5 issue there.

6 We know that there are single strand
7 breaks in every cell. It occurs daily. You cannot
8 transcribe and translate information unless you break
9 the DNA, unwind it, transcribe it, wind it back up and
10 like it. It goes on daily. You indicated that there
11 was no repair at the very low dose but you said
12 yourself there's eight double strand breaks a day in
13 every cell. It's metabolic damage depending on the
14 proximity those can be realigned.

15 You've got non-homologous end joining
16 techniques that can repair them but it might be error
17 prone. But this isn't new, so I would caution you
18 that we've known that different tissues have different
19 sensitivities to radiation. Rapidly population
20 tissues are more sensitive than slowly dividing
21 populations. We know that there aren't 10^{14} sensitive
22 cells. Many of those are internally differentiated,
23 subject to cancer but we hear these things. I mean,
24 I've heard 10^{14} unfortunately at least three times in
25 the last hour, that's not the case. Not every cell is

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1 going to be --

2 DR. BARCELLOS-HOFF: But in terms of the
3 initial events, those 10^{14} cells get the same thing,
4 and I'm just using it to emphasize that there's a lot
5 of biology.

6 DR. HOLAHAN: We sit there, we radiate the
7 liver. We have liver functions. If the cells don't
8 divide, you could have all sorts of double strand
9 breaks but you haven't lost any genetic material.
10 Partial hepatectomy, sure you brought that up. What
11 happens? We express that damage, the organ falls
12 apart.

13 We also know that the immune surveillance
14 we talked about yesterday, that again isn't new
15 either. We go to that palpable one centimeter tumor,
16 10^7 cells. The first thing we do in a radiobiology
17 course, we sit there and say, "Given the slope of the
18 radiation survival curve the D sub not, how many Gys
19 of radiation do we have to kill to sterilize that
20 cell"? We're talking 35 Gy? Can't do that in a
21 single exposure because we destroy the normal tissues,
22 so we fractionate it.

23 Dr. Mossman said, five fractions, two Gy,
24 six weeks, do we sterilize the cell? No, we've got
25 10^4 , 10^5 , 10^6 cells still there but it's the normal

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1 immune suppression that keeps it in check, ergo we
2 have basically got cancer survivors that are in
3 remission. And we hope the immune system keeps it in
4 check unless it emerges again.

5 So go back to Hall's book, make sure these
6 kids read this stuff. They're not going to see it on
7 line because too often what we find is we're using new
8 techniques to do that same thing over again. Back in
9 my day we looked at single strand breaks, you gave,
10 you know thousands of rads because the techniques
11 weren't sensitive enough to detect anything else other
12 than that.

13 Now, gee, you know, we don't use BUDR to
14 look at exchanges. We've got these great probes,
15 antibody probes, beautiful band-aid techniques, much
16 more sensitive and that's where the excitement is
17 going to be, looking at many of the same problems we
18 used to look at 20, 30, 40 years ago, with the new
19 techniques. And I say, DOE keep pushing on that
20 because we'll get a much better understand.

21 DR. BARCELLOS-HOFF: Well, you can't see
22 this probably from the other side there, but this is
23 my systems biology slide for the old -- you know, what
24 is systems biology? It's linking physiology, cell
25 biology and molecular biology. It's what we used to

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1 call -- physiology is what I want people to think
2 about in terms of radiation biology because we've been
3 down here for so long that we have forgotten all these
4 other levels exist and so my next slide is the
5 oxygenation, repopulation and repair. They're exactly
6 the same levels of organization and that's what I was
7 saying yesterday, radiation biology actually deserves
8 a round of applause. We've always been systems
9 biologists. We've always considered all the way from
10 the molecular to the physiological response to
11 radiation but it's so hard to get people like you say,
12 to move out of their particular box, their favorite
13 Google window and think about what actually is
14 occurring. Did I show you that? Yeah.

15 So it's the same thing. I think it's you
16 know, just needs a new framework and unfortunately it
17 requires a new word and that's systems biology but
18 it's basically --

19 DR. LE GUEN: Well, it would be one of the
20 conclusions in your letter to create a science -- the
21 3-D approach as I said.

22 CHAIRMAN RYAN: Yeah, it's been a very
23 rich discussion on the biology question and so we
24 appreciate all. And thank you, Vince, for your
25 emphasis on making sure -- I mean, there is stuff that

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1 was discovered before 1970.

2 DR. BARCELLOS-HOFF: Oh, yeah. And
3 actually I think we're going to go back to the cell
4 membrane, so another 50 years from now.

5 DR. HOLAHAN: Ron Koss was looking at the
6 microtubule exchange back in the '70s, Bill Dewey's
7 lab, looking at what's being exchanged between cells
8 for hypothermia. And I'm one of the feeder folks. We
9 use feeder cells all the time. Increase survival, two
10 orders of magnitude--

11 DR. BARCELLOS-HOFF: Bystander effect;
12 right?

13 DR. RYAN: Dr. Land, you've been quietly
14 taking all this in. What do you think?

15 DR. LAND: Actually, I--well, okay, I'll
16 say something. I don't think I've heard anything that
17 suggests a need for anything, except the LNT with the
18 DDREF. I think it's the same as it was.

19 DR. RYAN: I'm sure you say the current
20 biological work is probably saying an interesting and-
21 -

22 [Simultaneous conversation]

23 DR. LAND: Of course it does. I don't
24 "cue" easily.

25 DR. HAMMITT: A couple points to make and

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1 I'm not sure where they best fit, but one is--this is
2 partly, would have come well after Dr. Puskin's
3 remarks. One is this idea of looking for acceptable
4 risks in ALARA and stuff like that, and it relates to
5 the medical exposures versus occupational and natural
6 background.

7 And that is, to my mind, there's always
8 this question of how much can we reduce risk and what
9 do we give up to do it. And that's the central
10 question. Talking about acceptable risk is saying
11 there's some level of risk, such that if it was below
12 that, we wouldn't bother to reduce it. So if it was
13 above that, we would reduce it, ignoring whatever we
14 give up to reduce the risk.

15 And ALARA is basically saying that it's
16 easy to reduce the risk, let's do it, even if we don't
17 reduce it much. If it's hard to reduce the risk,
18 let's not do it, even if it might be very beneficial.
19 So both of those are incomplete because they focus on
20 only one side.

21 And as a way to think about this, the kind
22 of, the risk of a fatal crash per car trip is
23 something like one in a million. So that's very, very
24 small; right? So from that, I might argue any time
25 you fasten your seatbelt you're just wasting your

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1 time. And this is an acceptable risk. Why do we
2 bother to fasten a seatbelt? Because it's easy. You
3 know, it doesn't hurt us much to fasten it.

4 Another framing of the same thing is the
5 risk of dying in a traffic crash in the U.S., over the
6 lifetime, is about one percent. That's huge; right?
7 So why don't we ban traffic, ban cars, ban trucks?
8 All because there are a lot of advantages to having
9 them.

10 Well, why don't we reduce the speed limit
11 to 10 miles an hour. That would eliminate most of
12 these deaths; right? Well, that's very costly in a
13 bunch of ways. So it's kind of always how much
14 benefit you get against how much of what else that you
15 care about do you give up, and any approach to kind of
16 ignore that tradeoff might be a useful heuristic, in
17 many cases might work well, might avoid complicated
18 calculations, but it's an oversimplification that will
19 be misleading, at least some of the time.

20 The other point has to do with this choice
21 of model. So I think it's very clear that a very low
22 dose is where we can't measure the arm directly, we're
23 always kind of extrapolating, and it seems to me there
24 were comments about--maybe you said two different
25 groups looked at the same data and came to

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1 diametrically opposite conclusions.

2 I don't know the details of that, but it
3 seems to me that some of what goes on is people kind
4 of have a null hypothesis and then say, well, we can't
5 reject that null, and that low dose risk, so all
6 reasonable nulls are not rejectable. It could be
7 linear null threshold. We can't reject that. There
8 could be some threshold in the lower than EPI range,
9 we can't reject that, and that's not really a useful
10 way to think about the problem.

11 Most people, when they learn statistics,
12 do learn this kind of frequent as classical style, as
13 a null hypothesis, can you reject it? Failure to
14 reject is not the same as evidence in favor of the
15 hypothesis, of course, although we slip over that a
16 lot of the time, and there's very little power, you
17 can't reject anything reasonable. And so what I
18 think, the way I handle this is to recognize there's
19 a false suite of models or risk levels that might be
20 true. We can't differentiate among them very well.

21 We just need to acknowledge all these
22 things are possible, and from biological theory and
23 various sorts of evidence and EPI evidence, we maybe
24 able to look, assign kind of rough probabilities to
25 different models, and then we need to work with

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1 expected value over those models in the uncertainty,
2 and then risk over those different models.

3 DR. RYAN: I mean to borrow some risk
4 language, it sounds like you're talking about see if
5 you can come up with central tendencies, in a range
6 around some central tendency as the real predictor.

7 DR. HAMMITT: Exactly. You know, we, as
8 humans, are always uncomfortable with uncertainty and
9 tend to be unwilling to admit how much uncertainty
10 there is about anything we care about, and that's just
11 a problem.

12 DR. RYAN: That's a good point.

13 DR. HAMMITT: But, you know, to some
14 extent--maybe this example would help. If we think of
15 different models. So what we care about as a person,
16 or a government official, is whether somebody gets
17 cancer or doesn't get cancer. We don't care per se
18 about the probability of cancer. That's not
19 important. It's the outcome that's important.

20 If I have a .5 risk and I don't get
21 cancer, I have a .1 risk and I don't get cancer, it's
22 all the same to me.

23 So you can think of these different dose
24 response models as essentially like buckets of balls
25 where there's some--you know, in this bucket there are

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1 two or three black balls and if you draw a black ball
2 you get cancer, and a lot of white balls--or here,
3 there are ten or fifteen black balls and a lot of
4 white balls, and these represent the different dose
5 response functions.

6 So if we know the dose response function,
7 then we're drawing from this bucket, we know the
8 probability of getting cancer. If we don't know the
9 dose response function, essentially we're saying, you
10 know, I'm drawing from this bucket or this one or this
11 one, and maybe I have some rough probabilities for how
12 likely it is I'm drawing from each.

13 But in that sense, uncertainty about the
14 model is no different than uncertainty about the
15 outcome. It's just sort of compound. First, there's
16 the lottery, which bucket am I drawing from? which
17 dose response functions; true. Then there's the
18 lottery--which ball do I pick from? So conceptually,
19 it's not really much of an addition, but I think people
20 overemphasize, too much, results conditional on the
21 model and are unwilling to say I'm uncertain about the
22 model, and I can handle that by thinking about it as
23 a risk over which model is actually most accurate.

24 DR. RYAN: That's a very important
25 insight, I think, for us to think about. You don't

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1 have to pick the, quote, right model. You have to
2 explore all the reasonable probable models, and
3 understand what that means in terms of the overall
4 outcome. Thank you.

5 DR. HAMMITT: And there are cases where
6 the slopes of these models will be pretty similar, in
7 which case uncertainty about the model doesn't really
8 matter.

9 DR. RYAN: I think the graphic
10 presentations you gave really explain that well too.
11 Yes. Thank you. I didn't mean to cut you off. Is
12 there anything else? Okay.

13 Jerry.

14 DR. PUSKIN: As a response to that, I'm
15 very sympathetic with what you're saying. Let's
16 assume that LNT is correct and the implication of it
17 would be, that really matters, is the collective dose
18 and not maximum individual dose, and the problem, of
19 course, from a regulatory standpoint is that people
20 are--you have the equity as well, that nobody wants--
21 you know, I think part of it is acceptability of risk.
22 People like to feel like, well, my risk is trivial, my
23 kids' risk is trivial, and that's important to them,
24 aside from the fact of what's the expected number of
25 cancers in the population.

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1 From a public health protection
2 standpoint, you know, we want to minimize the number
3 of cancers, the right thing to do is to minimize
4 collective dose. But we don't do that occupationally.
5 If we can, you know, if we could reduce the collect
6 dose, in some cases is the case, as I understand it,
7 you could reduce the collective dose by allowing a few
8 people to have really higher doses and don't have, you
9 know, allow an individual to stay in there and get
10 five or ten rads at a time, so we don't keep changing,
11 getting a extra dose every time you change--

12 DR. RYAN: I don't think any of those
13 ALARA strategies have a huge impact on collective
14 dose, anyway.

15 DR. PUSKIN: Right. But anyway, you can
16 imagine that. The same thing with regard to--well, in
17 the case of environmental exposures. Generally, it's
18 just from a public policy, public perception
19 standpoint, regulating on individual, the maximum
20 individual doses is more palatable, and that's what
21 ICRP's kind of come down that way now too. They said
22 what matters is people's risk. I'm sort of
23 sympathetic to the idea that people don't really die
24 of risk, but they do die of cancer, and what really
25 matters is what the collective dose is.

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1 DR. HAMMITT: If I could comment on that.

2 DR. RYAN: Please. Yes.

3 DR. HAMMITT: I think often, a lot of what
4 happens is we kind of frame things, so you worry about
5 the risk of getting cancer from radiation and you
6 don't like that being distributed unequally within a
7 population. But that risk is pretty small compared
8 with the total risk of dying or dying of cancer, and
9 dying within a year, and I think--you probably know
10 the work of Daniel Kahneman and Amos Tversky,
11 psychologists, who developed this idea of heuristics
12 and biases, which sort of explain the way--heuristics
13 we use to deal with quantities and probabilities and
14 stuff, and, you know, certain attributes can be very
15 salient, and we frame things, we segment stuff.

16 So, you know, I'd be quite willing to
17 tolerate a cancer radiation risk, I don't know, 10 or
18 a 100 times after than the average, if my risk of
19 heart disease went down 5 percent, cause that's
20 probably a much bigger increase in survival
21 probability or--you know, I'm making up these numbers
22 but you know the point.

23 And there were proposals kicked around
24 with Superfund cleanups, where there are claims that
25 a number of sites, the cost of cleanup relative, is

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1 very high relative to the health benefit, and it's
2 logical for the community around that site to say,
3 yeah, clean it up.

4 But what if the responsible parties could
5 go to the community and say, well, look, instead of
6 cleaning this up, we'll give you half as much money as
7 it would cost to clean it up and you can use that
8 money for things that you might actually find more
9 valuable, and it's sort of likely the community would
10 find stuff they'd much rather have than these pretty
11 small risk reductions.

12 So framing is important in this more
13 comprehensive view, and can protect us sometimes from
14 focusing too much on stuff.

15 DR. RYAN: Let me see if our members have
16 any questions.

17 Jim, do you have any questions or
18 comments?

19 DR. CLARKE: Just a quick comment, if I
20 could. Again, I think it's been another wonderful
21 day, and it's got me thinking about a lot of things.
22 As I mention, I come in from the risk analysis with
23 chemicals and Superfund sites into the radiation
24 arena, and I still think--it kind a pains me when I
25 hear people say I work with chemicals and I work with

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1 radiation. It always seemed to me that there's very
2 fertile ground there, where those intersect.

3 But I liked your comment. I've been in
4 two very serious automobile accidents. Both times I
5 had my seatbelt on. Both times the air bag came out.
6 I guess I'm glad I did it.

7 And that's the problem with probabilities.
8 You know, they all go to zero or one, and it's really
9 the outcome that we're interested in. So again this
10 has stimulated a lot of thinking about chemicals,
11 initiators, promoters, radiation.

12 Vince's chart with the practical
13 threshold. What do we do with that? Well, we
14 probably look at it the same way the EPA looks at
15 chemicals that don't cause cancer. Incorporate some
16 safety factors.

17 So again I think there's very fertile
18 ground here, and thank you all.

19 DR. RYAN: Ruth.

20 DR. WEINER: I too want to thank the
21 panel. This has been really great. But I do have
22 some questions and these are things, these are
23 problems that are of concern in how we apply some of
24 these to, in my case, to environmental impact
25 assessment, and I'd particularly like to address Dr.

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1 Puskin.

2 You mentioned that the real thing is
3 collective dose. Well, how do you handle the question
4 of the microdoses to mega populations question,
5 especially when, if you continue to multiply, and then
6 multiply your result--if you continue to have a larger
7 and larger population and then you multiply your
8 result by some linear conversion factor to latent
9 cancer fatalities, which is what is done in
10 environmental impact statements, and this is
11 presented, then presented to the public as you have X
12 events and that's going to result in Y cancers.

13 And what people take away from that is,
14 you know, radiation gives me cancer. They don't look
15 at, oh, the probability is small compared to some
16 other probability.

17 And there is a certain, I don't know
18 whether to call it misuse or fallacy or what, but the
19 notion--getting back to what Dr. Mossman said
20 yesterday, if the individual isn't harmed, the group
21 isn't harmed.

22 How do you square that with your statement
23 about collective dose and how do you apply the very
24 small average dose to large populations? How do you
25 handle the microdose to mega population?

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1 DR. LAND: Can I add something on that?
2 When I present, or my coworkers present the results,
3 or our estimates of risk from, say, fallout in the
4 Bikini tests in the Marshall Islands, one way we can
5 do it is we put this is the excess and this is what
6 you would have without that--what they would have had
7 without that, is what you would predict without that
8 particular thing. So it tends to be a rather small
9 amount, except for the people who really did get an
10 awful lot of dose, and in that case you tend to
11 overestimate the risk an awful lot because we don't
12 know that much about the risk from really high doses.

13 DR. WEINER: If I could respond to that.
14 Yes, we all present it that way. It's presented that
15 way in every EIS. Oh, the risk of cancer is 25
16 percent and this raises it to 25.06 or some such
17 number.

18 I do not think that that conflicts with
19 the message that people--people don't look at the
20 relative size of the probabilities. They look at
21 cancer or no cancer.

22 Yes, I quite agree with you--the number
23 that you come up compared, with some more realistic
24 number, is always very small, but we're still sending
25 a message that you have this event, and what happens?

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1 You get cancer from it. And I think we've somehow got
2 to send a different message, and if I could get back
3 to something that Dr. Mossman said, and I wish he had
4 been able to stay.

5 Your slide, Dr. Puskin, your slide 17,
6 which said help the public put risks into perspective.
7 And that's what you're saying. I think we've had 20
8 years of that and it hasn't worked, to be perfectly
9 frank, blunt, about it. With every talk, we put the
10 risk into perspective, and the perspective is always
11 there, and it's always the same, and we still have--
12 you know, we have whatever "spin" is put on this, it
13 is that you can say it's safe, it's safe, it's safe,
14 but at the same time you say it gives me cancer.

15 DR. PUSKIN: I have to think of what the
16 actual situation is where you'd have such a large
17 population but--

18 DR. WEINER: Would you like an actual
19 situation? I'd be happy to provide it right now. But
20 go ahead.

21 DR. PUSKIN: Well, as I said when I did
22 that slide, that it is a problem, and I don't have a
23 magic solution to it.

24 But I would say this--and maybe I'm wrong
25 about this--but what is it that the nuclear industry

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1 is not able to do because of this? Sure, there's some
2 resistance, but is it really that large, that it's
3 such a huge problem to our society? Actually, I don't
4 see it. I see a resurgence of nuclear power, people
5 accepting it, I think is one example.

6 I don't know what to do beyond explaining
7 to you. I think we can do better at explaining what
8 a risk means. For example, ten to the minus four risk
9 is one that we often use. A one in 10,000 risk means
10 that in a city of three-quarters of a million people,
11 that's one case a year.

12 Now if the murder rate in your city were
13 one case a year, would you really be worried about
14 getting murdered? And one in a million risk is one
15 every 100 years.

16 I think partly, maybe we need to be more
17 creative in terms of explaining what these risks mean.
18 I know one thing that's true is that oftentimes, the
19 risk is concentrated in the people who are closest by.
20 It's not just a huge--the effect of including
21 everybody doesn't really make that much difference.

22 DR. WEINER: Let me give you the example
23 that I was thinking of, and this is a real example.
24 In the Yucca Mountain environmental impact statement,
25 we calculated the risks from routine transportation of

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1 spent nuclear fuel from 77 sites around the country to
2 Yucca Mountain.

3 If you do this in trucks, with four
4 assemblies per truck, this is 53,000 shipments. If
5 you calculate the population dose from that, and
6 multiply by, at the time we used five times ten to the
7 minus four, latent cancer fatalities, which should be
8 latent fatal cancers--but anyway, latent cancer
9 fatalities per rem, you get two cancers.

10 DR. PUSKIN: Over what time period?

11 DR. WEINER: Twenty-four years. Now I
12 believe that we can all come to the conclusion that it
13 is very unlikely that there will be two cancers from
14 those 53,000 shipments over 24 years.

15 You take that number with an EIS that I
16 reviewed recently--

17 DR. PUSKIN: What do you mean "unlikely"?

18 DR. LAND: How do we come to this
19 agreement that that's very unlikely?

20 DR. WEINER: I find it hard to believe
21 that taking what is a very small average dose, on the
22 order of ten to the minus eighth, ten to the minus
23 eighth, ten to the minus seventh rem--we did this in
24 rem--taking that and simply multiplying by the number
25 of people by the side of the road--

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1 DR. RYAN: Part of the problem I think in
2 these scenarios, and this one, in particular, is that
3 there is no central tendency evaluation of what is a
4 likely dose. It's all bounding case.

5 DR. PUSKIN: I'm assuming the dose--
6 [Simultaneous conversation]

7 DR. RYAN: A bounding case masks the real
8 central tendency of the risk. So I think that's part
9 of it.

10 DR. PUSKIN: I would say there's nothing
11 wrong with the idea of adding up a lot of very small
12 risk--for example, as we've said, ten to the fourteen
13 cells in the body, one of them is going to turn into
14 a cancer cell. So the odds of any one of them is one
15 out of ten to the fourteen, and yet we see finite
16 numbers of cancers.

17 So you can add up a lot of very small risk
18 to get something finite, and obviously it's not
19 observable.

20 DR. RYAN: And I think the other point is
21 if there is some estimate--Ruth, excuse me for jumping
22 in--but if there's two cancers that are excess because
23 of an activity, that it's really, the question, the
24 second part of this, Can you distinguish that from the
25 cancer that will occur in the affected population

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1 anyway?

2 DR. LAND: The fact is you'll never find
3 out.

4 DR. RYAN: Right. I mean, there could be,
5 you know, three extra cancer deaths in a family of
6 heavy smokers that moved in during the 24 years. So,
7 you know, something else, and it really is well down
8 in the variant rate that's going to occur anyway.

9 DR. WEINER: As a matter of fact, in the
10 same environmental impact statement, we did a number
11 of traffic fatalities. You compare it with this, you
12 compare it with that, and to a member of the public
13 who wishes to focus on the cancers from ionizing
14 radiation, this doesn't make any difference.

15 Now let me just carry this one step
16 further--

17 DR. RYAN: Just one.

18 DR. WEINER: Just one. This is another
19 real-life environmental impact statement. Instead of
20 53,000 shipments over 25 years, 24 years, we have
21 something like 150 shipments over larger distances,
22 larger populations along the side of the road, over a
23 period of 40 years, with the result of 1150 cancers.

24 Now you might be able--and I'm sure that
25 even those 1150 are a tiny fraction of what you would

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1 get anyway. But that's a big number, and if I saw
2 that number in an environmental impact statement, I
3 don't think I would want that project.

4 DR. RYAN: So what's your question?

5 DR. WEINER: So my question is, is this an
6 appropriate use of collective dose? I've been
7 hearing, yes, collective dose is fine. But when you
8 just keep multiplying and multiplying, you get a
9 ridiculous number.

10 DR. LAND: So what's your alternative?

11 DR. WEINER: The alternative would be to
12 look at the maximally-exposed individual, to look at
13 individual doses rather than collective doses, because
14 multiplying an average dose by the number of people
15 somehow strikes me as not a dose calculation.

16 DR. RYAN: Ruth, I would point you back to
17 some of the things Dr. Hammitt talked about, that we
18 discussed, and that is that if you can get at a
19 central tendency, and some range of behavior around a
20 central tendency, you're really exploring the risk for
21 what it is. You know, then you can judge it based on
22 those various parameters of risk. A bounding case is
23 misinformed.

24 DR. WEINER: Yes.

25 DR. RYAN: They're misinformed, and they

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1 mask risk, not--

2 DR. HAMMITT: That may be useful if we can
3 calculate--

4 DR. RYAN: In some contexts, quite
5 frankly, you know, the more they use the less I like
6 them, because they really do overestimate, typically,
7 and they miscommunicate reality.

8 You know, just to give an example, 10 CFR
9 61 is based on the agricultural and true-to-scenario,
10 that grows his food in radioactive trash, which is
11 plastic tie-back booty shoe covers, shovels and picks.
12 I mean, he has to grind up metal and grow food in
13 them. It's not a realistic scenario.

14 By the way, nobody that I know grows all
15 their own food.

16 DR. HAMMITT: Certainly not in soil like
17 that.

18 DR. RYAN: Certainly not in soil--and by
19 the way, has to be unemployed cause he has to get
20 external radiation exposure for 18 hours a day. And
21 on and on and on down through the scenario.

22 So, you know, the old thinking of, well,
23 if I bound the problem then, you know, I know I'm
24 better than that in reality, so I'm okay. Well,
25 that's not a good treatment of risk. That's an

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1 engineering type of judgment.

2 So I get back, to answer your question, at
3 least my view would be to follow, you know, our
4 predecessor in this committee, Dr. Garrick's view, and
5 let's get at, you know, a real treatment of
6 probability and risk.

7 DR. PUSKIN: I would guess that the
8 exposure's been--the collective dose has been greatly
9 overestimated. It's some sort of upper bound--

10 DR. WEINER: The dose has been--the dose
11 may be overestimated by a factor of about five or six.
12 But it is true, that other parts of this exposure have
13 been greatly overestimated. And Dr. Ryan's quite
14 right. If you do a central tendency or a more
15 realistic exposure, these things come down and--

16 DR. RYAN: So you got your answer.

17 DR. WEINER: I do have my answer, from
18 you. But there is--if you combine collective dose
19 with the conservative estimates, this is what you get--
20 -

21 DR. RYAN: Dr. Hammitt wanted to make a
22 comment.

23 DR. HAMMITT: I was going to try and add
24 two things. One is first on, back to the linear no-
25 threshold and so forth--well, imagine, we think

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1 there's some chance linear in our threshold is
2 correct, and a much higher chance that there's some
3 threshold that's relevant, such that there's really
4 zero risk.

5 If we calculate the expected risk, it's
6 going to be the probability that the linear no
7 threshold model's right times whatever risk it
8 suggests.

9 So if you think there's only a 10 percent
10 chance that LNT is right, that means you've reduced
11 your risk by a factor of ten, but that may not really
12 be enough to actually change any policy or change
13 policy very much, given the wealth of other
14 uncertainties here and what the dose is and everything
15 else.

16 DR. RYAN: And I mean that's a very
17 important point for us to take away as a complete and
18 thorough treatment of all the components of risk, and
19 the uncertainties in them, is really the right way to
20 get at it.

21 DR. HAMMITT: And then the other thing
22 was, on this first communication point, is I think a
23 very powerful book by a guy named Howard Margolis,
24 who's at Chicago Public Policy School, called "Dealing
25 With Risk," I think 1996 or thereabout, and he was

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1 sort of proposing, it makes a lot of sense to me, that
2 when people react to risks, what matters a lot is
3 whether there's some activity, whether the benefits
4 and/or the risks of it are on screen, to me, the
5 person making the judgment, and, you know, in the case
6 of people trucking nuclear waste by my doorstep or
7 having a nuclear power plant near me, I tend to not
8 really perceive the benefits. I perceive potential
9 harm to me, I think that's outrageous, and shouldn't
10 have it; right?

11 Whereas if it's driving a car or
12 something, I perceive the benefits, I perceive the
13 harms as well, and make it a somewhat more reasoned
14 judgment, and there are cases where, you know, I
15 perceive the benefit but I'm putting the risk off on
16 somebody else, then I don't worry about the risk
17 perhaps.

18 And so you mentioned nuclear power plants.
19 It seems like with climate change, and people worrying
20 about that, that will improve the discussion of our
21 nuclear power because there's a big clear benefit
22 associated with it, and that we're avoiding some other
23 harm that many people care about.

24 DR. LAND: One thing is would you rather
25 live next to a nuclear power plant or a coal power

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1 plant? And I know the answer.

2 DR. HAMMITT: But we've known the answer
3 to that for like 30 years--

4 [Simultaneous conversation]

5 DR. HAMMITT: --figured that out yet.

6 DR. WEINER: Nothing has happened.

7 DR. HAMMITT: But with climate, too, maybe
8 they'll get it.

9 DR. LAND: Maybe.

10 DR. RYAN: All right.

11 DR. LE GUEN: In fact about this, we are
12 exactly the same experience in France. People who are
13 living close to the nuclear power plants work in the
14 nuclear power plant, and live with the nuclear power
15 plants. So there is an economy region.

16 But when you are talking about waste, you
17 take waste from another place and you put in another
18 place, and people say, well, why we must accept waste
19 from other parts of France, because we have no benefit
20 about that? And so the acceptance's completely
21 different.

22 DR. PUSKIN: So what do you do then?

23 DR. LE GUEN: Well--

24 DR. PUSKIN: Are we able to take it?

25 DR. LE GUEN: Well, we have, we try to

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1 create now an economic region around the west, and we
2 provide money for that, and from the industry we use
3 the waste--and this, now, we have decided to give
4 money, to give grant, and so on, in order to develop
5 a real economy around the waste disposal.

6 DR. LAND: An economy that depends on
7 having the waste, that it used the waste, or--

8 DR. LE GUEN: Sorry?

9 DR. LAND: An economy that depends on the
10 waste, that isn't perceived as sort of a bribe for
11 having to live next to the stuff?

12 DR. LE GUEN: It's the expectation much
13 more than--that's why I fully agree with James.

14 DR. LAND: No, but what I mean is that the
15 economy wouldn't be there if it were not for the
16 waste, not just because--

17 DR. LE GUEN: Absolutely. No, no, no.
18 There was nothing.

19 DR. LAND: I mean, the economy depends on
20 having the waste there, in more than sort of a bribery
21 sense. That's what--

22 DR. LE GUEN: Yeah; yeah. Okay.

23 DR. LAND: Yeah.

24 DR. LE GUEN: Okay.

25 DR. RYAN: Any comments?

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1 Neil, you had a comment?

2 MR. COLEMAN: Neil Coleman, ACNWM staff.

3 One of the take-aways I have from this
4 meeting is the idea that we might never be able to
5 differentiate the most applicable biological response
6 model in the low dose zone.

7 And it has some significance on the
8 economic models as well. But I'm going to slightly
9 take issue with that because I think one of the models
10 is directly amenable to testing, can be tested with
11 unsophisticated but somewhat difficult experiments.

12 Yesterday, Tom Tenforde spoke about the
13 idea of extreme low dose effects, where experiments
14 could be done in very low background environments, the
15 idea being to see if test subjects actually do suffer
16 in the absence of background radiation, which in the
17 U.S. averages about 350 millirem, is this hermetic
18 effect real as some experiments actually do suggest
19 now?

20 Unlike the other biological response
21 models, you can validate this with controlled
22 experiments. This would help address the unfortunate
23 public perception that each and every ionization event
24 carries a cancer risk, leading some people to fear
25 even getting a simple diagnostic dental x-ray.

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1 The question is: How could such
2 experiments be done in a credible way with results
3 that the public would believe and accept?

4 DR. RYAN: So there. Does everybody
5 accept the question? I'm not sure I agree with the
6 question but--

7 DR. METTING: Mike, can I say something.

8 DR. RYAN: Sure. Please just come to the
9 microphone and tell us who you are for the record.

10 DR. METTING: I'm Noelle Metting. I run
11 the low dose program. This is an interesting concept.
12 Of course you know that people have been suggesting
13 that we do that, that we lower the background, and
14 it's been done, preliminary experiments have been done
15 with cells. The cells do look like they're worse off.
16 But I don't even want to get into that.

17 I wanted to make one comment about the low
18 dose program and just biological, the biological
19 experiments in general, and I think that you may have
20 missed this but what I think is it's giving, the
21 biology is giving us a reason to do the experiment, of
22 ignoring high dose epidemiology. Let's ignore it for
23 a while and see what just the low dose epidemiology
24 tells us. Why don't we take a look at that? Let's
25 pretend that the A-bombs didn't drop. Let's look at

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1 the low dose epidemiology. I think the biology says
2 it might be interesting. So there's an idea.

3 DR. RYAN: Great. Thank you.

4 Any other final comments? Questions? We
5 have some other--you've been waiting patiently.

6 MS. MITCHELL: Jocelyn Mitchell from the
7 Office of Research. I wanted to mention that the NRC
8 and the Commission of European Communities, about ten
9 years ago, attempted to get a group of experts, four
10 from the U.S. and four from Europe, to give
11 likelihoods, degrees of belief, if you will, on
12 possibilities for what would be the low dose response,
13 and it's actually written up in a new reg report, a
14 new Reg CR report.

15 Unfortunately, the deviation from LNT was
16 so insignificant, that it just didn't exist for all
17 practical purposes. Only one person gave a nine/zero
18 likelihood to something that was not LNT. And I don't
19 know whether we didn't have the right experts, whoever
20 they were, but we did attempt to do that, and I don't
21 know how you would get folks to give you numbers like
22 that.

23 DR. RYAN: Thank you. Is there anybody on
24 the bridge line? Hello? Nobody else is there. We've
25 had one request for an individual to make comments.

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1 Let's see. It's Mr. Dennis Nelson. Dennis, now is a
2 good time.

3 DR. NELSON: Right here?

4 DR. RYAN: Right up there is fine. This
5 is Mr. Dennis Nelson from the organization SERV, S-E-
6 R-V, and he'll tell us a little bit about that and
7 make his comments.

8 DR. NELSON: Good afternoon. My name is
9 Dennis Nelson. I'm a retired naval officer. I have
10 a PhD in biochemistry. I did biomedical research in
11 the Navy for a number of years, although my research
12 was not specifically in the area of radiation, it was
13 biological. I did work on hemoglobin. I did work on
14 immune function.

15 But there are a couple of points that I
16 wanted to make, that I think you should try to
17 incorporate in your decision making, and one of those
18 is that--and I also want to follow up on the risk
19 management thing that was mentioned earlier.

20 Basically, the traditional view of
21 radiation damage in biological systems has been that
22 it damages DNA, and that the DNA damage then reflects
23 a altered protein or a defective protein which then
24 doesn't do what it's supposed to do.

25 And that's probably still very true.

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1 However, some of the recent studies have shown that
2 epigenetic effects may address more, not the integrity
3 of the gene and the protein but the actual turning on
4 and turning off of that gene and protein.

5 So it's possible that radiation epigenetic
6 effects may cause methylations or alkylations of
7 various control proteins, or substances, which may
8 turn on or turn off tumor suppressor cells or tumor
9 promoter cells. Sorry. Tumor suppressor genes or
10 tumor promoter genes.

11 And this may be the cause of cancer. It
12 may not be that you have just a defective protein but
13 you just turned on the wrong gene. So that needs to
14 be looked at. It needs to be looked at in terms of
15 dose, dose response.

16 Also, I think that you need to look at
17 latency, and that's something that's been bugging me
18 for many years. You know, what causes latency?

19 Now the traditional explanation is that
20 there's a multi-step model of carcinogenesis, that it
21 has to get hit once to cause it to transform, and then
22 another time to promote, and then to transprogress, or
23 whatever. I don't know all the procedures.

24 But suppose that there's another
25 explanation, and that other explanation is that

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1 latency is caused by a one-time hit, or defect cause
2 in a pluripotential stem cell, one that lies dormant
3 or quiescent for a decade, and then all of a sudden is
4 recruited in the dividing population when the needs
5 are there for repair or for growth or whatever.

6 So I think you said earlier that maybe
7 there aren't ten to the fourteen cells that are
8 susceptible. Maybe it's only--maybe it's a fraction,
9 one percent, maybe less, and maybe those are the
10 susceptible cells.

11 So we have to think about that. Maybe
12 it's just a one-time thing and when that cell finally
13 is recruited into the dividing population, it goes
14 berserk.

15 So there are many alternative, possible
16 models for carcinogenesis, and I think they all need
17 to be looked at.

18 Then lastly, the risk-benefit thing, I
19 wanted to address that because that I think is the
20 biggest sticking point, and it's a point that you made
21 earlier, that why can't people accept this. It's
22 because the same people don't suffer the risks that
23 get the benefit. And that's precisely why.

24 For example, we have nuclear medicine
25 patients that are floating around amongst us, that may

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1 sit next to you in an airplane, or in a theater, or on
2 a bus or a train. And they may be emitting 20
3 milliroentgens per hour, and you're sitting next to
4 them for two hours, you may get 40 millirems. And
5 next week you might go to another plane, and you might
6 sit next to another one, and you get another 20 or 40
7 millirems. These are not controlled sources. They're
8 just basically random events.

9 And you yourself have no benefit from
10 them. The benefit is derived by the person who is
11 sitting next to you but not by you. So why should you
12 have any risk whatsoever. So I think that these
13 people need to be controlled and I think that the NRC
14 needs to revisit its policy of allowing these people
15 to leave while they're still very highly radioactive.

16 And conversely, maybe it's not as big a
17 problem, but these shipments that I talked about
18 earlier, these radiative casks going to Yucca
19 Mountain, and as we get more and more medical
20 procedures, nuclear medicine procedures, as we get
21 more and more shipments, what we're talking about with
22 Yucca Mountain, these casks are going to be a lot more
23 prevalent on the highway.

24 And how do we know that they're going to
25 be protected? How do we know that the individual

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1 along the road, at the gas station or the truck stop,
2 who goes over and leans on the truck, isn't going to
3 get--well, I won't say a huge dose, but a larger dose
4 than he really deserves, because he's not getting any
5 benefit from that nuclear waste shipment.

6 So anyway, these are just my observations.
7 That if you want it to be accepted, it's going to have
8 to be fair, and it's going to have to impact or cause
9 risk to the people who benefit from it, not another
10 segment of the population. And that's really all I
11 have to say.

12 DR. RYAN: Mr. Nelson, thank you very
13 much. Would you mind telling us again what SERV was.
14 You mentioned it to me.

15 DR. NELSON: SERV. Support and Education
16 for Radiation Victims.

17 DR. RYAN: All right. Thank you very
18 much.

19 DR. NELSON: A group that I founded a few
20 years ago. I am also a down-winder. That's why I
21 have this interest in this subject, because my family
22 was affected by the bomb testing in Nevada back in the
23 late '50s, and I have three members of my family that
24 died at very young ages, and seven different kinds of
25 cancer in five family members. So to me, it's a

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1 personal thing. but I'm also a scientist and I want
2 to understand this scientifically. I'll reject things
3 that are not scientific but if it can be explained to
4 me scientifically, and it's defensible, and it's not
5 just, what I sometimes consider politics or propaganda
6 or economics or whatever, then it's a lot easier for
7 me to accept and understand.

8 DR. RYAN: Well, we appreciate.
9 Hopefully, you've gotten some benefit from the
10 scientific discussion here with a couple days--

11 DR. NELSON: I have. It was a great--

12 DR. RYAN: Thank you for sharing your--

13 DR. NELSON: --couple days and i really
14 enjoyed it, and I got something from every one of you.

15 DR. RYAN: Well, thank you very much for
16 coming, and thanks for sharing your views as well.

17 Are there any other comments from anybody?

18 DR. TENFORDE: I have a question, Mr.
19 Chair.

20 DR. RYAN: Come on up, Mike.

21 MR. BOYD: Okay.

22 DR. RYAN: Yes. And Tom, why don't you
23 ask that question in the meantime.

24 DR. TENFORDE: Real quick. I had the
25 impression that the outcome of this discussion would

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1 be a letter report from the Advisory Committee to the
2 commissioners.

3 DR. RYAN: That's correct. Yes. We
4 actually address i to the chairman on behalf of the
5 whole Commission.

6 DR. TENFORDE: I'm wondering at this
7 stage, before some of us depart, we have some
8 continuing responsibility to review and comment on
9 your letter report?

10 DR. RYAN: No. What we do is take the
11 record of the transcript, and then we synthesize the
12 information into a letter to the Commission as we see
13 it, and it's not your report to the Commission. It's
14 our report of what information we gathered and our
15 assessment of that information to the Commission.

16 If you have anything else you want to
17 provide to us, in writing, or additional support
18 information, or you want to make any comments on that
19 key points, and that's--I think we hit some key points
20 about biology and some of the other issues, and
21 modeling, and so forth. From each of you I think
22 we've gotten, you know, rich views and key points, and
23 we'll be faithful to summarize those, and that's the
24 typical scheme for letterwriting here with the ACNW.
25 And of course once our letter is prepared, we actually

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1 read it out in public before it's finalized.
2 Anybody's welcome to come and attend that session,
3 which will be next May, or next month, in May, I
4 forget which week at the moment, and then we finalize
5 the letter, we vote on it as a committee and then
6 that's prepared in final form and sent to the
7 Commission, at which point it's a public letter.

8 Mike Boyd.

9 MR. BOYD: Mike Boyd with EPA, and I'm
10 really sorry that Ken Mossman left, because he's the
11 person I wanted to say this to, but I--

12 DR. RYAN: You can say it and he'll get--

13 MR. BOYD: I'll say it and it'll get into
14 the record; right. And this is mainly just a little
15 bit of a defense of the risk assessment process at EPA
16 and the risk-based cleanup process as opposed to dose-
17 based, and why I think that the risk-based process
18 that we use, the classic Superfund approach, actually
19 has some real advantages.

20 And one of the things is that effective
21 dose, as you know, is a surrogate for risk, and it
22 tries to wrap up, and, you know, just a handful of
23 tissue weighting factors and radiation weighting
24 factors, you know, all the risks, biokinetics that we
25 have over, what, Jerry? 3200 risk coefficients--four

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1 risk coefficients for each of over 800 radionuclides.
2 So there's a lot of complexity that we have in our
3 risk coefficients that gets sort of summarized in the
4 effective dose term.

5 And another thing that we do, when you do-
6 -for doing occupational radiation protection, it
7 absolutely makes sense to use dose as your metric.
8 But when you're looking at long, you know,
9 perspective, or retrospective assessments, the risk
10 assessment approach that we use allows you to account
11 for decay. I mean, instead of a committed dose, you
12 actually are looking at a true decaying dose, over
13 time.

14 So, for example, people say EPA regulates
15 it 15 millirem, which is three times ten to the minus
16 four risk. That's not true. 450 millirem happens to
17 work out, using our risk estimates, to be about three
18 times ten to the minus four risk, but that's assuming
19 a 30 year default exposure, and a myriad of other
20 default exposure factors. So there's a lot that goes
21 into that three times ten to the minus four number.

22 So to say that say that 15 millirem is
23 three times to the minus four is really not capturing
24 it, by any means. But I just wanted to point out that
25 when you do a risk--if you were to do a three times

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1 ten to the minus four target risk-based cleanup under
2 Superfund, you would come up with target cleanup
3 values that almost, across the board, would be a
4 higher concentration than you would have to clean up
5 to, to achieve NRC's license termination rule at 25
6 millirems.

7 So I wanted Ken to know that, really, from
8 my perspective, there is no difference, and I just
9 wanted to say that the risk approach that we use does
10 capture a lot of variables that I think are useful.
11 You can capture, you know, weathering, decay,
12 occupational exposure factors. I'm probably just
13 babbling at this point but--

14 DR. RYAN: No, no, Mike, I think that's an
15 important point. There is--and you know, you
16 highlighted in that discussion, I think many of the
17 points we've heard today, that you really can't pick
18 one number or one parameter and really understand the
19 whole profile of dose and risk. You have to look at
20 it as a system.

21 MR. BOYD: System; right.

22 DR. RYAN: So that's a good point. And
23 even on the--and you're talking about the assessment
24 side and all the things that go into that. So we
25 appreciate that comment. Thank you.

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1 MR. BOYD: Sure.

2 DR. RYAN: Anything else?

3 Going once. Going twice. We are a little
4 bit ahead of schedule but--I'm sorry.

5 DR. LAND: I was just going to make one
6 last--

7 DR. RYAN: I'm sorry. I didn't see your
8 hand. Excuse me, Dr. Land.

9 DR. LAND: The discussion about how do you
10 express risk, I think the one thing you don't do is
11 say that there isn't any risk. Or you say that it's
12 a risk and it's too small to worry about; don't worry
13 about it. That never works.

14 DR. RYAN: Fair enough. My doctor says
15 don't worry about it. I still worry about it
16 sometimes. I'm with you.

17 DR. NELSON: There was one thing that I
18 forgot to say, and that is--

19 DR. RYAN: Yes, please, and just again,
20 just for the record, this is Mr. Nelson again.

21 DR. NELSON: David Nelson.

22 DR. RYAN: Just come to the microphone.

23 DR. NELSON: This is Dennis Nelson from
24 SERV again, and I just wanted to say that if you go
25 back and look at history, you'll see that there has

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1 been a progressive decline in the level, which was
2 seen to be biologically significant over 50 years.
3 Way back when, you know, 50 rads was not much, and
4 then it went down to twenty, and then it went down to
5 ten, then to five. Now we're talking in the one rad
6 range.

7 I just try to extrapolate that
8 historically and say, well, who knows what's going to
9 happen over the next 15, 20 years. Maybe we'll get
10 down to effect seen at millirads.

11 DR. RYAN: Thank you. With that, unless
12 there are any other closing remarks--yes? I did. Mr.
13 Early may call back. So I'm going to suggest we take
14 our 15 minute break and come back briefly for 3:15.
15 We do have a call-in time, that other folks may be
16 calling in, so we'll have to honor that obligation for
17 stakeholder input. So if you wouldn't mind, we'll
18 just take a 15 minute short break and reconvene at
19 3:15 and if there are other comments, we'll take them
20 at that time, and if there are no other comments at
21 that time we'll finish up. Thank you for your
22 patience.

23 (Whereupon, the meeting went off the
24 record at 3:00 p.m. and continued at 3:19 p.m.)

25 DR. RYAN: Thank you all for your

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1 patience. I know a couple of folks had to duck out.
2 I'd like to reconvene if we could, just for a minute
3 and check. Are there any commentators or members of the
4 public, or stakeholders, that wish to make any
5 comments on the bridge line?

6 It is the appointed hour for any
7 additional--is there anybody in the room that wants to
8 make any additional comments or observations? Hearing
9 none on either the bridge line or the room, we'll
10 adjourn the meeting, and again I thank you all very,
11 very much for your participation and your information.

12 It's been really enlightening for the committee and I
13 think we'll have a very rich letter to offer to the
14 Commission on these topics and the science involved.

15 So thank you all very much.

16 (Whereupon, the meeting went off the
17 record at 3:19 p.m. and went back on the record at
18 3:43 p.m.)

19 DR. RYAN: The committee is here. You
20 okay? All right. We have the microphone. You can go
21 ahead and take five minutes or so and make your
22 statement.

23 MR. EHRLE: Thank you very much. There
24 was much discussion of the problem, the uncertainties
25 related to dose, and I think those were well-taken.

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1 It was a difficult task for Dr. Hammitt to quantify
2 the specifics relative to any kind of dose cost-
3 benefit analysis. It's been very difficult.

4 I've been conversant with some of those
5 issues over the past several years. But what really
6 peaked my curiosity was the inability of the committee
7 to deal with the superlinear model, and Dr. John
8 Gofman, who of course was former associate director of
9 Lawrence Livermore, I have his 1981 book, and it
10 appears as though that was the first book that ever
11 really looked at this particular issue.

12 And he used the Land-McGregor RERF study,
13 and analyzed it, and concluded that, indeed, it does
14 show, using the RERF statistics, a superlinear model,
15 and so he explains it at some length there.

16 But then he goes on and in his 1990 book,
17 which was very favorably reviewed in New England
18 Journal of Medicine, he points out that a single
19 primary ionizing radiation track, operating
20 independently, these tracks from each other, are never
21 innocuous with respect to creating carcinogenic
22 injuries in the cells which they traverse.

23 Every track, without help from any other
24 track, has a chance of inducing cancer by creating
25 such injuries. And then he cites a study by

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1 Brackenbush and Brady, which is 1988.

2 "Since most cells repair radiation damage
3 with a characteristic time ranging from a few minutes
4 to a few hours, it is evident that irreparable or
5 misrepaired damage must dominate the low LET radiation
6 effect at low-dose rates."

7 And then he cites UNSCEAR, 1986, and
8 quotes: "The error-free repair of the DNA, which is
9 the most likely target involved leaves some fraction
10 of the damage unrepaired and the error-prone repair
11 may produce misrepaired sequences in the DNA."

12 And then he quotes Albrecht Kelleher, who
13 apparently was on the BEIR VII committee and he
14 describes the type of radiation-induced lesion which
15 would be difficult to repair.

16 A simple example would be two neighboring
17 single-strand breaks on opposing strands of DNA which
18 interfere with excision repairs.

19 And then he points out that there are nine
20 low-dose studies, human studies, the highest of which
21 is .9 rad, it isn't even a single rad, which would
22 have been of course 10 millisievert. So at that
23 level, he points out that the observation of
24 radiation-induced cancer means that repair is failing
25 to become flawless, even when it has to cope with the

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1 average track frequencies per nucleus of only 12
2 tracks, only ten, only six, only two, only one track,
3 only .67, and only .29 track. Those of course
4 correspond to the nine studies.

5 If repair had been flawless, it would have
6 successfully undone every carcinogenic lesion, and so
7 there would have been no excess cancer, at all, in any
8 of the nine studies.

9 He then discusses the question of
10 unrepaired, unrepairable, or misrepaired carcinogenic
11 injuries which occur at low dose, right down to the
12 lowest conceivable dose, or dose rate. And so here we
13 have evidence, at these very low ranges, and when Dr.
14 Mossman indicated that we don't have any information
15 at low doses, obviously there are numerous studies in
16 the literature, in the peer review literature, which
17 demonstrate, at these very low doses, every
18 significant excess impact.

19 Unfortunately, the studies that you're
20 using, and that ICRP, NCRP and even NRPB, and the UK,
21 which has now been reorganized, they all use of course
22 the Japanese study. Consequently, they do not deal
23 with internal dose. This is external gamma dose.

24 It is internal doses which have been
25 estimated to be at least 20 times more effective in

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1 terms of the inhalation into the lungs, and then
2 distribution throughout the other parts and organs in
3 the body, that has the greatest effect, and UNSCEAR
4 has recognized this again. In fact the British
5 National Radiological Protection Board, in 1995, said
6 that it may be argued, and I'm quoting, that a single
7 radiation track, the lowest dose and dose rate
8 possible traversing the nucleus of an appropriate
9 target cell, has a finite probability, albeit low, of
10 generating the specific damage that will result in
11 tumor-initiating mutation.

12 So I would hope that the members of the
13 committee, and others, would call for some of these
14 experts who have been studying this issue for years,
15 to be involved in future conferences, and that a
16 careful analysis of the superlinear model would be in
17 order, and would hope that the committee will
18 recognize that by elevating the hormesis thesis to the
19 level of LNT is a disservice to the scientific
20 community and to the public at large, because it has
21 been vetted by these committees on numerous occasions
22 and had been found wanting, and obviously, if there is
23 a superlinear effect, and I mentioned earlier the
24 comment, I ran into and got in on a meeting at Mayo
25 Clinic where Tom Hay from Columbia was giving a talk,

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1 and he showed with a diagram how the superlinear model
2 works.

3 So it's been recognized by persons in the
4 field who have high standing, that indeed, this is
5 worthy of further investigation and hopefully the
6 committee will respond in kind.

7 Thank you for your time. I appreciate the
8 work that you've done on this particular conference
9 and hope that it will lead to other conferences which
10 will have an expanded scope. Thanks again.

11 DR. RYAN: Thank you, Mr. Ehrle. We
12 appreciate your comments. Have a good afternoon.

13 MR. EHRLE: You too.

14 DR. RYAN: All right. We're done. Thank
15 you.

16 (Whereupon, the meeting adjourned at 3:50
17 p.m.)

18

19

20

21

22

23

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25

CERTIFICATE

This is to certify that the attached proceedings
before the United States Nuclear Regulatory Commission
in the matter of:

Name of Proceeding: Advisory Committee on
Nuclear Waste & Materials
188th Meeting

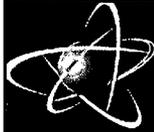
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Location: Rockville, MD

were held as herein appears, and that this is the
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James Salandro
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U.S.NRC

UNITED STATES NUCLEAR REGULATORY COMMISSION
Protecting People and the Environment

**Effects of Low Radiation Doses
Science and Policy
NRC Staff Perspective**

E. Vincent Holahan, Ph.D.

Senior Level Advisor
Office of Nuclear Regulatory Research

April 8, 2008

1

Outline

- System of Radiological Protection
- Technical basis review
- Where is the science today?
- How might the science impact NRC regulations?

2

System of Radiological Protection

- Three basic fundamentals
- Dose-based system
- Assumptions
 - LNT for stochastic health effects
 - Gender / Age averaged
 - Protect the most exposed individual
- Coherent and Predictable
- 10 CFR Part 20 – last major revision (May 1991)

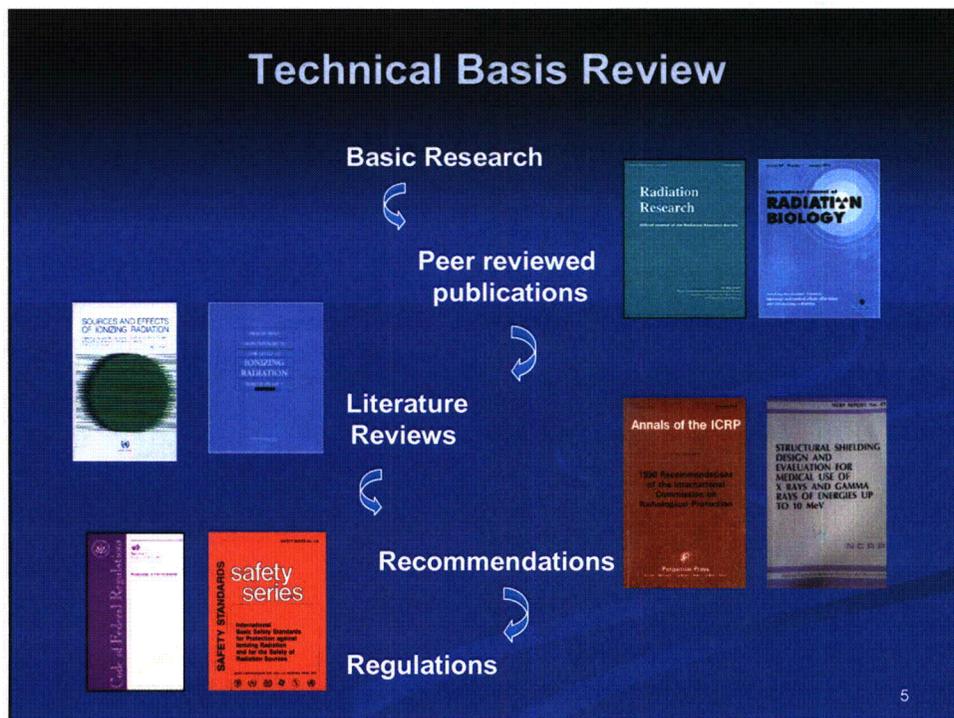
3

What would prompt a staff recommendation to revise NRC regulations?

- When a revision would prompt a substantial increase in the overall protection of the public health and safety or the common defense and security and that the direct and indirect costs associated with the change are justified in view of the increased protection. (Backfit)
- Updated scientific information
- Reduction in burden
- Risk informed Regulation
- Interagency alignment

4

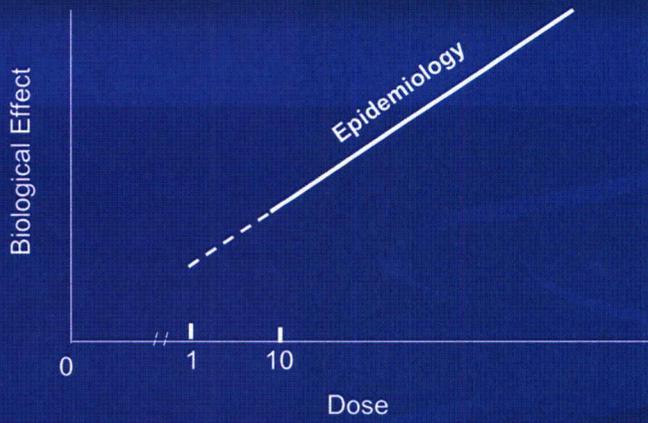
Technical Basis Review



Major technical developments

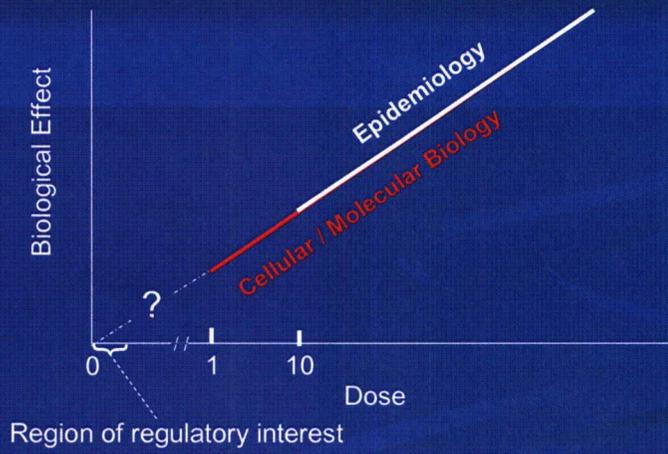
- Developments in basic science (e.g., DOE low dose research program, JCCRER, RERF, IARC)
 - UNSCEAR Reports (2000 – 2006)
 - BEIR V (1990) and BEIR VII (2005)
 - French National Academy report (2005)
 - ICRP Reports 60 – 103
- A small number '6' is visible in the bottom right corner of the slide.

Science and Radiation Protection



7

Science and Radiation Protection



8

Low Dose Radiation Phenomena

- Bystander effects
 - cellular damage response signals may be passed from an irradiated cell to a non-irradiated neighbor
- Genomic instability
 - Radiation exposure alters the state of a cell in a way that generally leads to a persistent elevation of mutation rate over many generations
- Adaptive response
 - Low priming dose of radiation influences the subsequent response to a second higher dose
- Hyper-radiation sensitivity
 - Modest increase in cell killing associated with low doses of x- or gamma-radiation exposure

9

What issues might prompt regulatory change?

- Reduced threshold for lens opacification.
- Increased incidence of non-cancer diseases.
- Significant difference in gender sensitivity to radiation.
- Significant difference in age sensitivity to radiation.
- Protect the most sensitive vs the most exposed individual.

10

Civil Rights Act - 1964

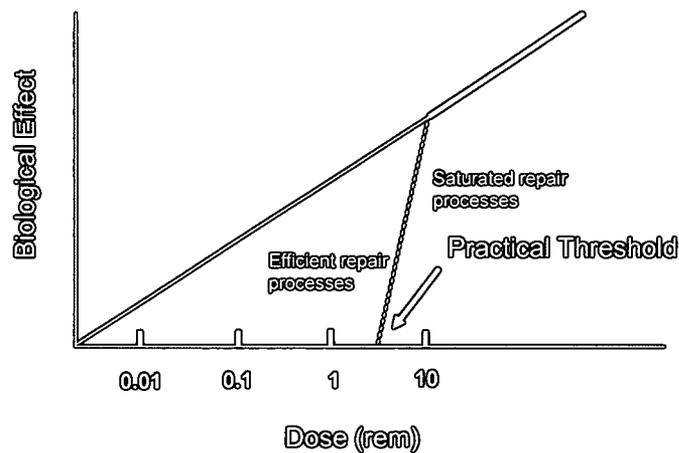
United Auto Workers vs Johnson Controls

The Supreme Court overturned a U.S. Court of Appeals decision that banned women from working in areas where they would be exposed to lead. It held that Title VII of the Civil Rights Act of 1964, as amended, forbids sex-specific fetal-protection policies. The majority of the court concluded with a single statement:

"It is no more appropriate for the courts than it is for the individual employers to decide whether a woman's reproductive role is more important to herself and her family than her economic role. Congress has left the choice to the woman as hers to make."

11

Science and Radiation Protection



12

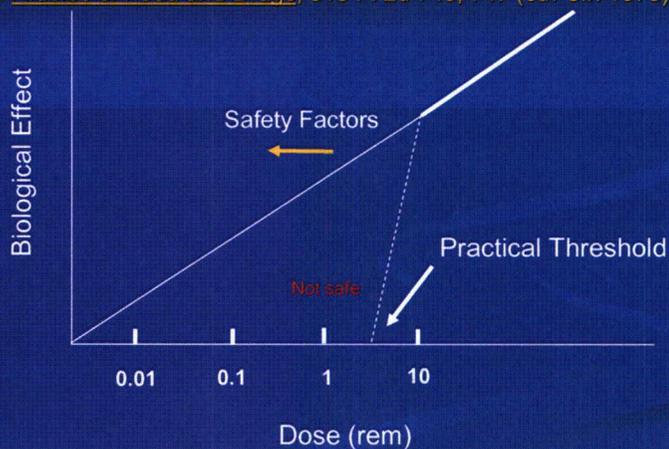
How might a threshold impact our system of regulatory protection?

- What exposures must be monitored and recorded?
- Is there a single or multiple thresholds?
- If so, which threshold should we regulate from?
- Does historical exposure fade?
- Is the practical threshold an annual or lifetime threshold?
- How are workers with different exposure histories managed?

13

Science and Radiation Protection

Lack of studies showing adverse effects of a substance cannot establish general recognition of safety. United States v. Articles of Food and Drugs, 518 F. 2d 743, 747 (5th Cir. 1975).



14

Safety Factors

- Acceptable levels of human exposure to toxicants in environmental and occupational settings generally are derived by reducing experimental no-observed-adverse-effect levels (NOAELs) by a product of uncertainty factors. These factors are presumed to ensure safety by accounting for uncertainty in dose extrapolation, uncertainty in duration extrapolation, differential sensitivity between humans and animals, and differential sensitivity among humans. The common default value for each safety factor is 10.
- Carcinogens or suspected carcinogens are excluded from this system of regulatory protection by EPA or FDA.

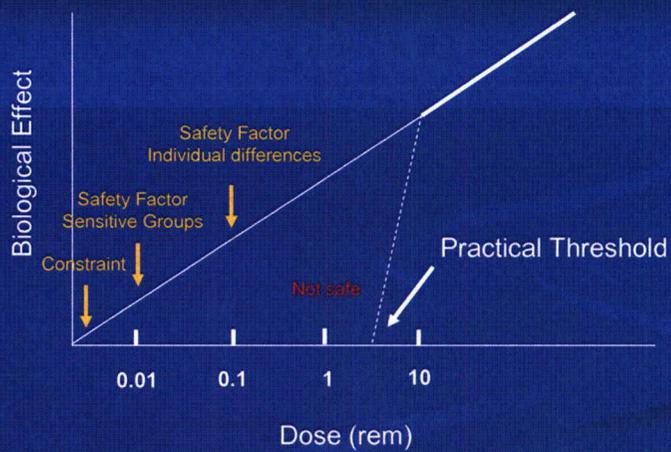
15

Safety Factors

- FDA uses a safety factor of 200-2000 to account for animal to human safety, below the NOEL.
- 1996, Food Quality Protection Act, set tougher standards to protect infants and children. Additional safety factor of 10.

16

Science and Radiation Protection



17

Minimum Risk Level

Ionizing Radiation¹

- NOAEL - 360 mrem / yr (chronic exposure)
- UF (for human variability) – 3
- MRL – 100 mrem / yr
 - ▶ however,
 - Human variability could be higher (UF 10)
 - No UF for infants and children included
 - Source constraints

¹ Agency for Toxic Substances and Disease Registry, HHS (Sept. 1999)

18

Conclusions

- NRC regulations and standards are adequately protective of public health and safety.
- Adoption of new biokinetic models, risk coefficients, weighting factors, etc. will not significantly improve public health and safety.
- No radical developments anticipated in the near future.

19

Thank you !

Comments ?

20



EPA Perspective

April 9, 2008

Presented by:

Jerome S. Puskin

US EPA/ Radiation Protection Division

Why we use LNT

Default for mutagens

Epidemiological studies have insufficient statistical power to test LNT at very low doses

So far, biological research has not filled the gap left by epidemiology at low doses

Advice from ICRP, NCRP, NAS

- BEIR VII (NAS) says scientific weight of evidence still favors LNT



Scientific basis for LNT

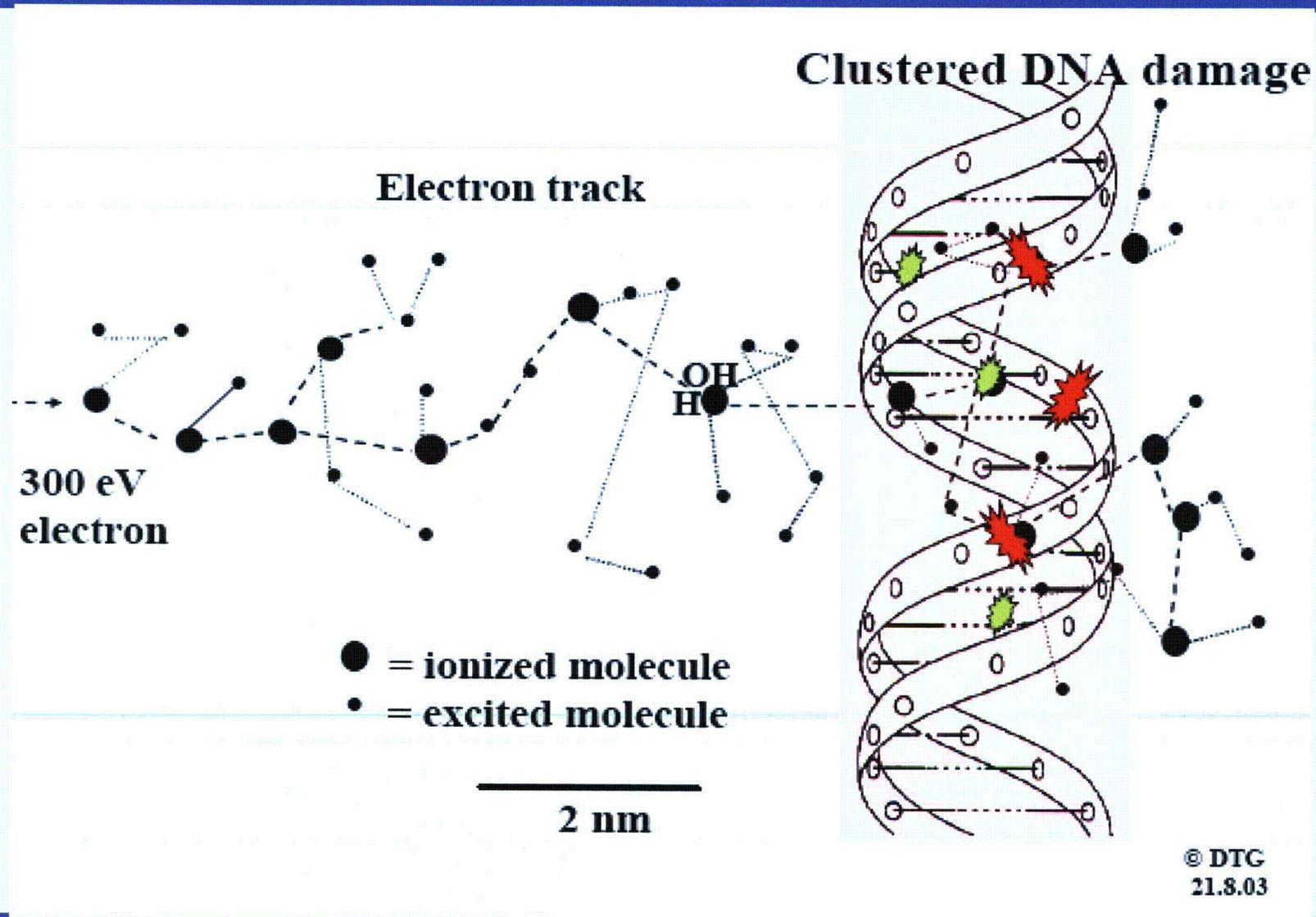
Animal and human data generally consistent with linearity of risk down to lowest doses for which we have statistical power to measure

Single ionizing tracks produce clusters of ionizations leading to complex damage in DNA, which cannot always be faithfully repaired

Evidence that a single mutation in a cell can increase the probability that the cell will become malignant



Generation of clustered damage



What is a threshold?

A threshold might be strictly defined as a radiation dose (or dose rate) below which no harm to any individual in a population would occur

For regulatory purposes, however, a “practical threshold” might be adopted if there were compelling evidence that, below this level, the risk is much lower than predicted by LNT, but not necessarily zero



Low Dose Threshold?

Epidemiology is sensitive down to ~100 mGy (low-LET) above background.

Each person receives about 75 mGy, lifetime, from background + a greater amount, on average, from medical exposures and other sources.

If there were to be a “threshold”, it would most likely reflect a dose rate or a dose increment received over some critical time period – *e.g.*, the time required for DNA repair.



Low Dose Phenomena

Adaptive response (+)

Bystander effects (+/-)

Genomic instability (-)

Low-dose hypersensitivity (+/-)

(+) *Potentially protective*

(-) *Potentially risk enhancing*



Epidemiology generally trumps radiobiology

Before a threshold is accepted, we would probably need confirmation with human epidemiological data – or, at least, with some sort of biomarkers in human tissues that clearly relate to cancer.



*Contrary to some assertions, there is
epidemiological evidence for risks
below 100 mSv.*



Epidemiological evidence of carcinogenic effects at low doses and low dose fractions

Prenatal x-rays (5-10 mGy x-rays)

TB patients (♀) (8 mGy x-ray fractions)

Scoliosis patients (♀) (4 mGy x-ray fractions)

In the above, excess cancers were observed in subjects receiving no more than a few ionizing tracks to any one cell during a week or so.

Tinea Capitis (17 mGy daily fractions)



Ongoing epidemiological studies of chronically exposed cohorts

Chernobyl population and “liquidators”

Mayak workers

Techa River Cohort

Semipalatinsk population

Occupants of ^{60}Co -contaminated buildings in Taiwan

Nuclear workers

Such studies may provide evidence of risk at 0.1-1 mGy/day, or even lower



Risk Principles Applied to Standards

Radiation protection standards need to account for uncertainty

- Need to ensure that we are not greatly underestimating risks

Changing regs based on new science

- If evidence shows regulations are too lax, rules likely get strengthened
- If evidence shows the opposite, rules *may* be relaxed (if statute permits; if there is a compelling need; ...)



Before rejecting LNT, EPA would want –

Scientific consensus (as reflected in reports from NAS, UNSCEAR, NCRP, ICRP, etc.)

Concurrence from EPA's Science Advisory Board

Acceptance among Federal agencies

A transparent public process for considering scientific evidence



Regulating with a threshold

A threshold below the level of radiation received from unavoidable sources would have no impact on current regulations

A practical threshold substantially above background might mean certain regulations could perhaps be relaxed or reinterpreted, including:

- Derived soil cleanup levels
- Drinking water MCLs



Issues in setting threshold-based standards

Magnitude of threshold dose or dose rate

Uncertainty in threshold dose

Consideration of sensitive subpopulations

Contribution of multiple sources

- Example: If threshold = 10 mSv/y, then an individual source limit might be set at 1 mSv/y



Downsides of LNT

Actions taken to limit the estimated risk may not be warranted from a cost-benefit standpoint

Perception of a finite risk at low doses may cause members of the public to oppose beneficial nuclear technologies or to shun advisable medical procedures



Living with LNT

Education – help public to put risks into perspective and to balance risks and benefits

- Risk is unavoidable
- LNT implies risks from low doses are low

Attempts to deny or to minimize risks, in the absence of convincing scientific evidence, can damage the credibility of radiation protection community



Summary

Radiation protection is currently based on LNT, consistent with current science and recent NAS recommendations

Before adopting a threshold, EPA would need a scientific consensus

Compelling evidence for a threshold might influence environmental standards

A change in standards would require statutory authority and need to consider safety factors (multiple sources, sensitive subgroups, etc.)



An Economic Perspective on Regulatory Decision Making: Benefit-Cost Analysis under Linear & Nonlinear Models

James K. Hammitt

Harvard Center for Risk Analysis

Outline

Economic perspective on decision making

Context for decision

Individual or population

Exposure and exposure-response function known
or uncertain

Example: Radon in drinking water

Economic Decision Making

Maximize wellbeing

Health

Resources available for other uses

Choose exposure level to

Minimize harm or maximize benefit to health

Minimize control costs

→ Compare benefits of health with costs of control

Value of health improvement defined as

Willingness to pay (WTP) for improvement

= Maximum value of other goods one would forgo

Economic Evaluation of Regulation

Choose alternative to maximize

Net Benefits = Benefits - Costs

Health benefit = product of

Number of people affected

Willingness to pay (WTP) for individual risk change

– Maximum value of other goods one would forgo

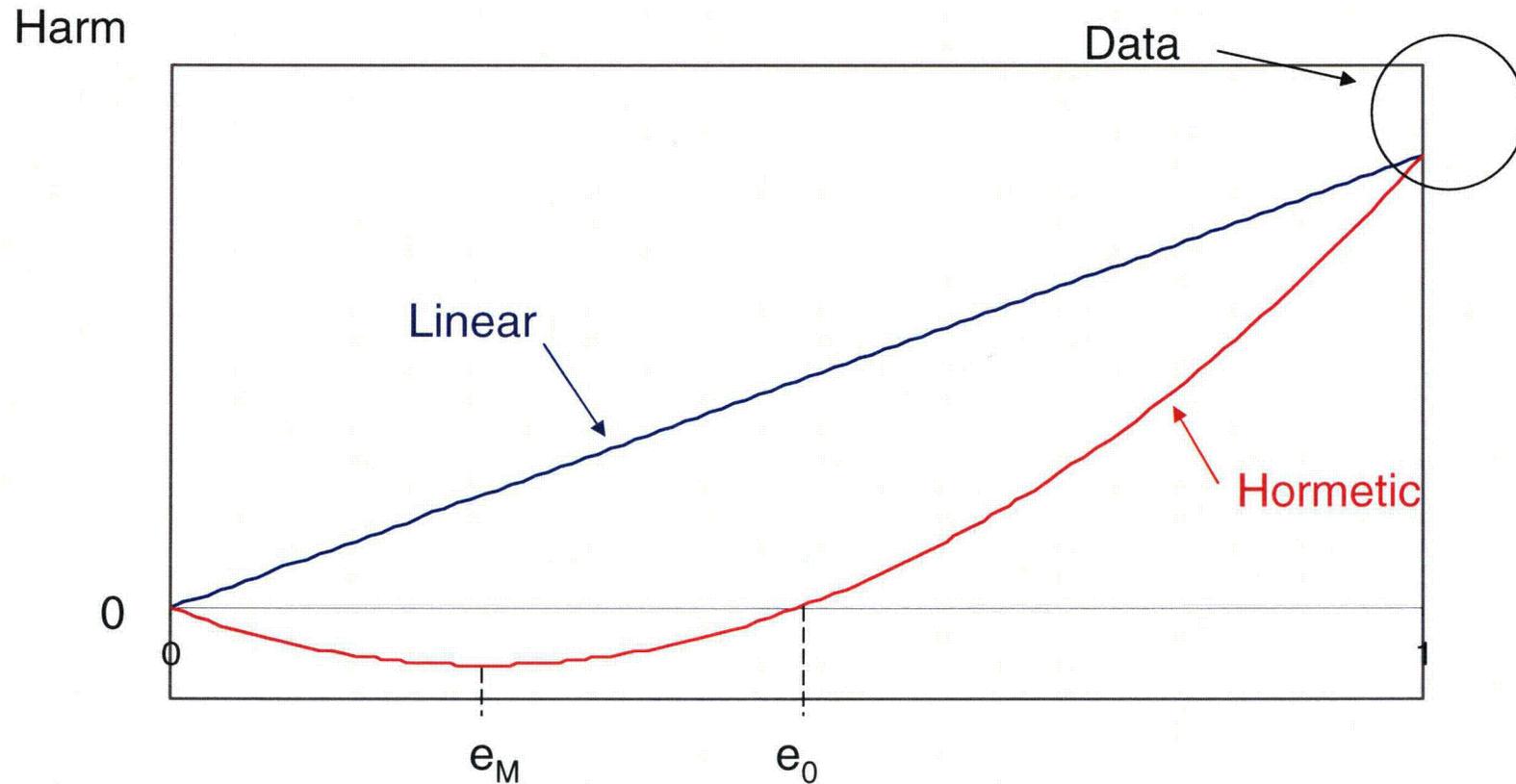
= product of

Expected reduction in number of cases

Value per statistical case

Hormetic exposure-response is

Steeper at high exposure
Flatter at low exposure



Optimal Control of Exposure

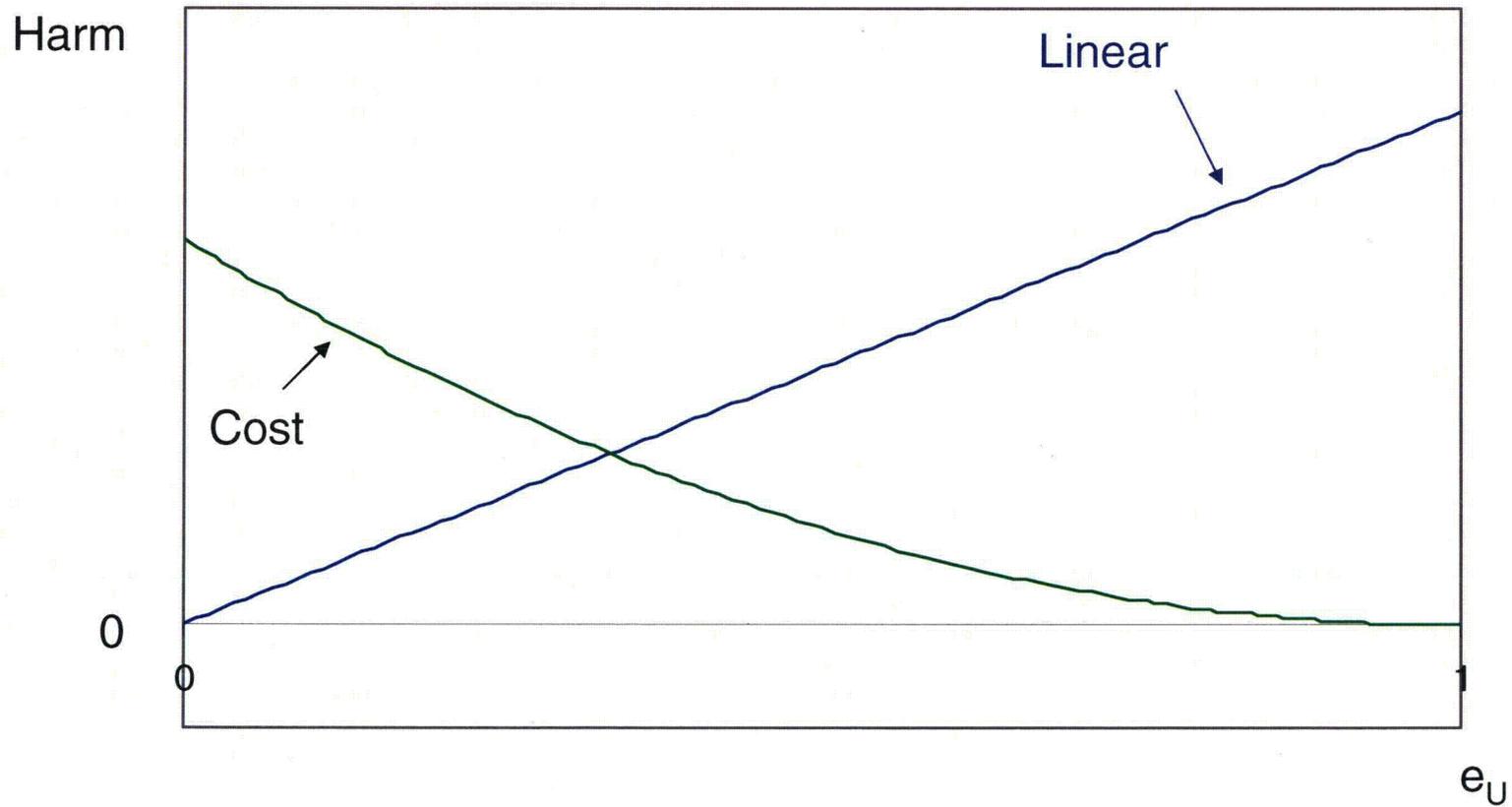
Decision for individual

Exposure and exposure-response
function are known

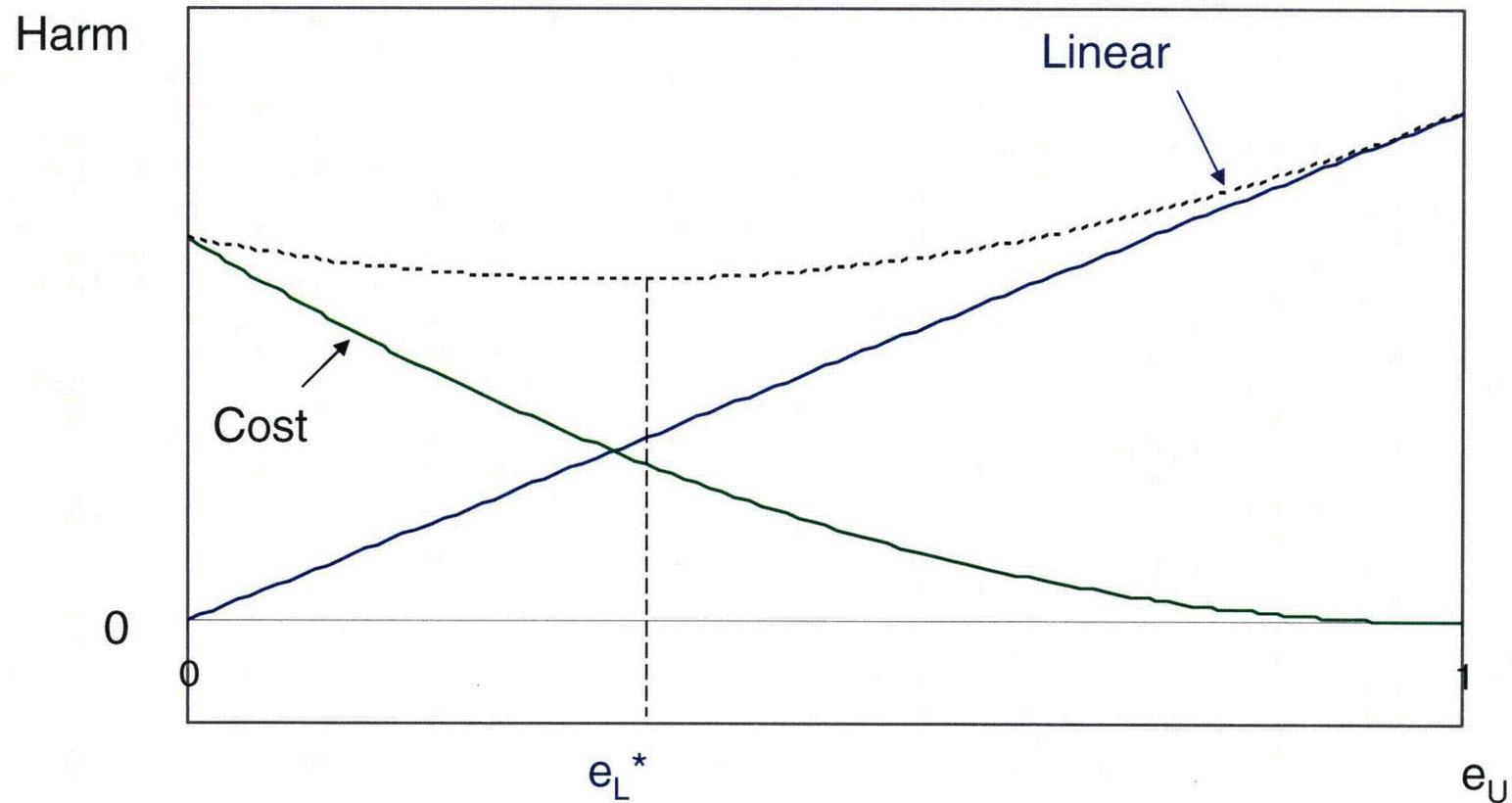
Simplest case

Unrealistic, but helpful to examine

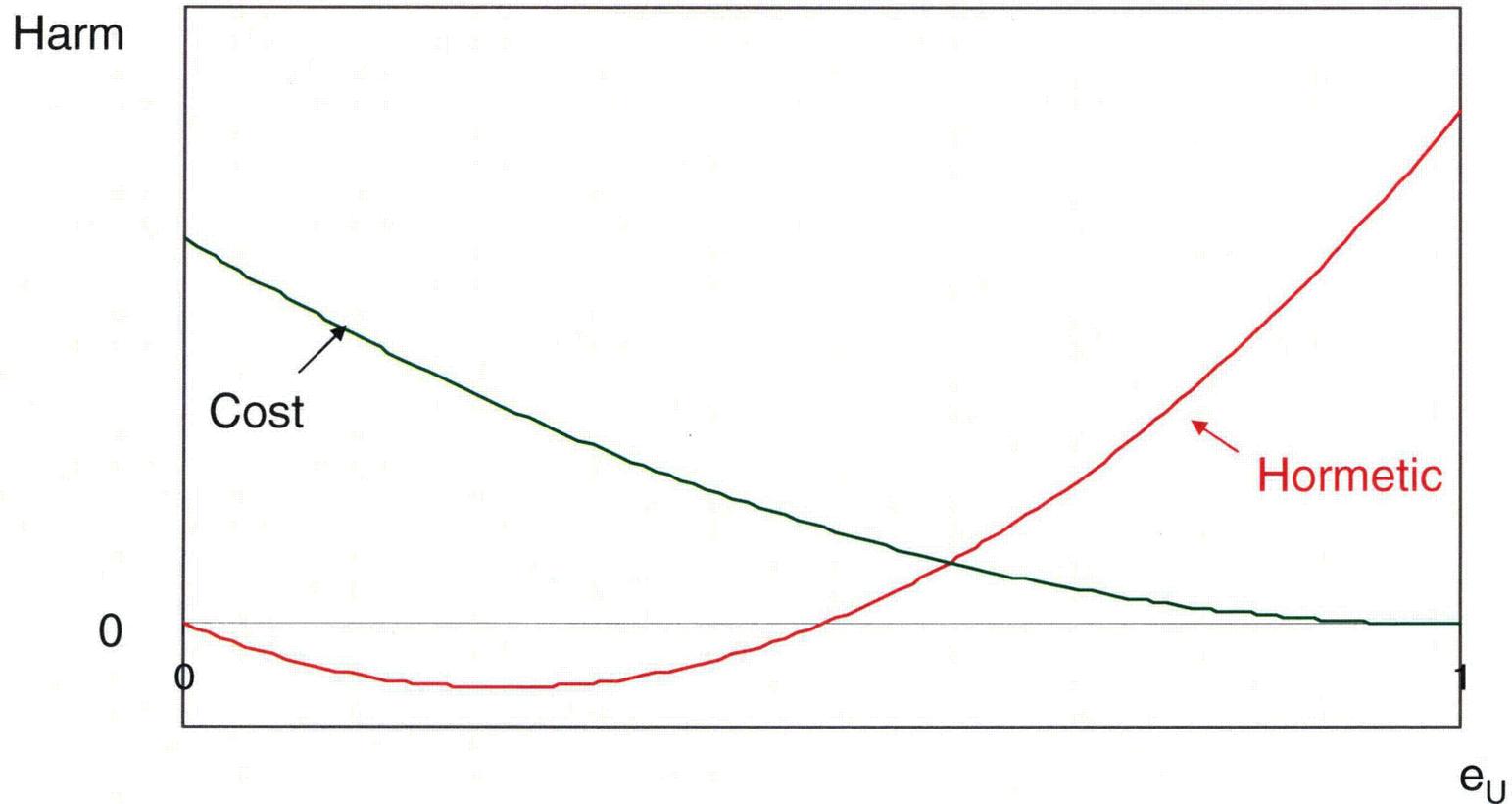
Individual, Known Exposure-Response



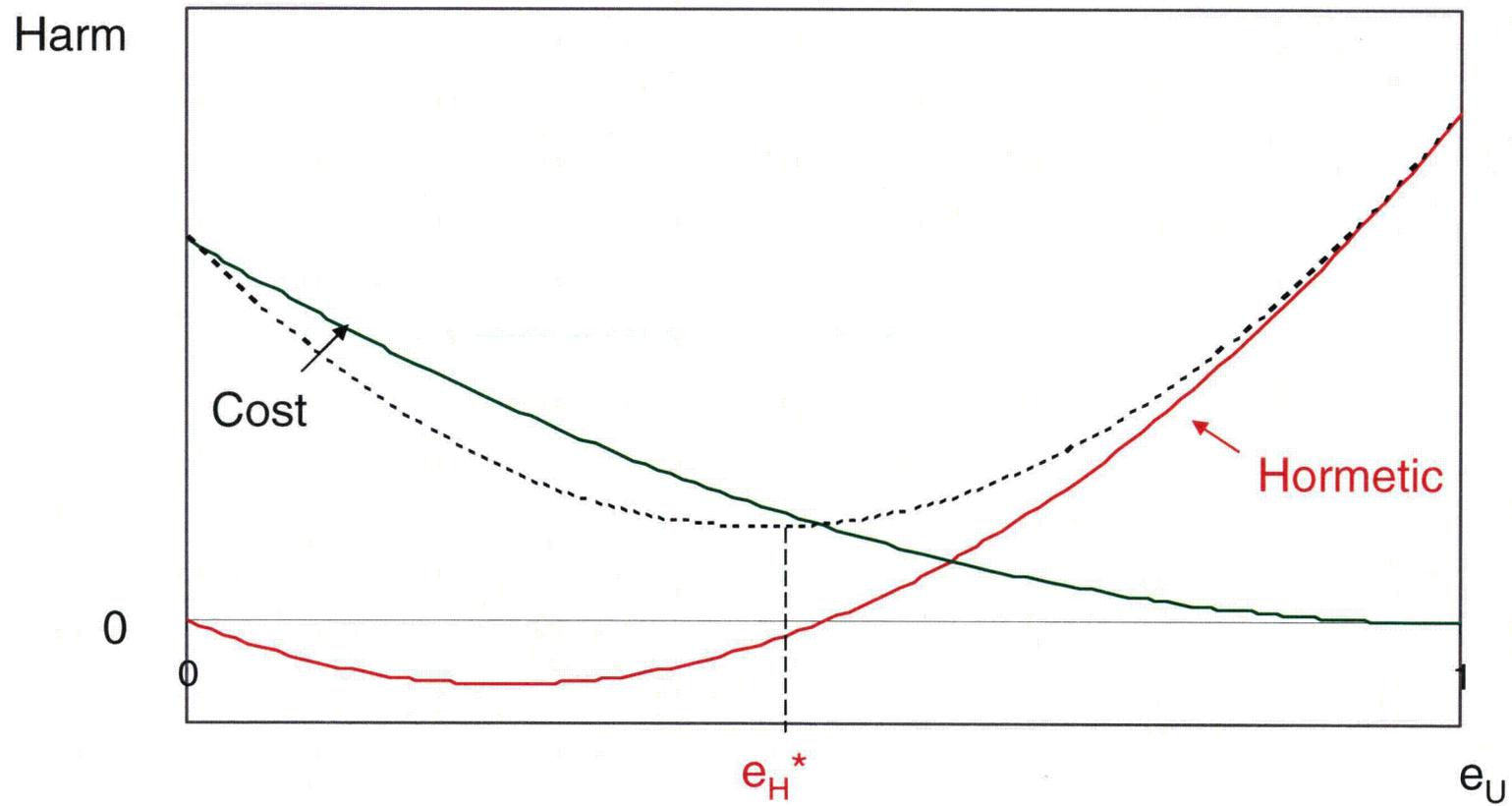
Individual, Known Exposure-Response



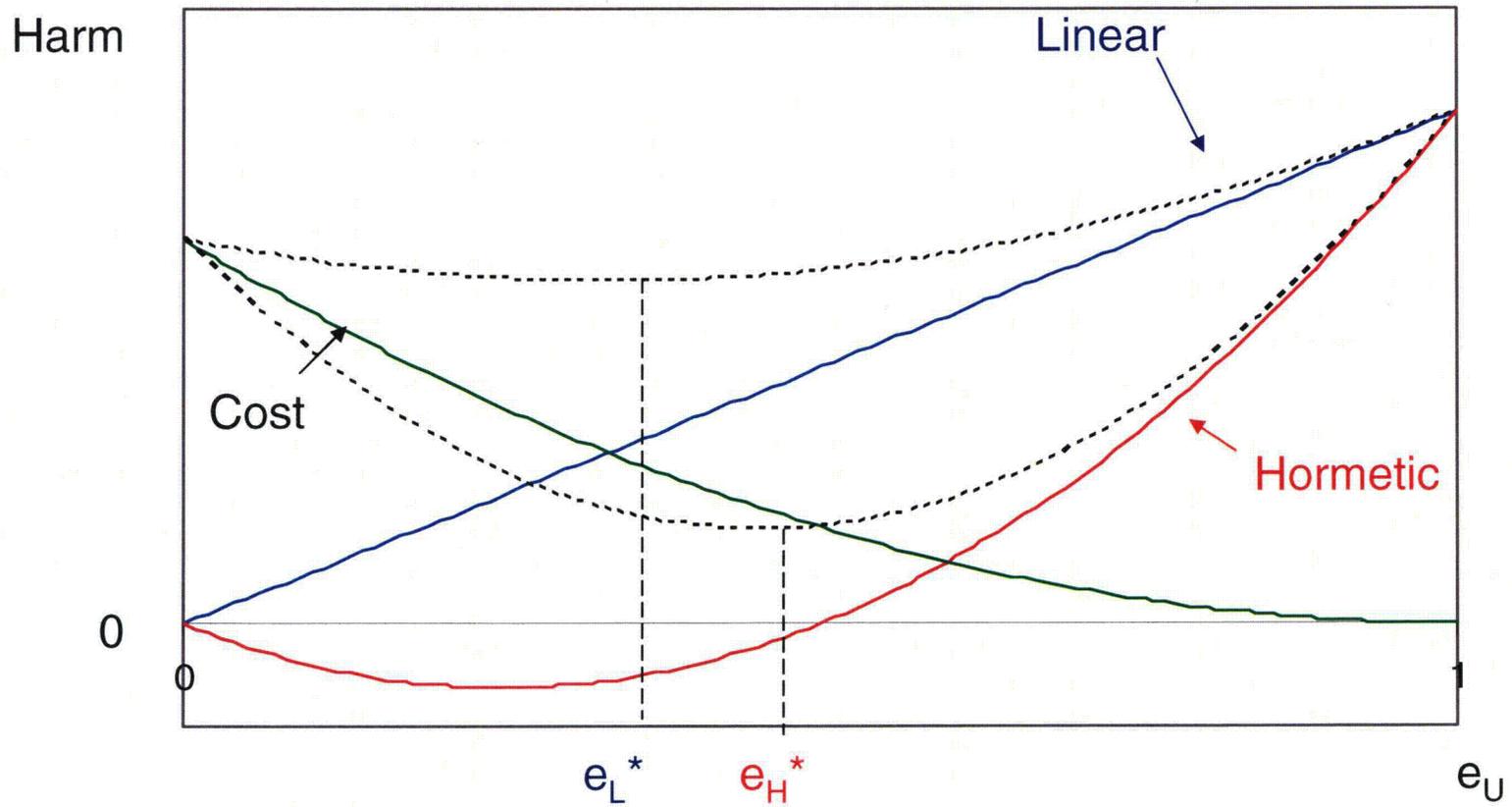
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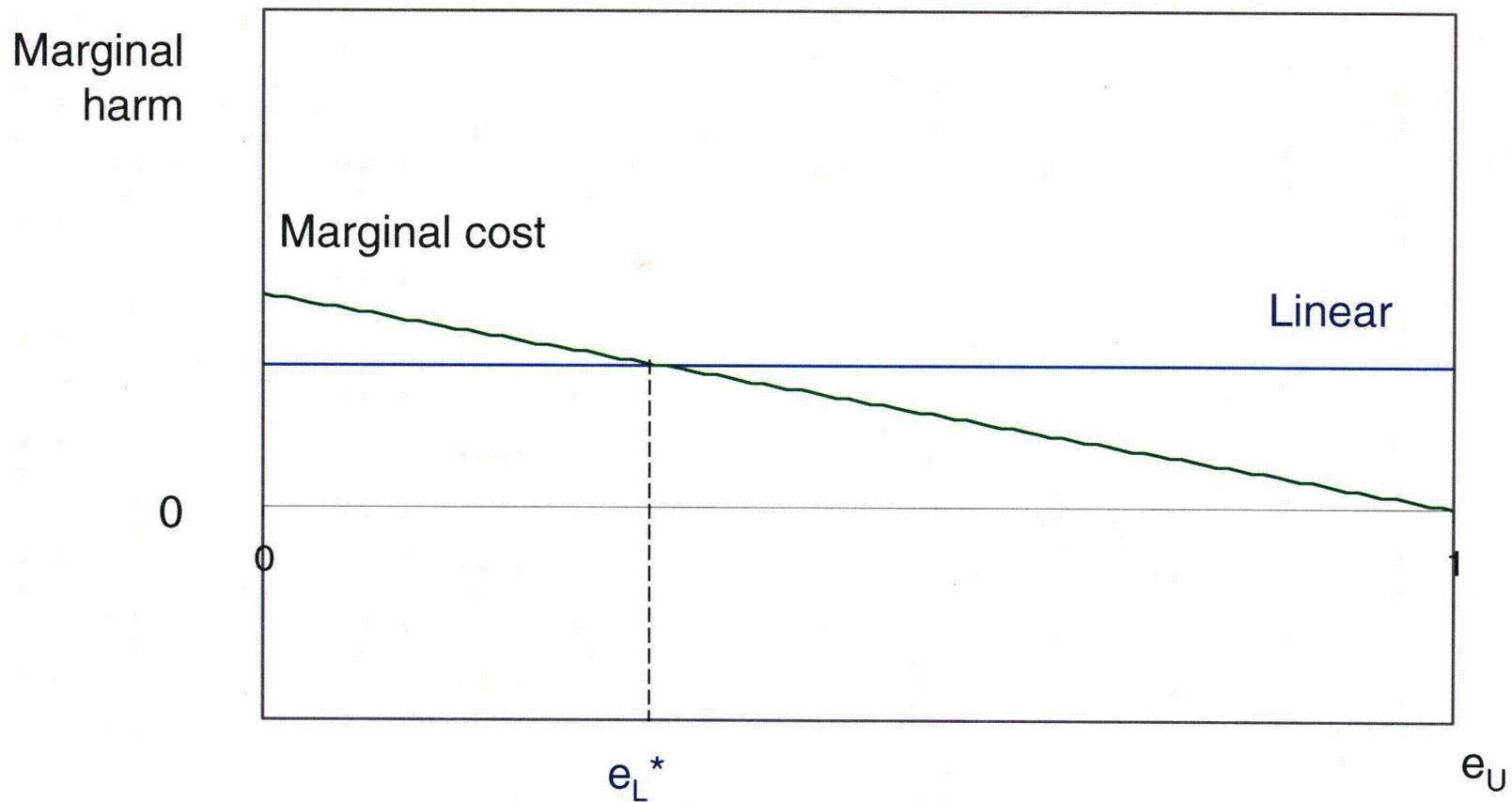
Individual, Known Exposure-Response



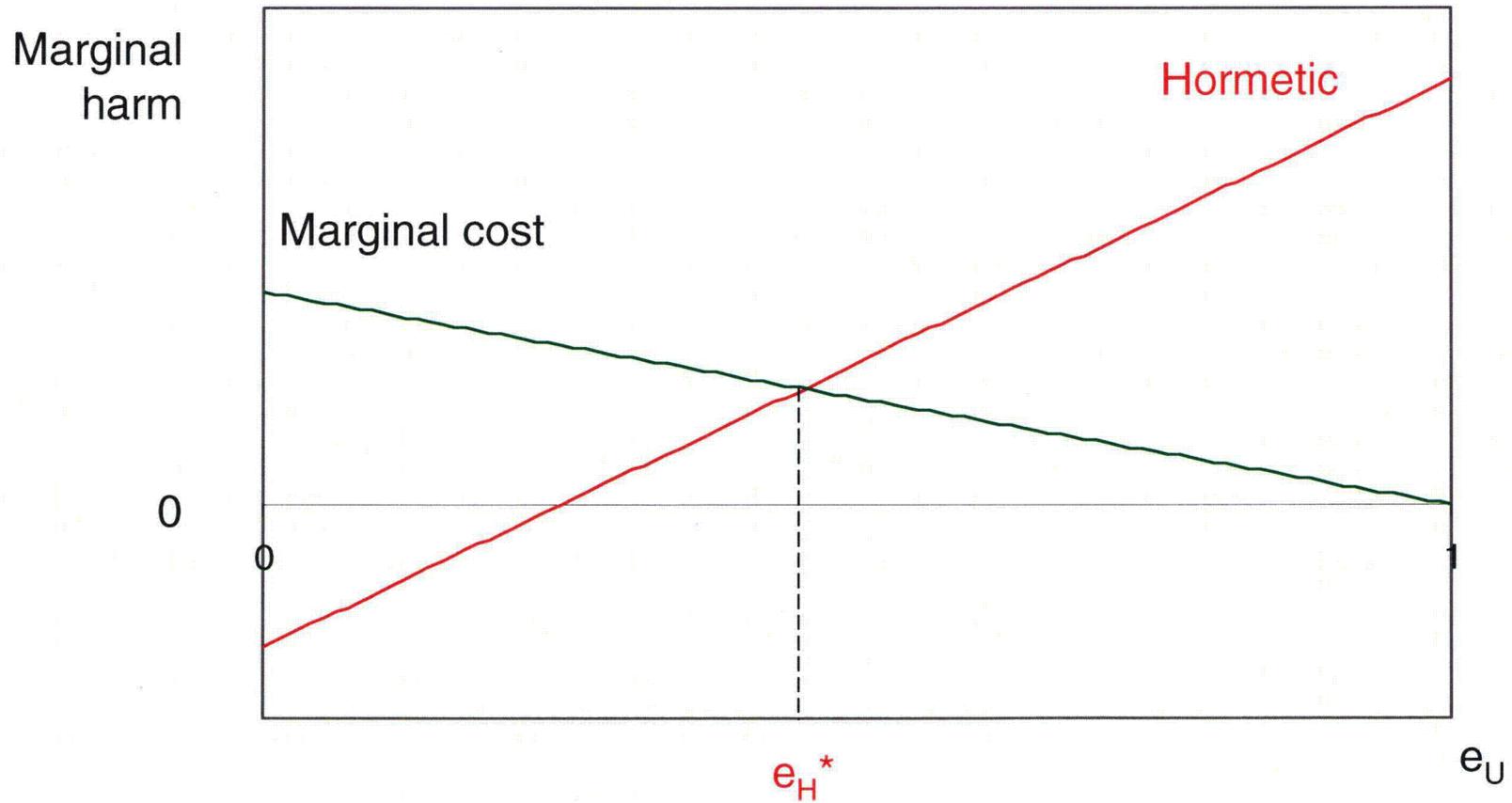
Individual, Known Exposure-Response



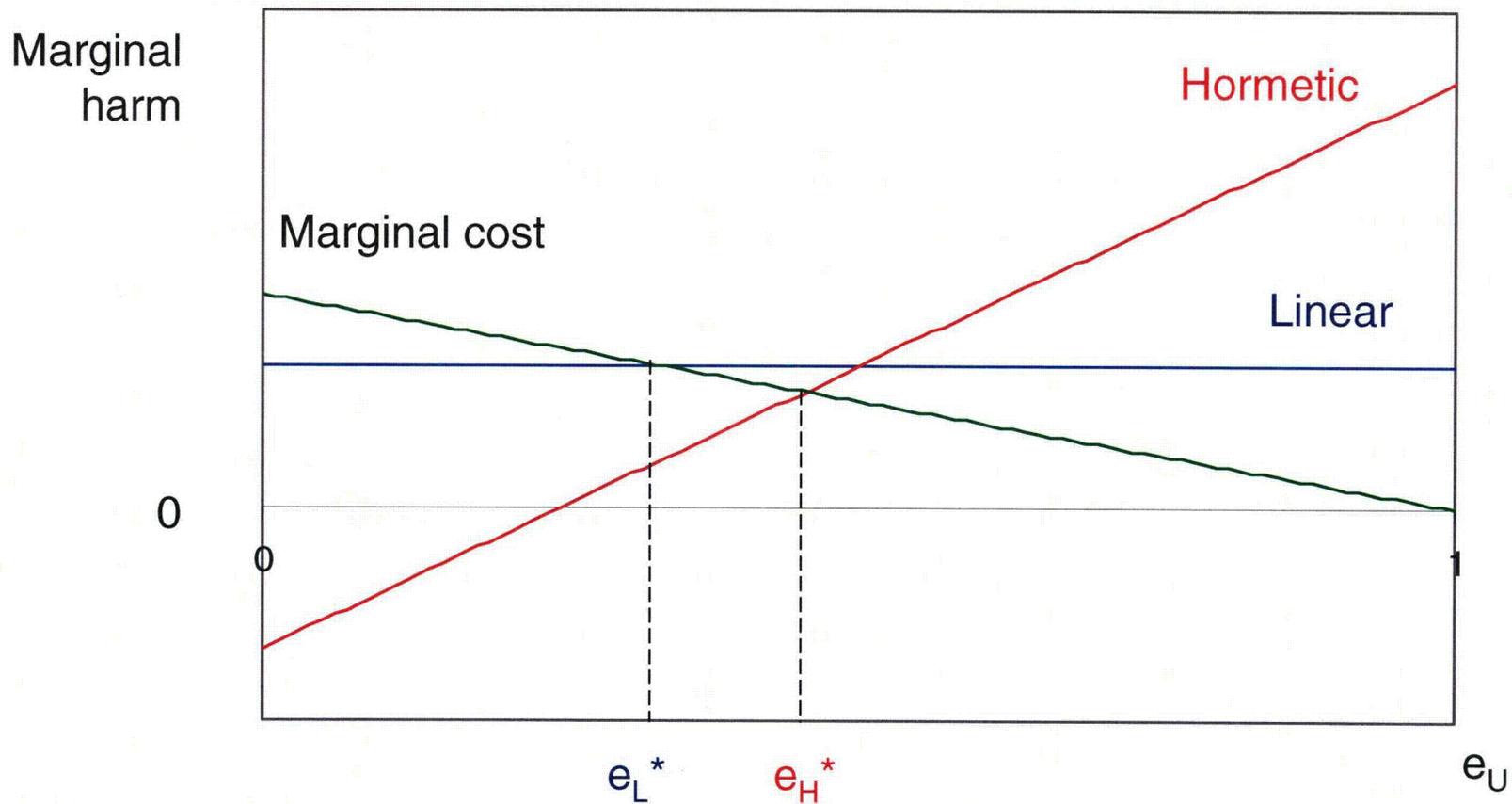
Individual, Known Exposure-Response



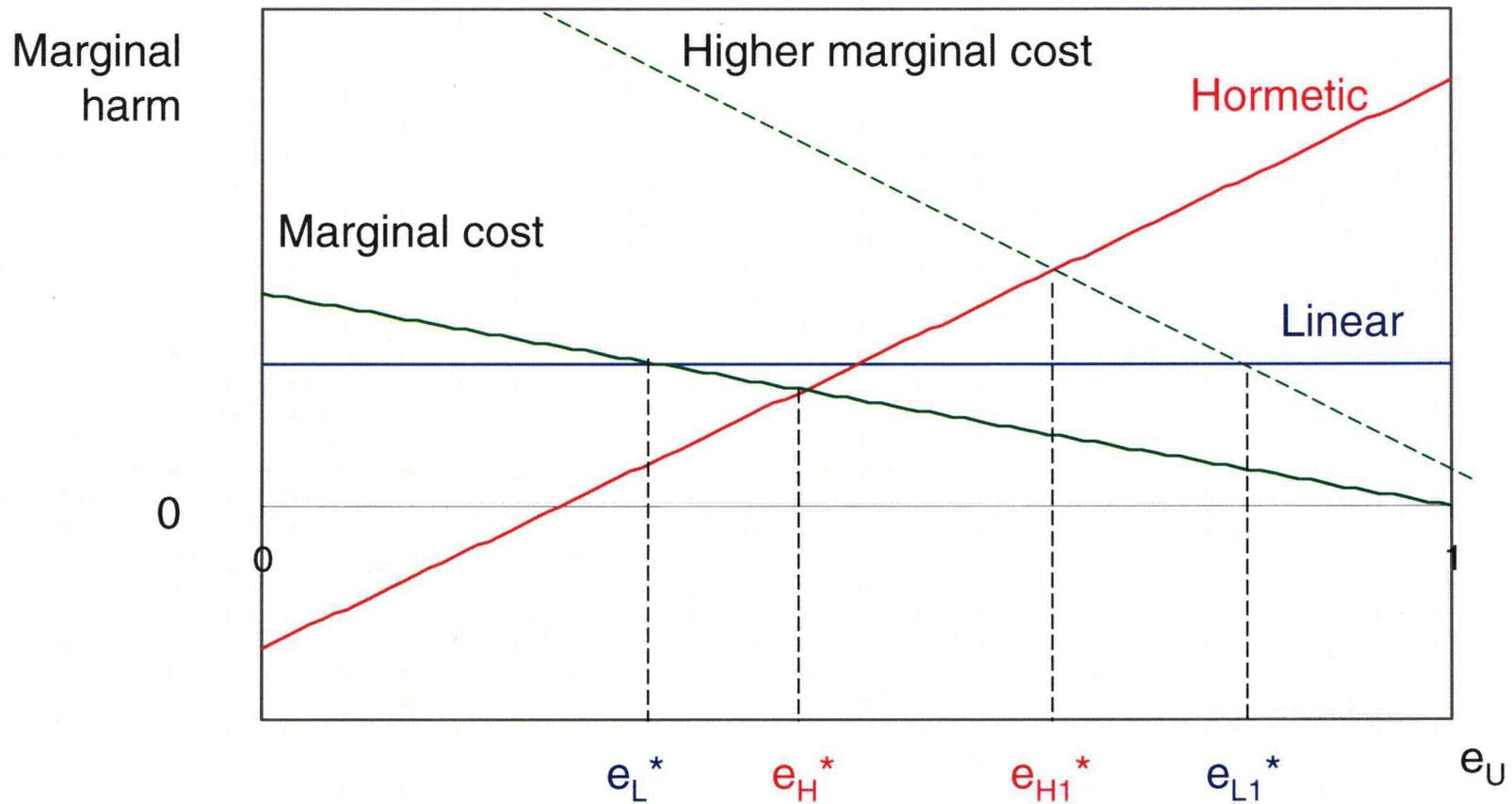
Individual, Known Exposure-Response



Individual, Known Exposure-Response



Individual, Known Exposure-Response



Optimal Control of Exposure

Decision for individual

Exposure-response function uncertain

Describe uncertainty using probability

Minimize expected value of harm + costs

Expected marginal benefit = marginal cost

Individual, Uncertain Exposure-Response

If exposure-response may be linear or hormetic

Expected harm =

$$p * E(\text{harm if linear}) \\ + (1 - p) * E(\text{harm if hormetic})$$

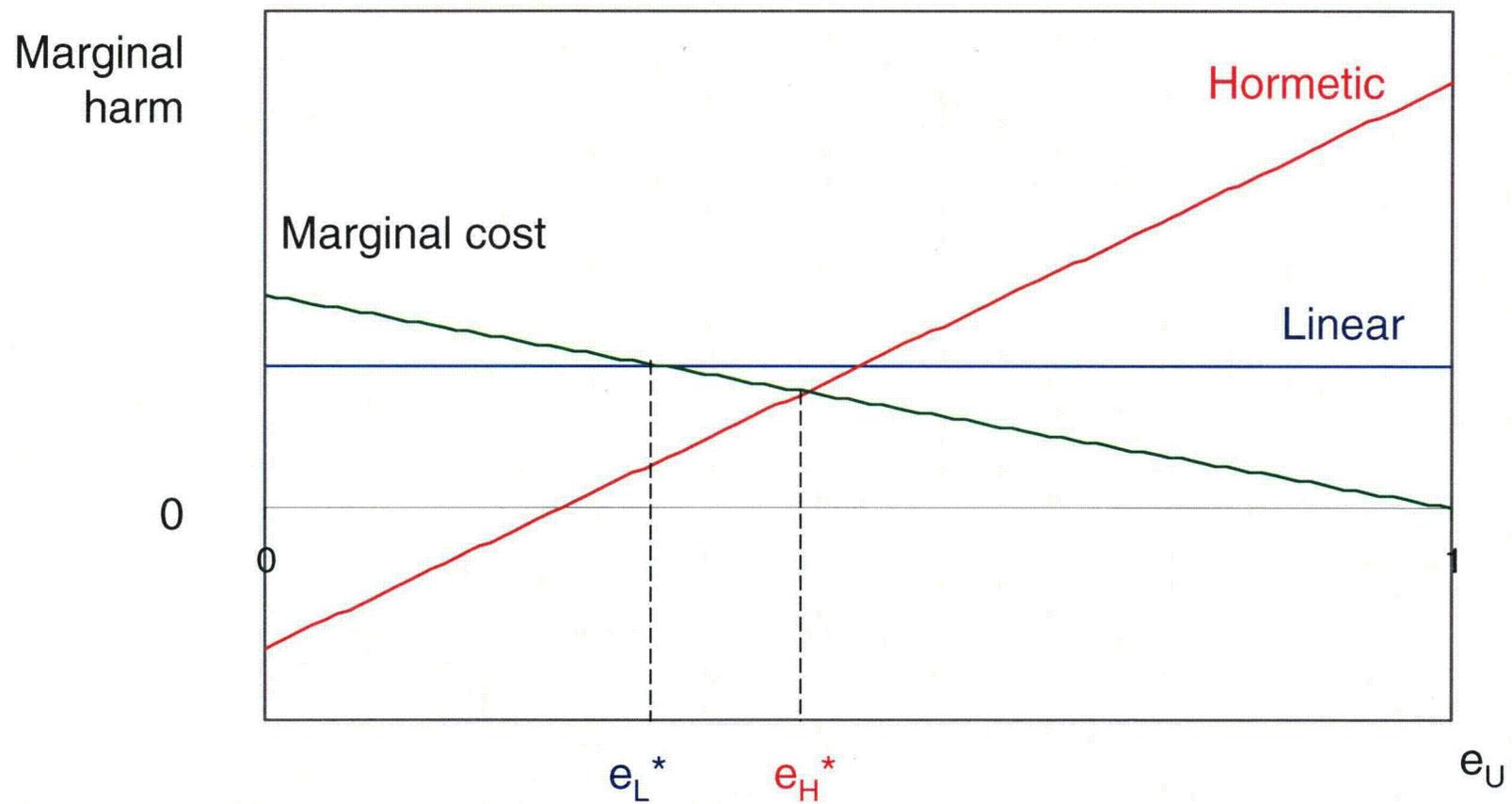
Expected marginal benefit =

$$p * E(\text{marginal benefit if linear}) \\ + (1 - p) * E(\text{marginal benefit if hormetic})$$

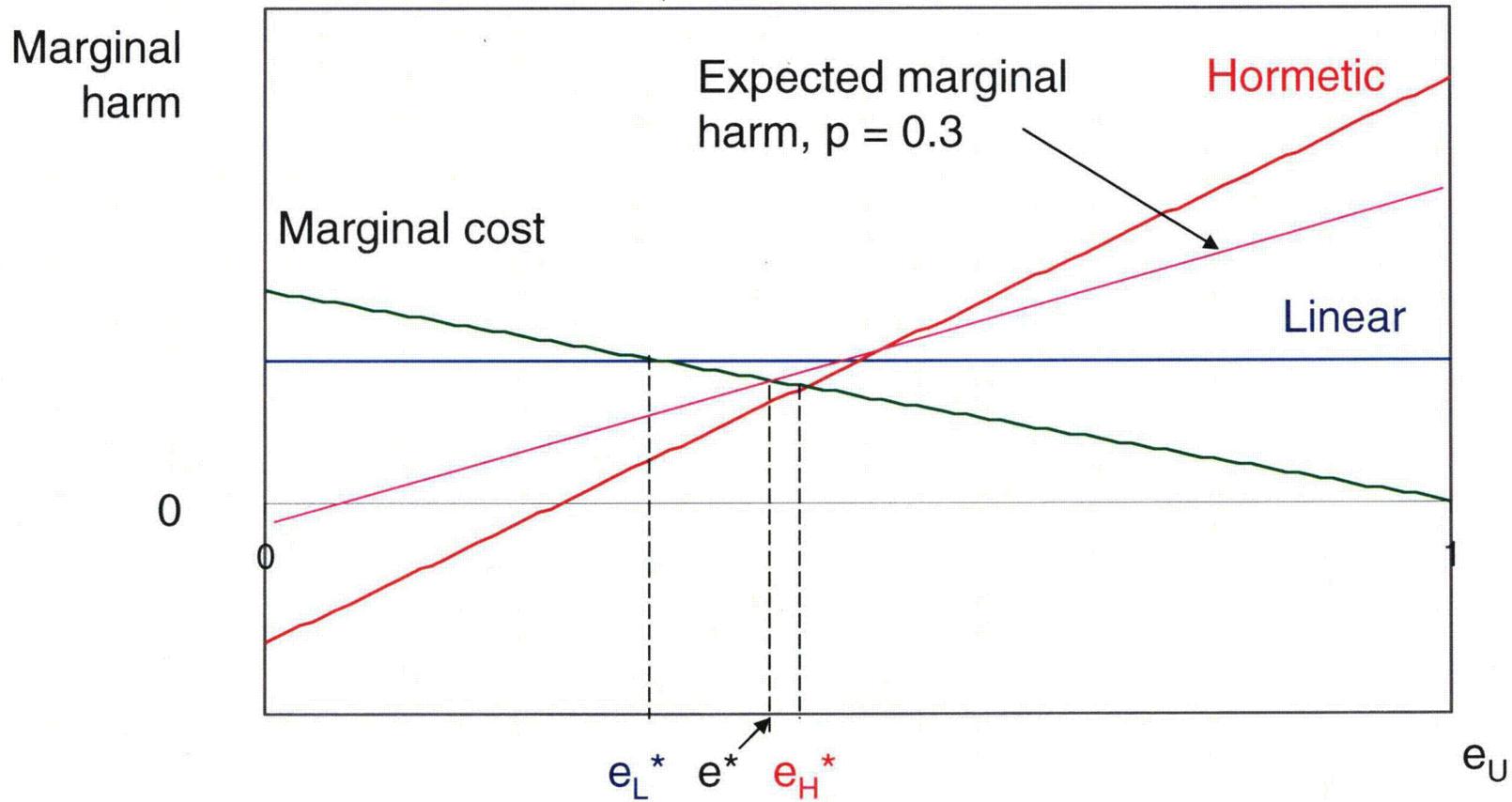
Solution is between linear, hormetic solutions

Depends on probabilities, marginal harms of alternative models

Individual, Uncertain Exposure-Response



Individual, Uncertain Exposure-Response



Uncertainty About Exposure-Response Function

If exposure is larger than e_0 , slopes of hormetic and linear models may be similar

Average slope of hormetic function

$$= \text{slope of linear model} / [1 - e_0/e_U]$$

Effect on optimal control level inversely proportional to slope of marginal cost function

If exposure smaller than e_0 , uncertainty is important

Reducing exposure may be beneficial or harmful

Population-Level Decision

Exposure, exposure-response function
may differ between individuals

May also be uncertain

Decision cannot be optimal for everyone

Balance more benefit to some against less benefit
to others

Social Welfare

No objective method for comparing
changes in wellbeing between people

Pareto improvement

No one is harmed, someone benefits

Social Welfare

Potential Pareto improvement

Those who benefit could compensate those who are harmed

Compensation converts change to Pareto improvement

Kaldor-Hicks compensation test

Add monetary value of changes across people

Benefits exceed costs → Potential Pareto improvement

Justifications for Kaldor-Hicks Compensation Test

Those who gain and lose on individual
decisions will vary over time

In the long run, everyone benefits from use of
decision rule

Redistribution can be better handled by
other means

Taxes, welfare systems

Population Effect of Exposure Reduction

Linear no-threshold model

Independent of individuals' exposure levels

Can estimate using average slope

Hormetic model

Dependent on individuals' exposure levels

Need to know exposure levels and slopes of
exposure-response function for subpopulations

Example: Radon in Drinking Water

Radon exposure associated with lung cancer mortality

EPA proposed regulations in 1999, published (draft) regulatory assessment

Primary exposure pathway is via indoor air

EPA has no authority to regulate indoor air

Bogen (1997, 2001) estimated hormetic exposure-response function for radon in indoor air

Policy Alternatives, Evaluation

Set maximum contaminant level (MCL) for community water system

Benefits

Estimate distribution of radon levels in drinking water

Calculate reduction of radon in water as function of MCL

Estimate change in indoor air concentration as 1/10,000 change in water concentration

Costs

Aeration or granular activated carbon treatment

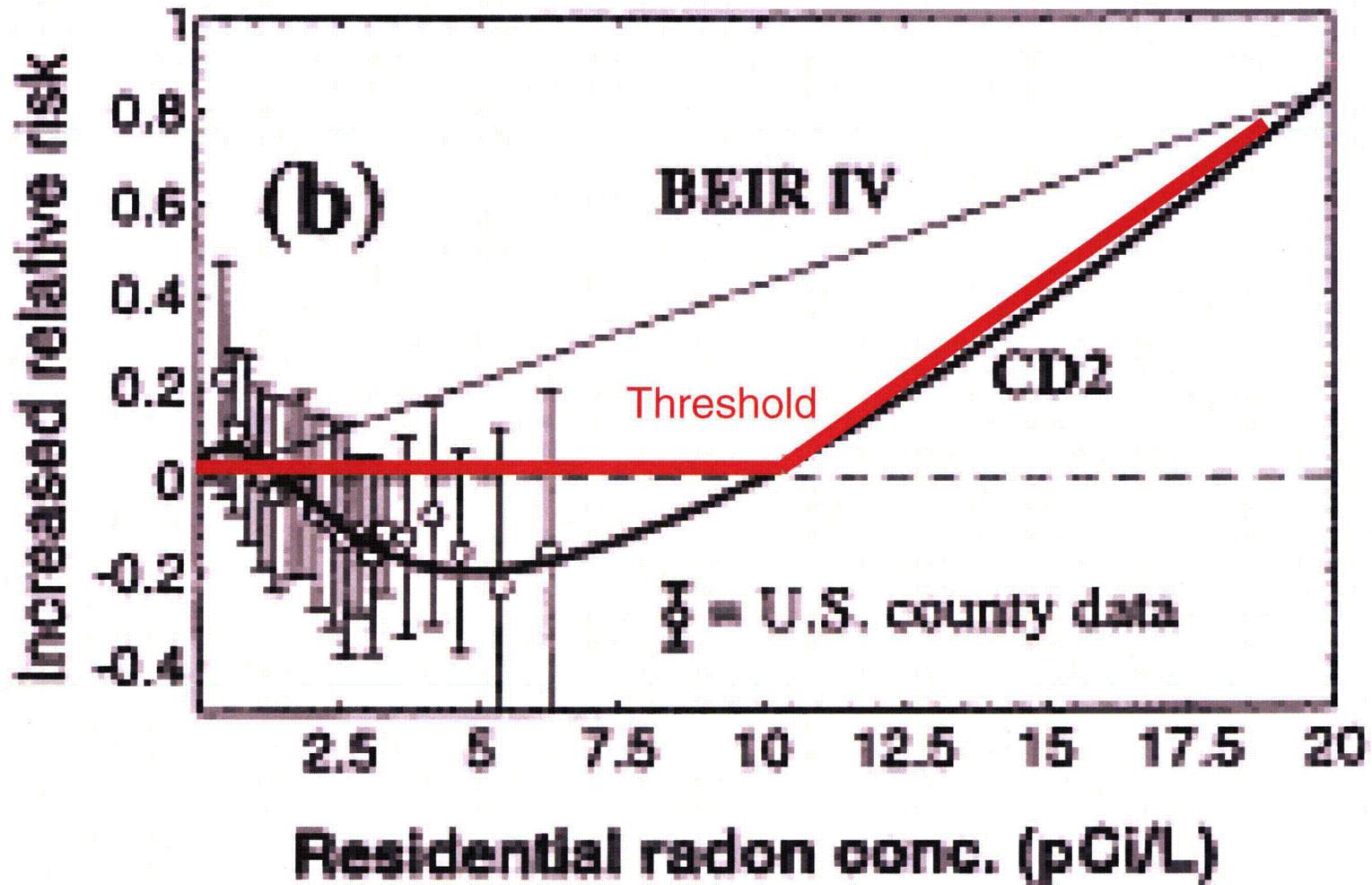
In example, use smaller costs (e.g., large system)

Change in Drinking Water Rn

(Change in air = 1/10,000 as large)

MCL (pCi/l)	Pop'n > MCL (1,000s)	Pop'n wtd mean Rn > MCL	Red'n in pop'n wtd mean Rn
4000	77	5,000	1,000
2000	380	3,400	1,400
1000	1,700	1,900	930
700	3,600	1,400	660
500	6,900	1,000	490
300	17,000	650	350
100	56,000	330	230
0	88,000	230	230

LNT and Hormetic E-R (Bogen, 1997)



Distribution of Rn in Indoor Air

Action level = 4 pCi/l

Only 5% of households exceed

Approximately lognormal

Geometric mean = 0.67 pCi/l

GSD = 3.1

98th percentile = 6.5 pCi/l

Hormetic exposure-response

Min (max beneficial effect) near 5 pCi/l

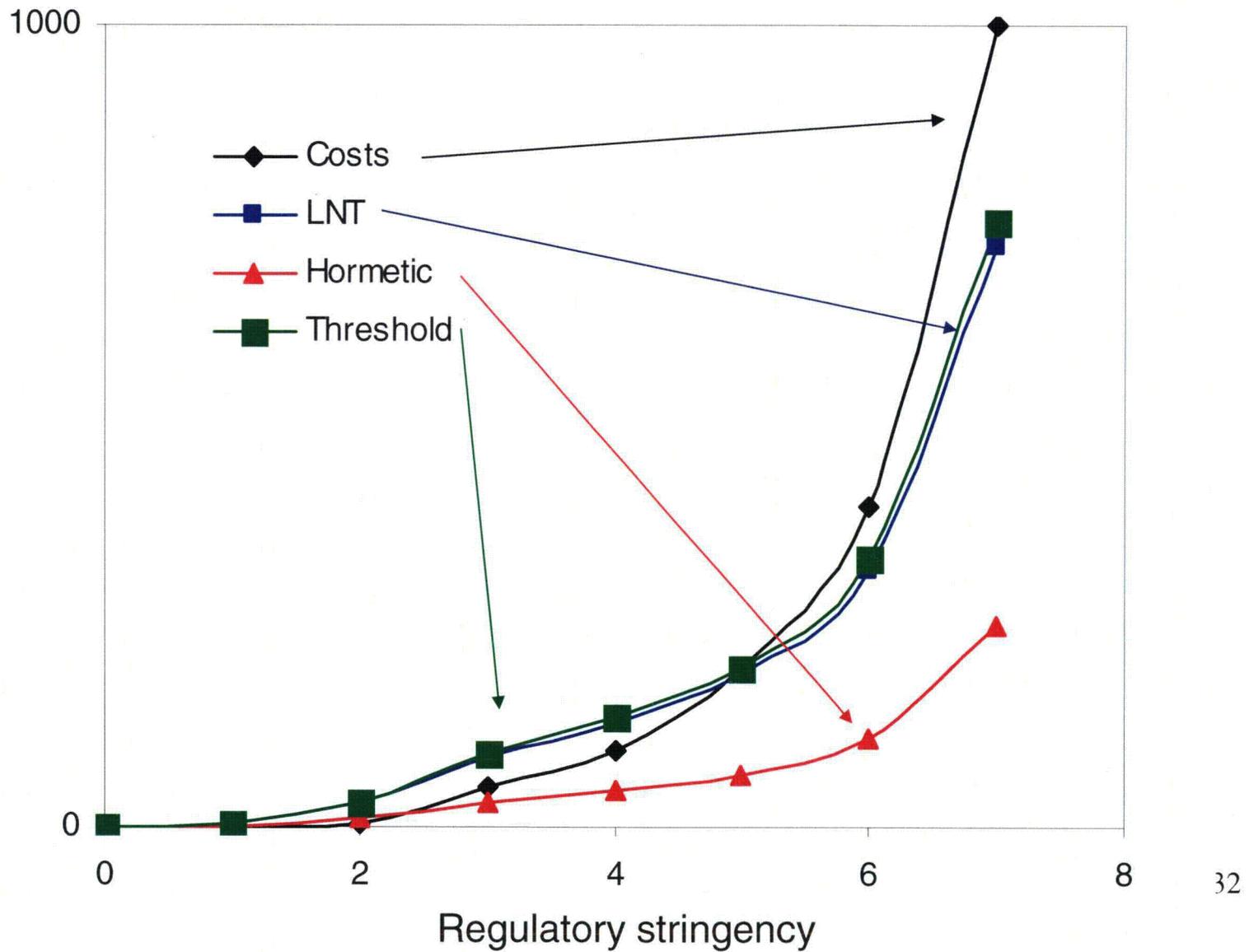
→ No benefit to reducing Rn levels in home, drinking water

Hypothetical Background Distribution

(High exposure region,
Independent of Rn in drinking water)

Rn (pCi/l)	Popn distn	Relative slope
2	25%	-2.8
5	25%	0
10	25%	1.8
15	25%	2.4
Mean	100%	2.1
LNT	100%	1

Total Benefits & Costs (\$M/yr)



Total (Net) Benefits & Costs (\$M/yr)

MCL	Costs	LNT	Hormetic	Threshold
4000	1	4 (3)	1 (0)	4 (3)
2000	5	30 (25)	10 (5)	31 (26)
1000	50	87 (37)	30 (-20)	91 (41)
700	95	130 (36)	46 (-49)	140 (45)
500	200	190 (-10)	66 (-130)	200 (0)
300	400	320 (-80)	110 (-290)	330 (-70)
100	1000	720 (-280)	250 (-750)	760 (-240)

Uncertainty about Response

Uncertain whether response is best modeled as LNT or nonlinear

If nonlinear, which function?

- Location of threshold or no-effect level
- Shape of hormetic response

Calculate expected benefits = sum of

Prob (response function i) * response function i

Example: prob (LNT) = 0.6, prob (hormetic) = 0.4

Uncertainty about Response

$$p(\text{Hormetic}) = 0.4, p(\text{LNT}) = 0.6$$

MCL	Costs	LNT	Hormetic	Expected
4000	1	4 (3)	1 (0)	3 (2)
2000	5	30 (25)	10 (5)	22 (17)
1000	50	87 (37)	30 (-20)	65 (15)
700	95	130 (36)	46 (-49)	97 (2)
500	200	190 (-10)	66 (-130)	140 (-60)
300	400	320 (-80)	110 (-290)	240 (-160)
100	1000	720 (-280)	250 (-750)	540 (-460)

Conclusions

Economic evaluation can accommodate nonlinear exposure-response functions

Complicates assessment of marginal benefit of control

Need to account for joint distribution of background and change in exposure

Uncertainty about exposure-response can be accommodated using expected response

Optimal control may be more or less stringent than under LNT model