

# **Comment Submissions - Proposed Adoption of Exposures to Listed Chemicals in Coffee Posing No Significant Risk**

Published Name:

CERT's Submission No. 3 regarding the Quantitative Risk Assessment for Acrylamide in Coffee of Dr. Steven P. Bayard.

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Post date:

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August 15, 2018

*Via electronic submission to <https://oehha.ca.gov/comments>*

Monet Vela  
Office of Environmental Health Hazard Assessment  
P.O. Box 4010  
Sacramento, California 95812-4010

Re: Proposed Adoption of New Section Under Article 7: No Significant Risk Levels  
Section 25704: Exposures to Listed Chemicals in Coffee Posing No Significant Risk

## CERT'S SUBMISSION NO. 3

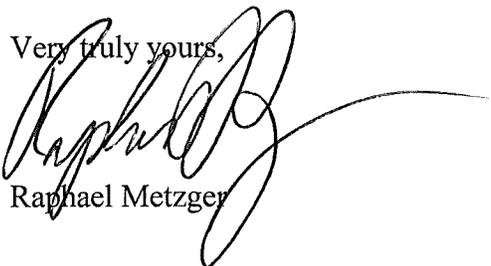
Dear Ms. Vela:

Enclosed herewith are the following documents that are being submitted on behalf of our client, the Council for Education and Research on Toxics (CERT) regarding the Quantitative Risk Assessment for Childhood Leukemia from Maternal Consumption of Coffee During Pregnancy of Dr. Steven P. Bayard:

1. Exhibit A - Statement of Steven P. Bayard: Calculation for the Increased Risk of Two Acute Childhood Leukemias Due to Maternal Coffee Drinking During Pregnancy
2. Exhibit B - Direct Testimony of Dr. Steven Bayard in *CERT v. Starbucks* (October 28, 2014 p.m.)
3. Exhibit C - Resume of Dr. Steven P. Bayard (2017).

Kindly include these materials of Dr. Steven P. Bayard in the record for this rulemaking proceeding.

Very truly yours,

  
Raphael Metzger

RM:ip  
encls: as specified

**EXHIBIT “A”**

## **Statement of Steven Bayard**

### **Calculation for the Increased Risk of Two Acute Childhood Leukemias Due to Maternal Coffee Drinking During Pregnancy**

#### **I. Introduction:**

I have been asked by Raphael Metzger to provide comments on a meta-analysis on maternal coffee consumption and the risk of childhood leukemia (Cheng et al. (2013; Plaintiff's Exhibit #51781), I have also been asked to calculate, if appropriate, a quantitative estimate of the increased risk of childhood leukemia (cases < 15 years of age) from maternal coffee drinking during pregnancy. **I have calculated that increased lifetime childhood acute leukemia risk due to maternal coffee drinking of 1-2 cups per day during pregnancy as 19 per 100,000.**

Below I refer to papers by Cheng et al. and Bonaventure et al. (2013; Plaintiff's Exhibit #48823). My purpose is not to do a hazard identification analysis for maternal coffee drinking as a cause of childhood leukemia, but to calculate the increased cancer risk estimates if others judge there to be sufficient cause to do so.

#### **II. Discussion of Cheng et al. Meta-Analysis**

Cheng et al. rely on seven studies of maternal coffee drinking and childhood leukemias: 4 from France, 1 each from Australia and Greece, and one from combined U.S. and Canada. This North American study analyzed only 84 infant (< 12.5 months) leukemias; the remaining studies all considered the age range up to 14-15 years. Although the North American study had the advantage of more recent recall of coffee drinking (and, therefore, may be the best quality as well as the most pertinent study), it was small, and will not be considered further here as part of the larger more homogeneous response group. For the remaining six studies, the total number of acute leukemia (AL) cases was 2,225 (3,638 controls). Most of these came from the four French studies (1735 cases; 2641 controls). In a

further breakdown (where specified; Cheng, Table 1), of AL to acute lymphoblastic leukemias (ALL) and acute myeloid leukemias (AML or ANLL), nearly 80% (1,285 out of 1,622) of the ALL, and all 203 the AML (ANLL) cases are from France. Thus, the majority of the epidemiologic evidence for maternal coffee drinking - childhood leukemia associations appear to come from French studies.

All 4 French studies appear to use the cases from the National Registry of Childhood Hematopoietic Malignancies (NRCH) and several of the same authors' names appear on three or all four studies. All four publications appear to come from the same (ESCALE) study. Three of the four studies used leukemia data from the same time period, 1995 -1999, and these three were all published from 2004-2007. Without my doing more in-depth checking, I cannot determine if some of the cases were used in all three studies. That would violate the independence requirements of a meta-analysis.

Because of my above concerns, I decided to focus on the 4<sup>th</sup> French study, that by Bonaventure et al. (2013). The Bonaventure study has the most cases (764) and controls (1681) of any of the seven studies, and appears to me to have been well conducted by authors who have published a great deal. However, my purpose here is not to do a critical review of the quality of the entire study, but to provide an estimate of the risk of childhood leukemia from maternal coffee drinking, if that risk is a real one. This is done below.

### **III. Analysis of Maternal Coffee Drinking and Increased Risk of All Childhood (0 – 14) Leukemia based on the Bonaventure et. al. (2013) Study.**

In their case-control study of French children who were diagnosed with acute leukemia in 2003-2004, the authors report an overall odds ratio (OR) of 1.2 ( $0.01 < p < 0.05$ ) for “regular” maternal coffee drinking during pregnancy of at least 1 cup/week, (see Bonaventure’s Table 2, reproduced below). Breaking “regular” drinkers into three increasing categories of “<1 cup/day”, “1 -2 cups/day”, and “>2 cups/day”, yielded increasing ORs of 1.0, 1.3 ( $p < 0.001$ ), and 1.6 ( $p < 0.001$ ), respectively, plus a highly statistically significant positive test for trend ( $p < 0.001$ ).

When AL was broken into AML and ALL, the results were similar, although only the high consumption levels and the trend tests were statistically significant ( $p < 0.01$ ).

While these results, by themselves, are suggestive, they show, in my opinion, even more credibility when they are compared with the authors' same analysis for tea, cola beverages, and alcoholic beverages during pregnancy (See Table 2 below). Doing the same statistical tests as were done for coffee, there were no statistical trends and few statistically significant results with any of these other drinks. To me, this is a good test for checking for "false positives." (A small caveat is that pregnant French women seemed to prefer coffee as a beverage, in general, over these other drinks. This means that there were more coffee than other beverage drinkers in the control group; thus, the coffee group comparisons would have more statistical power.)

#### **IV. Estimates of Increased risk of all childhood (0 – 14) Leukemia based on the Bonaventure et al. (2013) Study.**

In order to estimate the childhood leukemia increased risk in the U.S. based on the ORs in the Bonaventure study, I need to, 1) choose an OR (I use OR as an estimate of relative risk, RR, for the rare disease of childhood leukemia) that represents the coffee drinking habits of pregnant women in California, and 2) estimate the background childhood leukemia rates. This is done below:

To estimate 1) I chose the  $OR = 1.3$  for the 1-2 cups/day coffee drinkers as suitable. This seems reasonable to me, that pregnant women in California will be more health conscious and drink less coffee.

To estimate 2) the background childhood leukemia incidence rates I use the U.S. National Cancer Institute's results from their SEER Cancer Statistics Review 1975-2011, for years 2007-2011:

[http://seer.cancer.gov/archive/csr/1975\\_2010/results\\_merged/sect\\_13\\_leukemia.pdf](http://seer.cancer.gov/archive/csr/1975_2010/results_merged/sect_13_leukemia.pdf)

I use their tables for age-specific cancer incidence rates; Table 13.12 for four age-specific (<1, 1-4, 5-9, and 10-14) rates for ALL, and Table 13.13 for corresponding

age specific rates for AML. I added the two age-specific rates from each of these tables to get the four age group-specific rates for combined ACL plus AML. Then, I multiplied each of these rates by the number of years comprising each age-specific rate and summed these to get the lifetime probability, i.e.:

$$(1 \times 3.5) + (4 \times 8.9) + (5 \times 4.1) + (5 \times 2.7) = 73 \text{ per } 100,000$$

I then checked this result doing a life-table analysis to confirm that 73 per 100,000 is the lifetime probability of childhood (<14) ALL + AML.

Using this estimate of 73/100,000 U.S. lifetime probability for ALL or AML, I then assumed that some of this risk was due to increased leukemias from coffee drinking. From my previous statement on the calculation of the excess risk of cancer to the general population from acrylamide in coffee, I used the National Coffee Association survey to estimate that 65% of Californians drink coffee. With this figure, I estimate that the increased risk of childhood leukemia from California women drinking coffee during pregnancy is **19 per 100,000**. The calculation is:

$$0.35 \times \text{Risk}_{\text{no.coffee}} + 0.65 \times (1.3) \times \text{Risk}_{\text{no.coffee}} = 0.00073 \text{ lifetime risk}$$

Then,  $\text{Cancer Risk}_{\text{no.coffee}} = 0.00065$  and:

**Increased risk of childhood leukemia from maternal coffee drinking pregnancy is:**

$$(\text{Relative Risk} - 1) * \text{Unexposed Risk} = 0.3 \times 0.00065 = 19.5/100,000$$

Table 2 below is a direct copy of Table 2 from Bonaventure et al. (2013):

**Table 2** Associations between childhood leukemia and self-reported maternal consumption of caffeinated and alcoholic beverages during pregnancy

	Controls <i>n</i> = 1,681	All AL <i>n</i> = 764	OR <sup>a</sup>	95 % CI	AML <i>n</i> = 101	OR <sup>b</sup>	95 % CI	ALL <i>n</i> = 648	OR <sup>b</sup>	95 % CI
Coffee during pregnancy										
Never/occasionally	669	273	1.0	Ref.	238	1.0	Ref.	30	1.0	Ref.
Regular ( $\geq 1$ cup/week)	1,008	487	1.2	[1.0–1.5]*	406	1.2	[1.0–1.4]	71	1.6	[1.0–2.6]*
<1 cup/day	503	203	1.0	[0.8–1.3]	174	1.0	[0.8–1.3]	27	1.3	[0.7–2.1]
1 or 2 cups/day	259	130	1.3	[1.0–1.7]*	108	1.3	[1.0–1.7]	19	1.8	[1.0–3.3]
> 2 cups/day	246	154	1.6	[1.2–2.1]***	124	1.5	[1.1–2.0]**	25	2.4	[1.3–4.3]**
Missing	4	4		<i>p</i> for trend < 0.001	4		<i>p</i> for trend = 0.0027			<i>p</i> for trend = 0.0020
Tea during pregnancy										
Never/occasionally	1,009	481	1.0	Ref.	406	1.0	Ref.	64	1.0	Ref.
Regular ( $\geq 1$ cup/week)	666	280	0.9	[0.8–1.1]	240	0.9	[0.8–1.2]	36	0.9	[0.6–1.4]
<1 cup/day	164	85	1.1	[0.8–1.5]	69	1.1	[0.8–1.5]	15	1.5	[0.8–2.7]
1 cup/day	274	106	0.8	[0.7–1.1]	90	0.8	[0.6–1.1]	14	0.9	[0.5–1.6]
>1 cup/day	228	89	0.9	[0.7–1.2]	81	1.0	[0.7–1.3]	7	0.5	[0.2–1.1]
Missing	6	3			2			1		
Cola beverages during pregnancy										
Never/occasionally	1,087	446	1.0	Ref.	379	1.0	Ref.	61	1.0	Ref.
Regular ( $\geq 1$ glass/week)	583	312	1.3	[1.0–1.5]*	263	1.3	[1.0–1.5]*	40	1.1	[0.7–1.7]
1 glass/week	145	68	1.1	[0.8–1.5]	59	1.2	[0.8–1.6]	7	0.8	[0.4–1.8]
>1 glass/week and at most 1/day	269	145	1.3	[1.0–1.6]*	120	1.2	[1.0–1.6]	21	1.3	[0.8–2.2]
>1 glass/day	169	99	1.3	[1.0–1.8]	84	1.3	[1.0–1.8]	12	1.1	[0.5–2.1]
Missing	11	6			6					
Alcoholic beverages during pregnancy <sup>c</sup>										
No alcohol drinking	1,065	468	1.0	Ref.	401	1.0	Ref.	58	1.0	Ref.
Any alcohol drinking	616	296	1.2	[1.0–1.4]	247	1.1	[0.9–1.4]	43	1.4	[0.9–2.2]
<1 glass/week	259	148	1.3	[1.1–1.7]*	123	1.3	[1.0–1.7]	23	1.8	[1.1–3.0]*
1 or 2 glasses/week	201	73	0.9	[0.6–1.2]	60	0.8	[0.6–1.1]	11	1.1	[0.6–2.2]
>2 glasses/week	125	55	1.1	[0.8–1.5]	47	1.1	[0.7–1.5]	7	1.2	[0.5–2.8]
Missing	31	20			17			2		

AL acute leukemia, ALL acute lymphoblastic leukemia, AML acute myeloblastic leukemia

\*\*\*  $p < 10^{-3}$ ; \*\*  $10^{-3} \leq p < 10^{-2}$ ; \*  $10^{-2} \leq p < 0.05$

<sup>a</sup> Odds ratios (OR) and 95 % confidence intervals (CI) estimated by unconditional logistic models including the gender  $\times$  age quota variable, birth order, breastfeeding, maternal education, parental socio-professional category, and European ancestry

<sup>b</sup> OR and 95 % CI estimated by polytomous models of AML and ALL including the gender  $\times$  age quota variable, birth order, breastfeeding, maternal education, parental socio-professional category, and European ancestry

<sup>c</sup> Rudant et al. [23]

# **EXHIBIT “B”**

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SUPERIOR COURT OF THE STATE OF CALIFORNIA

FOR THE COUNTY OF LOS ANGELES

DEPARTMENT 323

HON. ELIHU M. BERLE, JUDGE

COUNCIL FOR EDUCATION AND RESEARCH ON )  
TOXICS, A CALIFORNIA CORPORATION, )

PLAINTIFF, )

VS. )

CASE NO.  
BC435759

STARBUCKS CORPORATION, A CALIFORNIA )  
CORPORATION, ET AL., )

DEFENDANTS. )

\_\_\_\_\_  
AND CONSOLIDATED ACTION. )

REPORTER'S TRANSCRIPT OF TRIAL PROCEEDINGS

MONDAY, OCTOBER 27, 2014

AFTERNOON SESSION

APPEARANCES:

FOR THE PLAINTIFF: METZGER LAW GROUP  
BY: RAPHAEL METZGER, ESQ.  
KENNETH HOLDREN, ESQ.  
401 EAST OCEAN BOULEVARD, SUITE 800  
LONG BEACH, CALIFORNIA 90802

FOR THE DEFENDANT: MORRISON & FOERSTER  
BY: JAMES SCHURZ, ESQ.  
MICHELE B. CORASH, ESQ.  
425 MARKET STREET  
SAN FRANCISCO, CALIFORNIA 94105

CCROLA JOB  
NO. 114689

KAREN VILICICH, CSR. NO. 7634  
OFFICIAL REPORTER PRO TEMPORE

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I N D E X

MONDAY, OCTOBER 27, 2014 (P.M.)

CHRONOLOGICAL AND ALPHABETICAL INDEX OF WITNESSES

<u>PLAINTIFF WITNESS:</u>	<u>PAGE</u>
STEVEN BAYARD	
DIRECT BY MR. METZGER	151
CROSS BY MR. SCHURZ	220

EXHIBIT INDEX

<u>PLAINTIFF</u>		<u>MARKED</u>
<u>EXHIBIT</u>		
130	DOCUMENT RE DEPOSITION TRANSCRIPT	152

1 CASE NUMBER: BC435759  
2 CASE NAME: CERT VS. STARBUCKS  
3 LOS ANGELES, CALIFORNIA MONDAY, OCTOBER 27, 2014  
4 DEPARTMENT 323 HON. ELIHU M. BERLE, JUDGE  
5 REPORTER: KAREN VILICICH, CSR NO. 7634  
6 TIME: P.M. SESSION  
7

8 (THE FOLLOWING PROCEEDINGS WERE HELD  
9 IN OPEN COURT:)

10

11 THE COURT: GOOD AFTERNOON, COUNSEL. BACK ON THE  
12 RECORD IN THE CASE OF CERT VERSUS STARBUCKS. ALL COUNSEL  
13 ARE PRESENT. DR. BAYARD IS PRESENT ON THE STAND.

14 THE CLERK: SIR, YOU HAVE PREVIOUSLY BEEN SWORN AND  
15 YOU ARE STILL UNDER OATH.

16 WOULD YOU RESTATE YOUR NAME FOR THE RECORD.

17 THE WITNESS: STEVEN BAYARD.

18 THE CLERK: THANK YOU.

19 THE COURT: GOOD AFTERNOON, DR. BAYARD.

20 THE WITNESS: GOOD AFTERNOON.

21 THE COURT: MR. METZGER, YOU MAY PROCEED.

22

23 DIRECT EXAMINATION

24 BY MR. METZGER:

25 Q DR. BAYARD, DID YOU GIVE A DEPOSITION IN  
26 THIS CASE?

27 A YES, I DID.

28 Q WAS THAT ON APRIL 28TH OF THIS YEAR?

1           A           YES.

2           Q           AFTER YOUR DEPOSITION, DID YOU MAKE  
3 CORRECTIONS TO YOUR DEPOSITION TRANSCRIPT?

4           A           YES, SIR.

5           Q           I WOULD LIKE TO SHOW YOU WHAT HAS BEEN  
6 MARKED AS EXHIBIT 130. OTHER THAN THE FIRST PAGE, WHICH  
7 IS THE TRANSMITTAL PAGE FOR SENDING YOUR DEPOSITION  
8 CORRECTIONS, WOULD YOU CONFIRM THAT THE DOCUMENTS WITHIN  
9 THAT EXHIBIT, THE PAGES WITHIN THAT EXHIBIT, ARE  
10 CORRECTIONS THAT YOU MADE TO YOUR DEPOSITION?

11          A           THESE ARE MY CORRECTIONS.

12          Q           DID THAT INCLUDE CORRECTIONS TO THE -- TO  
13 SOME WORDS AND NUMBERS IN THE TESTIMONY THAT YOU GAVE?

14          A           PLEASE REPEAT THE QUESTION.

15          Q           DID THE CORRECTIONS INCLUDE CORRECTIONS TO  
16 SOME WORDS IN THE TESTIMONY THAT YOU GAVE?

17          A           YES.

18          Q           DID IT ALSO INCLUDE SOME CORRECTIONS TO DATA  
19 THAT YOU PROVIDED IN THE TABLES IN YOUR REPORT, WHICH IS  
20 EXHIBIT 120?

21          A           THE CORRECTIONS WEREN'T TO THE DATA, THEY  
22 WERE TO THE RESULTS.

23          Q           FINE. LET ME SEE IF I UNDERSTAND. SO ARE  
24 THERE -- THERE ARE TABLES IN EXHIBIT 120, WHICH IS YOUR  
25 REPORT; CORRECT?

26          A           YES.

27          Q           AND THEY SET FORTH CERTAIN RESULTS OF  
28 CALCULATIONS THAT YOU PERFORMED; CORRECT?

1           A           YES.

2           Q           AND DID THE DEPOSITION CORRECTIONS THAT YOU  
3 PROVIDED INCLUDE CORRECTIONS TO SOME OF THOSE RESULTS?

4           A           YES.

5           Q           WERE THOSE COMMUNICATED IN OR ABOUT THE --  
6 ON OR ABOUT JUNE 9, 2014?

7           MR. SCHURZ: I WILL OBJECT.

8           THE COURT: WAIT A SECOND. COMMUNICATED BY WHOM TO  
9 WHOM?

10          Q           BY MR. METZGER: OKAY, DID YOU COMMUNICATE  
11 THOSE CORRECTIONS TO MY OFFICE ON OR ABOUT THAT DATE?

12          A           YES, BUT I THINK IT WAS SLIGHTLY EARLIER.

13          Q           ALL RIGHT. WHAT IS THE DATE OF THE  
14 TRANSMITTAL LETTER, WHICH IS THE FIRST PAGE OF  
15 EXHIBIT 130?

16          A           JUNE 13TH, 2014.

17          Q           ALL RIGHT.

18          MR. SCHURZ: WE WOULD OBJECT TO ANY  
19 CHARACTERIZATION THAT THIS IS CORRECTIONS.

20          THE COURT: JUST A SECOND. WHAT IS THE NEXT  
21 QUESTION?

22          Q           BY MR. METZGER: THE NEXT QUESTION IS: ARE  
23 THE CORRECTIONS TO THE RESULTS THAT ARE CONTAINED WITHIN  
24 EXHIBIT 130 THE SAME THAT YOU ARE PRESENTING HERE TODAY?

25          A           THE CORRECTIONS ARE THE -- THE CORRECTED  
26 RESULTS THAT I TURNED IN ARE WHAT I AM PRESENTING TODAY.  
27 THAT IS CORRECT.

28          THE COURT: THERE IS ONE THING MISSING HERE, WHAT

1 ABOUT COMMUNICATION OF THOSE CORRECTIONS TO DEFENDANTS?

2 Q BY MR. METZGER: WELL, IS EXHIBIT 130 THE  
3 TRANSMITTAL LETTER TO THE DEFENDANTS, DATED JUNE 13,  
4 2014?

5 THE COURT: HOW WOULD HE KNOW?

6 MR. METZGER: WELL, HE --

7 THE COURT: HE IS GOING TO READ -- DID YOU  
8 COMMUNICATE WITH THE DEFENDANTS?

9 THE WITNESS: NO, I DID NOT.

10 MR. METZGER: THAT IS THE DATE --

11 THE COURT: SO YOU ARE GOING TO ASK HIM IF YOU SENT  
12 SOMETHING TO THE DEFENDANTS?

13 MR. METZGER: NO, I CAN REPRESENT TO THE COURT THAT  
14 WE DID.

15 THE COURT: TELL ME. THAT IS WHAT I AM ASKING  
16 ABOUT.

17 MR. METZGER: I WILL REPRESENT TO THE COURT THAT ON  
18 JUNE 13TH, THAT THESE CORRECTIONS WERE TRANSMITTED TO THE  
19 DEFENSE COUNSEL, AS SET FORTH IN THE TRANSMITTAL LETTER,  
20 AND THAT INCLUDED THE CORRECTIONS TO THE RESULTS WHICH  
21 DR. BAYARD IS PRESENTING TODAY.

22 BY THE WAY, THERE IS A FILE & SERVEXPRESS  
23 E-SERVICE STAMP FOR JUNE 13, 2014 AT 3:47 P.M.

24 THE COURT: MR. SCHURZ, DID YOU RECEIVE THE  
25 CORRECTIONS?

26 MR. SCHURZ: WE RECEIVED AN ERRATA THAT INCLUDES,  
27 AS ERRATA DO, A CHANGE TO A WORD HERE, CHANGE TO A WORD  
28 THERE. THOSE ARE REFLECTED IN THE DEPOSITION ERRATA

1 SHEET. THEN WE WERE ALSO PROVIDED A SET OF 10 TABLES  
2 THAT ARE DENOMINATED "REVISED EXHIBIT TABLES." THERE IS  
3 NO CORRECTIONS HERE. THESE ARE NEW TABLES WITH NEW DATA  
4 THAT BEAR THE DATE STAMP THAT IT WAS REVISED ON JUNE THE  
5 9TH. THESE ARE NEW OPINIONS. THEY ARE NEW TABLES. THEY  
6 WERE NOT PART OF HIS ORIGINAL DEPOSITION, AND THEY WERE  
7 PROVIDED TO US MONTHS AFTERWARDS.

8 THE COURT: WE WILL LISTEN TO ARGUMENT AS TO  
9 WHETHER THIS IS NEW INFORMATION OR CORRECTIONS WHEN  
10 COUNSEL FINISHES EXAMINATION SUBJECT TO CROSS-EXAMINATION  
11 AND A MOTION TO STRIKE.

12 COUNSEL MAY PROCEED.

13 MR. METZGER: THANK YOU, YOUR HONOR.

14 Q ALL RIGHT. I BELIEVE WHEN WE LEFT OFF THIS  
15 MORNING, WE WERE GETTING TO YOUR CALCULATIONS FOR THE  
16 INDIVIDUAL DOSE-RESPONSE CANCER SLOPES.

17 DID YOU PREPARE TABLES SETTING THOSE FORTH?

18 A I'M SORRY, COULD YOU START AGAIN.

19 Q SURE. HAVE YOU PREPARED TABLES OF YOUR  
20 CALCULATIONS AND RESULTS FOR THE INDIVIDUAL DOSE-RESPONSE  
21 CANCER SLOPES?

22 A YES, I HAVE.

23 Q DID YOU PRESENT THOSE AT YOUR DEPOSITION  
24 WITHIN EXHIBIT 120?

25 A YES, I HAVE.

26 Q COULD WE HAVE SLIDE 63.

27 IS THIS AN -- THIS SLIDE WHICH IS TITLED,  
28 "MALE RAT THYROID FOLLICULAR CELL ADENOMA OR CARCINOMA,"

1 IS THIS AN EXAMPLE OF THE TABLES THAT YOU PROVIDED FOR  
2 THE 17 TUMOR SITES WHERE YOU WERE -- WHERE THE MODELING  
3 WAS SUCCESSFUL?

4 A YES.

5 MR. SCHURZ: YOUR HONOR, WE WOULD JUST INTERPOSE AN  
6 OBJECTION AND A CLARIFICATION. WE WERE PROVIDED THE  
7 DEMONSTRATIVES YESTERDAY, BUT THEY DON'T LINE UP WITH  
8 WHAT WE ARE SEEING HERE IN TERMS OF -- THEY DON'T LINE UP  
9 IN TERMS OF PAGE NUMBERS.

10 MR. METZGER: I THINK WE REMOVED A FEW BECAUSE WE  
11 THOUGHT THEY WERE REDUNDANT.

12 THE COURT: DO YOU HAVE THIS PAGE?

13 MR. SCHURZ: I HAVE IT AS 65, SO I AM CURIOUS  
14 WHERE --

15 THE COURT: IT IS PAGE 65. LET'S GO FORWARD.

16 MR. METZGER: ALL RIGHT.

17 Q DR. BAYARD, WOULD YOU EXPLAIN TO THE COURT  
18 WHAT -- FIRST, AT THE TOP OF THIS TABLE, THERE IS A --  
19 TWO LINES.

20 WOULD YOU EXPLAIN WHAT THAT IS REFLECTING?

21 A THE TOP TWO LINES OF THE TABLE ARE THE DOSE  
22 GROUPS GIVEN TO -- THIS IS FOR THE MALE RAT THYROID  
23 FOLLICULAR CELL ADENOMA AND CARCINOMA. THE TOP TWO  
24 LINES -- THE TOP LINE IS THE DOSE GROUP, THE FOUR DOSE  
25 GROUPS, AND THE CONTROL. THERE WERE 50 ANIMALS IN THE  
26 DOSE GROUP ORIGINALLY, BUT A FEW OF THEM DIED TOO EARLY  
27 AND THEY COULD NOT DO AUTOPSIES. THEY WERE ELIMINATED  
28 FOR OTHER PURPOSES.

1                   AND WHAT THIS SHOWS IS AN INCREASE IN --  
2 FROM BACKGROUND OF ZERO --

3           Q           BEFORE YOU GET INTO WHAT IT SHOWS, JUST TAKE  
4 US THROUGH THE DATA. THEN I WILL ASK YOU YOUR  
5 INTERPRETATION OF IT.

6           A           THE DATA SHOW FOR THE FIRST -- FOR THE  
7 LOW-DOSE GROUP, I THINK THAT IS THREE OUT OF 48.

8           Q           YES.

9           A           FOR THE NEXT ONE, IT IS FOUR OUT OF 47. FOR  
10 THE HIGH-DOSE GROUP, IT IS SIX OUT OF 48. AND FOR THE  
11 MAXIMUM-TOLERATED DOSE GROUP, IT IS NINE OUT OF 48  
12 RESPONSE. THOSE ARE ANIMALS THAT HAD THYROID FOLLICULAR  
13 CELL ADENOMA OR CARCINOMA.

14          Q           THE MALE RATS WHO HAD THOSE?

15          A           YES.

16          Q           WHAT DO YOU -- WHAT IS THE SIGNIFICANCE OF  
17 THAT INFORMATION TO YOU AS A RISK ASSESSOR?

18          A           WELL, FIRST OF ALL, THERE ARE SOME  
19 SIGNIFICANT, STATISTICALLY SIGNIFICANT RESULTS CERTAINLY  
20 FOR THE MID AND THE HIGH-DOSE GROUP, COMPARING SIX OUT OF  
21 48 VERSUS ONE OUT OF 47, AND NINE OUT OF 48 VERSUS ONE  
22 OUT OF 47. THOSE ARE STATISTICALLY SIGNIFICANT. AND THE  
23 DOSE-RESPONSE TREND IS STATISTICALLY SIGNIFICANT.

24          Q           ALL RIGHT. NOW, PROCEEDING TO THE LARGER  
25 TABLE ON THIS SLIDE AND IN YOUR REPORT, WOULD YOU FIRST  
26 TELL US WHAT THE COLUMNS ARE REPRESENTING?

27          A           THESE COLUMNS REPRESENT THE FOUR MULTISTAGE  
28 MODELS THAT I RAN. THE MULTISTAGE LINEAR, AND THEN

1 LINEAR QUADRATIC, TWO-STAGE, THREE-STAGE, AND FOUR-STAGE.  
2 THE FIFTH ONE IS THE BEST NON-MULTISTAGE MODEL FIT THAT I  
3 FOUND. SO OF THE FIFTH -- THE LAST COLUMN, THERE -- IT  
4 ONLY REPRESENTS THE BEST OF THE REMAINING SEVEN OR EIGHT  
5 MODELS THAT WERE RUN FOR THE DATA.

6 Q ALL RIGHT. AND NOW, IF YOU WOULD TELL US  
7 WHAT THE DIFFERENT ROWS ARE SO WE COULD UNDERSTAND THAT.

8 IT SAYS "B.M.D. 10"?

9 A WELL, THE B.M.D. 10 IS THE BENCHMARK DOSE  
10 10, 10 PERCENT RESPONSE.

11 Q OKAY.

12 A SO THAT REPRESENTS THE ESTIMATED DOSE AT  
13 WHICH THE ANIMALS RESPONDED, 10 PERCENT.

14 Q AND THE NEXT ROW IS "B.M.D.L. 10." WHAT  
15 DOES THAT SIGNIFY?

16 A THAT IS THE LOWER CONFIDENCE LIMIT ON THE  
17 BENCHMARK DOSE, 10.

18 Q OKAY. CAN YOU EXPLAIN TO US WHAT THAT  
19 MEANS?

20 A WELL, OKAY.

21 Q THE LOWER CONFIDENCE LIMIT.

22 A WELL, WE ARE FITTING THE DATA; RIGHT?

23 SO WE ARE FITTING THAT UP FOR THE INDIVIDUAL  
24 RATS HERE, AND IF YOU LOOK AT THE DATA, IT DOESN'T TAKE  
25 MUCH TO FIGURE OUT THAT THESE DATA ARE PRETTY LINEAR,  
26 OKAY?

27 IF YOU LOOK, THREE, FOUR, SIX, NINE, ROUGHLY  
28 DOUBLING OF DOSES, I THINK, EVERY TIME. SO THAT IS

1 PRETTY LINEAR. SO YOU WOULD EXPECT A MULTISTAGE TO FIT,  
2 BUT IT IS NOT EXACTLY LINEAR. THERE ARE SOME DEVIATION.  
3 THE B.M.D.L. 10 ACCOUNTS FOR THE DEVIATION ON THE  
4 CONFIDENCE LIMIT IN THE FITTED MODEL TO THE DATA.

5 Q OKAY. ALL RIGHT. THE THIRD ROW IS  
6 DENOMINATED "CANCER SLOPE FACTOR," AND WHAT IS THAT  
7 REPRESENTING?

8 A WELL, WHEN I WAS EXPLAINING BEFORE ON THE  
9 B.M.D. 10, REMEMBER, I SAID YOU DRAW A STRAIGHT LINE FROM  
10 HERE DOWN TO ZERO. THE WAY TO DRAW A STRAIGHT LINE IS  
11 JUST TO TAKE THE BENCHMARK DOSE, I'M SORRY, .1, THE 10  
12 PERCENT RISK, AND DIVIDE BY THE BENCHMARK DOSE.  
13 REMEMBER, RISK IS NOTHING MORE THAN SLOPE TIMES DOSE.

14 SO YOU TAKE THE SLOPE -- I'M SORRY, YOU TAKE  
15 THE RISK, YOU DIVIDE BY -- WHICH IS 10 PERCENT, YOU  
16 DIVIDE BY THE BENCHMARK DOSE, AND YOU COME UP WITH THE  
17 SLOPE FACTOR. SO THAT REPRESENTS THE STRAIGHT LINE FROM  
18 THE 10 PERCENT RESPONSE DOWN TO ZERO.

19 Q THE NEXT ROW IS LABELED "A.I.C." WHAT IS  
20 THAT?

21 A THAT IS THE AKAIKE INFORMATION CRITERIA,  
22 WHICH WE SAID WE WERE GOING TO BASE CHOOSING THE BEST  
23 MODEL ON THE LOWEST A.I.C. WITHIN ANY KIND OF REASON.  
24 THAT GIVES -- SO THAT IS BASICALLY A FIT OF THE MODEL.  
25 HOW WELL DOES THE MODEL FIT THE DATA.

26 Q THEN THE LAST ROW IS "P-VALUE G.O.F." WHAT  
27 IS THAT?

28 A THAT IS THE GOODNESS OF FIT FOR THE CHI

1 SQUARE TEST. THAT JUST SHOWS ANYTHING GREATER THAN .05  
2 SHOWS THAT THE MODEL FITS THE DATA OKAY.

3 Q SO ABOVE .05 IS ACCEPTABLE?

4 A YES, FOR A MULTISTAGE MODEL, THAT'S CORRECT.

5 Q SO NOW THAT YOU HAVE IDENTIFIED THE COLUMNS  
6 AND THE ROWS, WOULD YOU TELL US -- WELL, LET'S START WITH  
7 THE FIRST ROW, THE B.M.D. 10, FOR THE DIFFERENT -- FOR  
8 THE FIVE MODELS THAT YOU HAVE THERE. TELL US WHAT THE  
9 SIGNIFICANCE IS OF THAT.

10 A OKAY. WELL, I SAID WE FIT FOUR DIFFERENT  
11 MODELS, ALL OF THE CANCER MULTISTAGE FAMILY. IN THIS  
12 INSTANCE, THE DATA FIT THE LINEAR TERMS SO WELL, AS YOU  
13 CAN SEE, THAT THERE WAS NO NEED FOR THE PROGRAM TO -- THE  
14 PROGRAM -- THE ALGORITHMS DID NOT FEEL THAT ADDING A  
15 SECOND, THIRD, OR FOURTH DEGREE TERMS INTO THE EQUATION  
16 WOULD HELP THE FIT. SO IT JUST LEFT OUT THE SECOND,  
17 THIRD, AND FOURTH. SO THE MULTISTAGE -- LINEAR  
18 MULTISTAGE MODEL FIT THE BEST, THE ONE STAGE MULTISTAGE  
19 MODEL.

20 I DON'T MAKE THE CHOICE. THE PROGRAM MAKES  
21 THIS CHOICE. IT DECIDES WHETHER OR NOT THE SECOND,  
22 THIRD, AND FOURTH STAGE PARAMETERS ARE STATISTICALLY  
23 SIGNIFICANT. IF THEY DON'T HELP THE FIT, IT DOESN'T ADD  
24 THEM TO THE EQUATION.

25 Q NOW, THE LAST ONE, THE LOG PROBIT, IS AT  
26 1.17 WHERE ALL THE OTHER VALUES ARE 1.45. WHAT IS THE  
27 SIGNIFICANCE OF THAT?

28 A YOU KNOW, IF YOU GET ENOUGH DATA MODELS,

1 THEN YOU ARE ALWAYS GOING TO GET ONE WITH A LOWER  
2 BENCHMARK DOSE. THE LOWER THE BENCHMARK DOSE, THE HIGHER  
3 THE POTENCY, THE HIGHER THE RISK. SO THE LOG PROBIT,  
4 WHICH ACTUALLY FITS THE DATA BETTER, WOULD HAVE PRODUCED  
5 A HIGHER RISK. I JUST DID NOT USE IT.

6 Q PROCEEDING ON TO THE NEXT ROW, THE B.M.D.L.  
7 10. AGAIN, IT LOOKS LIKE FOR THE FOUR MULTISTAGE MODELS,  
8 IT IS THE SAME VALUE, AND THE LOG PROBIT HAS A DIFFERENT,  
9 WHAT IS THE IMPORT OF THAT?

10 A WELL, THE STRAIGHT LINE IS ACTUALLY THE  
11 SLOPE. THE LOW-DOSE SLOPE THAT WE HAVE CALCULATED  
12 ACTUALLY COMES FROM THE BENCHMARK, FROM THE 10 PERCENT  
13 RESPONSE, DIVIDED BY THE B.M.D.L. 10, THE LOWER  
14 CONFIDENCE LIMITS ON THAT 10 PERCENT RESPONSE.

15 SO IF WE WERE TO TAKE THE LOG PROBIT MODEL,  
16 WE WOULD SEE THAT THAT IS PRETTY LOW, THE .336 IS A  
17 PRETTY LOW DOSE, WHICH MEANS THAT YOU WOULD GET A  
18 10 PERCENT RESPONSE TO .336 MILLIGRAMS PER KILOGRAM PER  
19 DAY.

20 Q ALL RIGHT. AND PROCEEDING TO THE THIRD ROW  
21 OF THE CANCER SLOPE FACTOR, WHAT DID -- WHAT IS THE  
22 SIGNIFICANCE TO YOU OF THAT DATA?

23 A WELL, THE CANCER SLOPE FACTORS ARE WHAT WE  
24 ARE AFTER. WE HAVE TO CHOSE A MODEL BASED ON CERTAIN  
25 CRITERIA TO GET THESE CANCER SLOPE FACTORS. WHAT YOU SEE  
26 HERE IS THE MULTISTAGE ONE, HAS A SLOPE FACTOR OF .113  
27 FOR POTENCY. IF YOU TAKE THE -- IF YOU GO AND TAKE THE  
28 LOG PROBIT, WHICH I DID NOT, YOU WOULD HAVE HAD A SLOPE

1 FACTOR OF ROUGHLY TWICE AS HIGH. THAT IS JUST FOR THAT  
2 ONE TUMOR SITE.

3 MAY I SAY ONE MORE -- GO AHEAD.

4 Q GO AHEAD.

5 A THE REASON I FIT ALL THESE OTHER MODELS IN  
6 THE B.M.D.S. SOFTWARE IS BECAUSE I WANTED TO COMPARE  
7 THEIR AKAIKE INFORMATION CRITERIA AGAINST THE MODELS THAT  
8 I WAS USING.

9 Q OKAY. ALL RIGHT. NOW, LET'S PROCEED TO THE  
10 A.I.C. VALUE. AGAIN, IT APPEARS THAT IT IS THE SAME  
11 VALUE, 146.35 FOR THE FIRST FOUR COLUMNS ON THE  
12 MULTISTAGE, AND THEN THE LOG PROBIT IS 147.91.

13 WHAT IS THE IMPORT OF THAT?

14 A WELL, THEY ARE PRETTY CLOSE. THEY ARE  
15 CLOSE. SO THERE IS NOT MUCH TO CHOOSE BETWEEN IF YOU  
16 CHOOSE ONE MODEL BASED ON -- THEY ARE ALL GOOD FITS.

17 Q OKAY.

18 A THE MULTISTAGE MODEL ACTUALLY FITS THE DATA  
19 BETTER THAN THE LOG PROBIT MODEL DID, WHICH SOMETIMES IT  
20 HAPPENS, SOMETIMES IT DOESN'T. IT JUST MEANS THAT THEY  
21 ARE ALL PRETTY GOOD FITS.

22 Q AS FAR AS THE A.I.C. CRITERION, WHICH IS THE  
23 BEST FIT?

24 A I MEAN, IT LOOKS LIKE THE MULTISTAGE IS A  
25 LITTLE BETTER.

26 Q BECAUSE THE VALUE IS LOWER?

27 A YEAH, THE LOWER THE BETTER.

28 Q AND THE LAST ROW, THE P-VALUE FOR THE

1 G.O.F., WHAT DOES THAT --

2 A THAT IS THE GOODNESS OF FIT. IF THE CHI  
3 SQUARE EQUALS 1.0, THEN THE FIT IS PERFECT. IF IT IS  
4 LESS THAN .05, THEN THE MODEL IS DISCARDED AS TOO POOR A  
5 FIT.

6 Q WHAT IS THE IMPORT TO YOU OF THE DATA THAT  
7 YOU HAVE HERE FOR THE CHI SQUARE FIT?

8 A ALL THESE MODELS FIT THE DATA EXTREMELY  
9 WELL.

10 Q WHAT IS THE SECOND PART OF THIS TABLE AT THE  
11 BOTTOM OF THE SLIDE?

12 A SECOND PART IS A SCALED RESIDUAL. REMEMBER,  
13 WE SAID WE ARE GOING TO CHECK TO SEE -- I AM GOING TO  
14 CHECK TO SEE THE -- WHETHER OR NOT THE SCALED RESIDUAL OF  
15 THE POINTS FROM THE MODEL PREDICTED VALUE IS GREATER  
16 THAN -- ABSOLUTE GREATER, VALUE GREATER THAN 2.0.

17 SO I RAN A TABLE OF THE DEVIATIONS AT EACH  
18 ONE OF THE DOSE POINTS.

19 Q AND THE DOSE POINTS ARE THE DIFFERENT ROWS;  
20 IS THAT CORRECT?

21 A DOSE POINTS ARE THE DIFFERENT ROWS, THAT'S  
22 CORRECT.

23 Q AND WHAT DID YOU CONCLUDE FROM THE RESULTS  
24 HERE?

25 A WELL, AS YOU CAN SEE, NONE OF THE DEVIATIONS  
26 WERE GREAT AT ALL. EVERY ONE WAS FAR LESS THAN 2.0.  
27 OKAY?

28 SO ALL THE MODELS FIT THE DATA. THIS IS NOT

1 THE GREATEST TABLE BECAUSE THERE IS ONLY TWO MODELS  
2 REALLY, BUT THEY ALL FIT.

3 Q ALL RIGHT. SO WHAT DID YOU CONCLUDE FROM  
4 THIS EXERCISE WITH RESPECT TO THE MALE RAT THYROID  
5 FOLLICULAR CELL ADENOMA OR CARCINOMA?

6 A WELL, I DID NOT CONCLUDE ANYTHING UNTIL I  
7 LOOKED AT WHAT THE COMPUTER SAID TO ME. WHAT THE  
8 COMPUTER REPORTED. THE COMPUTER REPORTED THAT ALL THESE  
9 MODELS ARE VIABLE. OKAY?

10 SO IT IS -- SO THEY ALL FIT THE DATA PRETTY  
11 WELL. THEN IT LIKED -- THE COMPUTER ACTUALLY LIKED THE  
12 LOG PROBIT MODEL BETTER BECAUSE IT HAD THE LOWEST  
13 B.M.D.L. I CHOSE THE MULTISTAGE BECAUSE IT HAD THE  
14 LOWEST A.I.C., BUT I WAS GOING TO CHOOSE THE MULTISTAGE  
15 MODEL ANYWAY BECAUSE THAT IS WHAT CANCER MODELS ARE. I  
16 AM MODELING CANCER, SO I DON'T WANT TO CHOOSE A  
17 NON-CANCER MODEL.

18 Q I SEE. THE LOG PROBIT IS A NON-CANCER  
19 MODEL?

20 A IT CAN BE USED FOR IT, BUT IT DOESN'T HAVE  
21 CANCER'S THEORY BUILT INTO THE MODEL. THERE WAS ONE  
22 OTHER THING I WANTED TO ADD IF I COULD.

23 Q PLEASE.

24 A OH, I'M SORRY, I FORGET.

25 Q OKAY. ALL RIGHT. WELL, SO YOU GENERATED  
26 THIS TYPE OF DATA FOR THESE 17 SITES FOR WHICH THE  
27 MODELING WAS ACCEPTABLE?

28 A YES.

1 Q WOULD YOU TAKE A LOOK THROUGH -- WE ARE NOT  
2 GOING TO GO THROUGH THESE 17 SLIDES, BUT WHAT I WOULD  
3 LIKE YOU TO DO IS TO TAKE A LOOK AT THEM AND TELL US --  
4 GIVE US AN EXAMPLE OF ONE WHERE THERE WERE -- I THINK YOU  
5 INDICATED THAT THERE WERE DIFFERENT MODELS THAT SHOWED  
6 SOME DIFFERENCES, SO THAT WE COULD SEE --

7 A I WILL DO MY BEST HERE.

8 Q LET ME JUST DO IT THIS WAY: TAKE A LOOK  
9 THROUGH THE 17 AND TELL ME -- IDENTIFY A TABLE THAT YOU  
10 THINK SHOWS SOMETHING THAT IS SIGNIFICANT TO YOU. WE  
11 WILL JUST TALK ABOUT THAT AND THEN PROCEED ON FROM THESE  
12 TABLES.

13 A OKAY.

14 I HOPE THIS WORKS, I HAVE NOT PRACTICED THIS  
15 ONE, BUT -- HERE IS AN INTERESTING ONE, TABLE 2.

16 Q SLIDE 80, "THE FEMALE MOUSE SKIN  
17 FIBROSARCOMA, SARCOMA AND OTHER SARCOMAS."

18 WHAT STRUCK YOU ABOUT THIS DATA?

19 A WELL, LOOK AT THE SEQUENCE OF RESPONSE.  
20 THIS IS -- THE MOUSE AND DOSE GROUPS WENT FROM ZERO TO 10  
21 MILLIGRAMS PER KILOGRAM PER DAY. THIS IS ACRYLAMIDE IN  
22 DRINKING WATER. OKAY? AND THE MICE COULD DRINK AS MUCH  
23 AS THEY WANTED.

24 AT ZERO DOSE, THERE WAS ZERO RESPONSE. AT  
25 ONE, THERE WAS ZERO RESPONSE. AT TWO, IT WAS THREE. AT  
26 FOUR IT WENT 10. LOOKS LINEAR SO FAR. THEN IT GOES DOWN  
27 TO SIX. WELL, ANY MODELS YOU FIT FROM THIS PROGRAM ARE  
28 GOING TO HAVE TROUBLE FITTING THIS. OKAY?

1                   SO IT CALCULATES B.M.D.'S. AGAIN, HERE, THE  
2 B.M.D.'S ARE LOW, IS LOWER FOR THE DICHOTOMOUS HILL  
3 MODEL. THE ONE OUTSTANDING CHARACTERISTIC OF THE  
4 DICHOTOMOUS HILL MODEL, IT IS A STRANGE NAME, IS THAT IT  
5 ASYMPTOTES BELOW ONE.

6                   SO MOST MODELS WILL SAY IF YOU GIVE INFINITE  
7 DOSE, YOU ARE GOING TO GET INFINITE RESPONSE. THE  
8 DICHOTOMOUS HILL MODEL DOESN'T DO IT. THERE IS MORE  
9 INFORMATION YOU WILL WANT TO KNOW, I AM SURE.

10                  BUT IT CALCULATES THE SLOPES. SO THE CANCER  
11 SLOPES ARE CALCULATED. BUT WHAT IS INTERESTING HERE IS  
12 THAT LOOK AT THE FIT, THE FIT IS JUST GREATER THAN .05.  
13 OKAY, FOR THE MULTISTAGE MODELS, BUT LOOK AT THE FIT FOR  
14 THE DICHOTOMOUS HILL MODEL. IT IS TERRIFIC. OKAY?

15                  SO THE COMPUTER LIKES THE DICHOTOMOUS HILL  
16 MODEL BETTER, BUT I AM SAYING THIS DOESN'T LOOK SO GOOD  
17 TO ME. IT IS GREATER THAN .05, SO I WILL ACCEPT IT, BUT  
18 LOOK AT THE DEVIATIONS HERE. THE DEVIATION AT ONE OF THE  
19 POINTS IS GREATER THAN 2.0, WHICH WAS ANOTHER ONE OF OUR  
20 TESTS.

21                  SO THE COMPUTER KICKS OUT THE INFORMATION  
22 AND SAYS THIS IS A QUESTIONABLE MODEL. OKAY? BECAUSE  
23 THE RESIDUAL IS GREATER THAN 2.0 SOMEWHERE, BUT IT LIKES  
24 THE DICHOTOMOUS HILL MODEL. OKAY?

25                  AND I SAID, LOOK, I AM NOT GOING TO USE THE  
26 DICHOTOMOUS HILL MODEL BECAUSE IT IS NOT A CANCER MODEL  
27 AND --

28                  Q            ALL RIGHT.

1           A           SO WHAT I DID WAS I SAID I AM NOT GOING TO  
2           USE IT. I DON'T LIKE WHERE THE -- I DON'T LIKE WHERE THE  
3           DEVIANT IS GREATER THAN 2.0. I NOTICED THAT THE DEVIANT  
4           WAS GREATER THAN 2.0 RIGHT AT THE 10 PERCENT RESPONSE.  
5           THE REASON WE ARE DOING THIS TO BEGIN WITH IS WE ARE  
6           GOING TO EXTRAPOLATE FROM THE 10 PERCENT RESPONSE DOWN TO  
7           ZERO. SO IF THE DEVIANT IS GREATER OF -- THE FIT OF THE  
8           MODEL TO THE 10 PERCENT RESPONSE LEVEL IS TOO BIG, THEN  
9           IT DOESN'T GIVE US ANY CONFIDENCE IN THE MODEL.

10                    SO I USED IT -- I GO TO THE NEXT TABLE NOW.  
11           I SAID, LOOK, I AM NOT GOING TO USE THIS. I AM GOING TO  
12           TRY ANOTHER APPROACH.

13                    SO ON THE NEXT TABLE, IT SHOULD BE TABLE U.

14           Q           YES.

15           A           I DROPPED THE HIGH DOSE. NOTICE ABOVE HERE?

16                    I THINK THIS IS RIGHT.

17           Q           IT SAYS "DROPPED," I SEE.

18           A           SO I DROPPED THE HIGH DOSE. AND IF YOU WENT  
19           BACK, YOU WOULD NOTICE THAT THE HIGH DOSE WAS THE SIX OUT  
20           OF 10, SIX OUT OF 40 SOMETHING. OKAY?

21           Q           UH-HUH.

22           A           SO I FIT THE MODEL WITH THE DROP DOSE.

23           OKAY?

24           Q           UH-HUH.

25           A           THE TECHNICAL GUIDANCE FROM THE BENCHMARK  
26           DELL SOFTWARE ALLOWS THIS. THE E.P.A. -- ACTUALLY THE  
27           GUIDELINES ALLOW THIS. THE E.P.A. -- THE CANCER EXAMPLE  
28           IN THE TECHNICAL GUIDANCE ALL SUGGEST, "HEY, IF YOUR

1 MODELS DON'T FIT YOUR DATA, TRY SOMETHING, AND THESE ARE  
2 SOME OF THE THINGS CAN YOU DO WITH THEM."

3 SO I DROPPED IT. I DROPPED THE HIGH DOSE  
4 AND VOILA, I GOT MUCH BETTER FIT OF THE DATA. BASED ON  
5 THAT FIT OF THE DATA, I PICKED THE -- I THINK IT WAS THE  
6 ONE-STAGE MODEL. WAIT A MINUTE. I THINK I PICKED THE  
7 ONE-STAGE MODEL. I MAY HAVE PICKED THE TWO-STAGE MODEL.  
8 I HAVE IT IN THE SUMMARY TABLE.

9 Q LET'S GO TO THE SUMMARY TABLE.

10 IS THAT TABLE 1.2?

11 A YES.

12 Q TELL US, FIRST OF ALL, WHAT THE -- GO  
13 AHEAD -- WHAT YOU ARE DOING IN THE SUMMARY TABLE.

14 A I AM SUMMARIZING ALL THE SLOPE -- I AM  
15 SUMMARIZING WHAT THE SLOPE FACTORS ARE FOR ALL THE MODELS  
16 WHICH I JUST RAN FOR ALL THE TUMOR SITES.

17 Q OKAY. TAKE US THROUGH THE COLUMNS IN THIS  
18 TABLE SO WE CAN UNDERSTAND WHAT THIS IS.

19 A WELL, THE FIRST COLUMN IS WHAT WAS THE  
20 EVIDENCE, AND --

21 Q HOLD IT. THE FIRST COLUMN IS "SEX SPECIES."  
22 THESE ARE IDENTIFYING --

23 A SEX SPECIES, IT IDENTIFIES THE SEX PIECES.  
24 THEN THE TUMOR SITES. AND THEN THE N.T.P. EVIDENCE FOR  
25 THOSE TUMOR SITES.

26 AS YOU CAN SEE OF THE 17 TUMORS WHICH I RAN,  
27 I THINK I -- THERE ARE 19 TUMORS LISTED, BUT I WAS  
28 SUCCESSFUL ON 17 OF THEM. THE LAST TWO I WASN'T

1       SUCCESSFUL ON.

2                       CAN YOU SEE THAT?

3                       SO OUT OF THE 17 THAT I WAS SUCCESSFUL IN  
4       GETTING SLOPES FOR, 15 HAD CLEAR EVIDENCE AND TWO OF THEM  
5       HAD RELATED EVIDENCE.

6               Q           OF CARCINOGENICITY?

7               A           THAT'S CORRECT. THAT IS BASED ON THE N.T.P.  
8       CLASSIFICATION. CLEAR EVIDENCE IS A HIGHER DEGREE OF  
9       EVIDENCE.

10              Q           GOOD. THE NEXT COLUMN?

11              A           THE NEXT COLUMN SIGNIFIES THE MODELS THAT  
12       FIT THE DATA. AND IF YOU NOTICE, FOR MOST OF THESE  
13       MODELS, THEY WERE THE MULTISTAGE ONE MODELS. FOR THAT  
14       LAST DATA SET THAT I SHOWED YOU -- WHERE IS THIS THING  
15       GOING?

16                       THE FEMALE MOUSE SKIN FIBROMA SARCOMAS, IF  
17       YOU LOOK IN THAT SECOND SET OF DATA, THE FEMALE MOUSE,  
18       THE LAST ROW, YOU WILL SEE THAT I HAD TO DROP THE  
19       HIGH-DOSE GROUP, AND I USED A MULTISTAGE II MODEL.

20              Q           OKAY.

21              A           SO THAT IS THE -- SO I TALKED ABOUT CLEAR  
22       EVIDENCE IN THE THIRD COLUMN, AND NOW I HAVE TALKED ABOUT  
23       THE MODEL THAT I SELECTED IN THE FOURTH COLUMN.

24              Q           SO FOR MOST OF THE TUMOR SITES, THE  
25       MULTISTAGE I MODEL FIT AND WAS SELECTED?

26              A           YES.

27              Q           THE NEXT COLUMN IS "B.M.D. 10," AND IS THAT  
28       JUST A REPLICATION OF THE DATA FROM THE PRIOR SLIDES?

1 A IT IS A TRANSCRIPTION, YES.

2 Q AND THE SAME FOR THE B.M.D.L. 10?

3 A YES, SIR.

4 Q AND THEN YOU HAVE "S.E.B.M.D." WHAT IS  
5 THAT?

6 A THE STANDARD ERROR OF THE B.M.D.

7 Q THE LASTLY, YOU HAVE THE COLUMN "ANIMAL  
8 CANCER SLOPE FACTORS." WHAT DOES THAT SIGNIFY?

9 A THAT IS THE SAME -- THOSE ARE THE SAME  
10 FIGURES THAT I SHOWED YOU IN THE PREVIOUS SET OF 19 OR 17  
11 TABLES, WHICH WERE THE SLOPE FACTORS. THAT WAS 10  
12 PERCENT DIVIDED BY THE B.M.D.L. 10.

13 Q ALL RIGHT. SO WHAT DID YOU CONCLUDE FROM  
14 THE COMPILATION OF THIS DATA IN TABLE 1.2?

15 A I DID NOT CONCLUDE ANYTHING. I JUST LISTED  
16 ALL THE -- I JUST LISTED ALL THE INDIVIDUAL SLOPES. IT  
17 IS A SUMMARY TABLE.

18 Q WHAT DID YOU DO NEXT?

19 A I AM STILL LOOKING FOR -- WHAT I DID NEXT  
20 WAS CONTINUE ON MY PATH TO TRY TO FIND THE MOST SENSITIVE  
21 STUDY.

22 Q HOW DID YOU DO THAT?

23 A YOU HAVE TO COMBINE THE TUMORS, THE  
24 INDIVIDUAL TUMOR POTENCIES.

25 Q SO ALL OF THESE TUMORS LISTED IN TABLE 1.2,  
26 YOU HAVE TO SOMEHOW COMBINE FOR A COMBINED POTENCY?

27 A ONLY WITHIN INDIVIDUAL SEX SPECIES. SO YOU  
28 DO FOUR DIFFERENT COMBINATIONS. ONE FOR THE MALE RAT,

1 FEMALE RAT, MALE MOUSE, FEMALE MOUSE.

2 Q DID YOU GENERATE TABLES SHOWING THAT?

3 A YES, I DID.

4 Q LET'S GO TO SLIDE 86. ALL RIGHT.

5 IS THIS THE TABLE THAT YOU JUST DESCRIBED  
6 FOR THE MALE FISCHER 344 RATS?

7 A YES.

8 Q THAT IS THE TYPE OF RAT THAT WAS USED BY THE  
9 N.T.P.?

10 A OH, YES.

11 Q ALL RIGHT. TELL US WHAT IS IMPORTANT TO YOU  
12 IN THIS SLIDE, IN THIS TABLE?

13 A WELL, WHAT I DID WAS I ADDED UP -- I  
14 COMBINED THE INDIVIDUAL POTENCIES FROM THE INDIVIDUAL  
15 SLOPES FOR EACH OF THE SIGNIFICANT TUMORS, WHICH I WAS  
16 ABLE TO MODEL, TO GET A TOTAL POTENCY BASED ON ALL FOUR  
17 TUMOR SITES, BECAUSE THERE WERE FOUR SIGNIFICANT TUMOR  
18 SITES IN THE MALE RAT.

19 Q HOW DID YOU DO THAT COMBINING?

20 A I USED THE E.P.A. METHOD OF -- THAT THEY USE  
21 IN THEIR ACRYLAMIDE 2010 DOCUMENT.

22 Q OKAY.

23 A THEY COMBINED TUMOR SITES THE SAME WAY I  
24 DID.

25 Q WHAT WAS THE RESULT OF DOING THAT?

26 A WELL, I ACTUALLY DID IT IN STAGES, SO I  
27 TRIED TO KIND OF ADD RISKS TO SEE WHAT HAPPENED. HOW DID  
28 THAT POTENCY, THE COMBINED TUMOR POTENCY VALUE INCREASE

1 AS I ADDED EACH TUMOR SITE, EACH POTENCY. SO I DID IT IN  
2 STAGES.

3 Q SHOW US, PLEASE.

4 A WELL, FOR THE FIRST STAGE -- SINCE I CAN'T  
5 READ THAT, I AM GOING TO TRY TO --

6 Q I CAN READ IT FROM WHERE I AM. THE FIRST  
7 ONE I THINK SAID, "THYROID GLAND FOLLICULAR ADENOMA  
8 CARCINOMA." THAT IS THE FIRST ROW AND THEN "HEART  
9 SCHWANNOMA" IS THE SECOND.

10 A I DO ACTUALLY HAVE THAT IN MY EXHIBIT. I  
11 CAN READ THAT.

12 Q OKAY.

13 A SO WHEN I ONLY USED TWO, TWO TUMOR SITES,  
14 THEN I CAME UP WITH AN UPPER BOUND RISK OF 0.16.

15 Q IT SAYS "0.16635"?

16 A THAT IS CORRECT. I AM TRYING TO GET MY  
17 POINTER. I LOVE THESE POINTERS. THAT IS WHEN I ONLY HAD  
18 TWO. I PUT IN THE INTERMEDIATE VALUES AND THE VALUES  
19 THAT I USED SO THAT ANYONE COULD CHECK MY CALCULATIONS

20 Q WHAT DOES THAT ACTUALLY MEAN, THAT THE UPPER  
21 BOUND CUMULATIVE RISK IS THAT NUMBER?

22 A IT IS DEFINED AS THE CANCER SLOPE FACTOR.

23 Q OKAY. GOT IT. THEN WHAT DID YOU DO NEXT  
24 WITH -- BY ADDING ANOTHER TUMOR SITE, THE EPIDIDYMIS  
25 TESTIS MESOTHELIOMA?

26 A THAT INCREASED THE RISK. ADDING THAT  
27 INCREASED THE RISK FROM .166 TO .224.

28 Q SO YOU WENT THROUGH THIS EXERCISE ADDING TO

1 IN EACH ANALYSIS ANOTHER TUMOR SITE TO DETERMINE THE  
2 COMBINED CANCER RISK?

3 A YES, IT WAS JUST A WAY FOR ME TO SHOW PEOPLE  
4 HOW I DID THE CALCULATIONS.

5 Q OKAY. WHAT WAS YOUR CONCLUSION ABOUT THIS?

6 A MY CONCLUSION WAS THAT WHEN YOU PUT IN ALL  
7 THE INDIVIDUAL POTENCIES, THAT THE FINAL RISK ESTIMATE  
8 FOR THE FINAL CANCER SLOPE FOR THE MALE RAT IS .262.

9 Q OKAY. ALL RIGHT. YOU DID THIS EXERCISE FOR  
10 THE FOUR DIFFERENT SPECIES AND SEXES?

11 A MALE RAT, FEMALE RAT, MALE MOUSE, FEMALE  
12 MOUSE.

13 Q WHAT WERE YOUR SLOPE ESTIMATES THEN FOR THE  
14 FOUR OF THEM?

15 A WE PROBABLY COULD GO RIGHT TO THAT SLIDE.  
16 THAT SUMMARIZES WHAT THE RESULTS WERE.

17 Q ALL RIGHT. THE MALE RAT THAT WE JUST WENT  
18 THROUGH, WE SAID WAS .2617 MILLIGRAMS PER -- PER  
19 MILLIGRAM PER KILOGRAM PER DAY. FOR THE OTHERS, IT  
20 WAS -- FOR THE FEMALE RAT IT WAS .31. FOR THE MALE MICE,  
21 IT WAS .38. FOR THE FEMALE MICE, IT WAS .37.

22 SO WHAT I CONCLUDED WAS THAT THE MICE ARE  
23 MORE SENSITIVE THAN THE RATS BECAUSE THEY GIVE HIGHER  
24 RISK ESTIMATES.

25 Q SO WHAT WAS THE SIGNIFICANCE OF THE MICE  
26 BEING MORE SENSITIVE TO YOU?

27 A IT MEANT THAT THE MICE WAS THE MOST  
28 SENSITIVE STUDY.

1 Q AND YOU WERE LOOKING FOR THAT?

2 A THAT IS WHY I DID THIS WHOLE THING.

3 Q WHAT WAS THE NEXT STEP IN YOUR QUANTITATIVE  
4 RISK ASSESSMENT?

5 A WELL, BEFORE WE LEAVE IT, I WOULD LIKE TO  
6 SHOW YOU, JUST MAKE THE POINT, THAT BOTH THE MALE AND  
7 FEMALE MICE SEEMED EQUALLY SENSITIVE. SO THERE WAS NO  
8 REAL REASON TO CHOOSE BETWEEN THE TWO. SO I WOULD TAKE  
9 THE AVERAGE OF THE TWO.

10 Q OKAY.

11 A AND IF YOU LOOK, YOU WILL SEE THAT --  
12 COMPARE THE FEMALE MICE TO THE FEMALE RATS, AND YOU SEE  
13 THAT THAT IS ABOUT 20 PERCENT HIGHER. THE MICE HAS 20  
14 PERCENT HIGHER RISK. IF YOU COMPARE THE MALE MICE TO THE  
15 MALE RAT, THE MICE ARE ABOUT 45 PERCENT HIGHER RISK.

16 Q ALL RIGHT. SO WHAT DID YOU DO NEXT IN YOUR  
17 ANALYSIS?

18 A I AM TRYING TO REMEMBER.

19 SO I NOW HAVE THE MOST SENSITIVE SEX  
20 SPECIES. OH, I COMPARED THE SENSITIVITY WITHOUT THE  
21 HARDERIAN GLAND TUMORS.

22 THESE MICE, ACRYLAMIDE IN MICE SEEM TO  
23 AFFECT THE HARDERIAN GLAND ALMOST EXQUISITELY. SO SINCE  
24 THE -- SINCE THE RESULTS WERE SO DEPENDENT ON USING  
25 HARDERIAN GLANDS TUMORS, I TRIED TO MAKE THE COMPARISON  
26 WITHOUT THE HARDERIAN GLAND TUMORS.

27 Q WHAT DID YOU FIND DOING THAT?

28 A WELL, I THINK IT IS ON THE NEXT SLIDE.

1                   OKAY, THERE IT IS. THAT IS COMPARING THEM  
2 WITHOUT THE HARDERIAN GLAND TUMORS IN THE MICE. THE MALE  
3 AND FEMALE RATS ARE THE SAME, BUT NOW INSTEAD OF THE MALE  
4 MICE AND FEMALE MICE HAVING SLOPES OF .37 AND .38, THEY  
5 GO DOWN TO .09 AND .18.

6           Q           OKAY.

7           A           SO THE -- THE RISK ESTIMATES ARE HIGHLY  
8 AFFECTED BY THE USE OF HARDERIAN GLANDS TUMORS, AND THE  
9 SELECTION OF THE MOST SENSITIVE STUDY.

10          Q           OKAY. WITH THIS INFORMATION, DID YOU  
11 SOMEHOW RELATE THIS TO HUMANS?

12          A           WELL, I MEAN, I HAD A QUESTION ON IT MYSELF.  
13 THAT IS DO -- HUMANS DON'T HAVE HARDERIAN GLANDS. THEY  
14 ARE KIND OF LIKE NICTITATING MEMBRANES IN THE EYE.  
15 ALMOST LIKE A SECOND EYELID, I THINK, IF I AM NOT  
16 MISTAKEN.

17                    SO I HAD TO QUESTION -- DO I USE HARDERIAN  
18 GLAND TUMORS, AND I DEPENDED ON PRETTY MUCH THE N.T.P. I  
19 DEPENDED ON -- GOD, HEALTH CANADA AND EUROPEAN FOOD  
20 SAFETY AGENCIES THAT DID USE HARDERIAN GLAND TUMORS IN  
21 THEIR RISK ASSESSMENTS IN THE MOUSE FOR ACRYLAMIDE.

22          Q           DID YOU DO ANALYSES FOR TUMORS, TUMOR  
23 POTENCY AND CARCINOGENICITY, ONE USING THE HARDERIAN  
24 GLANDS AND ONE WITHOUT?

25          A           I DECIDED -- YES, I DECIDED THIS WOULD MAKE  
26 A GOOD SENSITIVITY ANALYSIS.

27          Q           DID YOU SOMEHOW DO SOME KIND OF EQUIVALENCE  
28 TO GET FROM THE RODENTS TO HUMANS?

1 A YES, I DID.

2 Q WHAT IS THAT CALLED?

3 A ANIMAL TO HUMAN SCALING FACTOR.

4 Q OKAY. IS THERE A FORMULA FOR THAT?  
5 HOW DOES THAT WORK?

6 A YES. OKAY, SO NOW IF YOU HAVE GOT THE MOST  
7 SENSITIVE STUDY, THAT IS THE MOST SENSITIVE ANIMAL STUDY,  
8 YOU HAVE TO GET THIS IN TERMS OF HUMAN EQUIVALENT  
9 POTENCY. THE WAY YOU DO THAT IS BY MULTIPLYING IT BY AN  
10 ANIMAL TO HUMAN SCALING FACTOR.

11 Q WHAT IS AN ANIMAL TO HUMAN SCALING FACTOR ?  
12 WHAT IS THE PURPOSE OF THAT?

13 A IT IS TO DETERMINE THE EQUIVALENT DOSE IN  
14 HUMANS WHICH WILL GIVE THE SAME RESPONSE THAT YOU SEE IN  
15 THE ANIMALS.

16 Q IS THAT A FUNCTION OF BODY WEIGHT?

17 A IT IS OFTEN A FUNCTION OF BODY WEIGHT. IT  
18 KIND OF GET ITS ORIGIN FROM THE FACT THAT IF YOU GIVE A  
19 HORSE THE SAME PILL ON THE SAME MILLIGRAMS PER KILOGRAM  
20 BASIS THAT YOU GIVE TO A RAT ON THE SAME MILLIGRAMS PER  
21 KILOGRAM WEIGHT OF THE HORSE, YOU WILL KILL THE HORSE.  
22 SO YOU BASICALLY -- JUST BECAUSE THE HORSE IS TWICE THE  
23 SIZE DOESN'T MEAN YOU GIVE THEM TWICE THE PILL, TWO PILLS  
24 INSTEAD OF ONE. LESS OF THE DOSE TO THE HORSE IS  
25 REQUIRED TO CREATE THE SAME EFFECT.

26 Q SO THIS SCALING FACTOR, ARE THERE ACCEPTED  
27 VALUES FOR THAT?

28 A THERE ARE MULTIPLE WAYS TO DO IT. THERE ARE

1 JUST MULTIPLE WAYS TO DO IT. I CHOSE THE DEFAULT.

2 Q WHEN YOU SAY YOU CHOSE THE DEFAULT, WHAT  
3 DEFAULT DID YOU CHOOSE?

4 WHERE DID THAT COME FROM?

5 A BOTH THE U.S. E.P.A. AND CALIFORNIA E.P.A.  
6 GIVE DEFAULT VALUES. THEY SAY WE HAVE BASIC SCALING  
7 FACTORS FROM ANIMALS TO HUMANS TO USE. THOSE SCALING  
8 FACTORS ARE -- THE FORMULA FOR THEM IS THE WEIGHT OF THE  
9 HUMAN DIVIDED BY THE WEIGHT OF THE ANIMAL ALL RAISED TO  
10 THE ONE-QUARTER POWER. IT IS BASICALLY A SIGNAL THAT  
11 SAYS THAT THE HUMAN WILL REQUIRE LESS DOSE THAN THE RAT  
12 WILL FOR THE SAME EFFECT.

13 Q OKAY. SO WHERE DO WE LOOK TO SEE YOUR  
14 ANALYSIS ON THAT?

15 A I THINK I HAVE A SLOPE -- I THINK I HAVE A  
16 -- I HAD A SLIDE ON IT.

17 Q IS THAT TABLE 1.D OR IS THAT THE --

18 A WELL, I HAVE THE SLIDES AS PAGES 96 AND 97.

19 Q WELL, LET'S TAKE A LOOK AT THOSE THEN IF WE  
20 COULD. 96 IS WHAT YOU JUST TOLD US. GO TO SLIDE 97.

21 TELL US WHAT THIS IS REPRESENTING.

22 A WELL, I TOOK -- THE MALE/FEMALE RATS AND  
23 MALE/FEMALE MICE ALL HAVE DIFFERENT SCALING FACTORS  
24 BECAUSE THEIR WEIGHTS ARE DIFFERENT. SO ALL AGENCIES  
25 THAT I KNOW OF -- WELL, CAL E.P.A. AND U.S. E.P.A.  
26 SPECIFY A 70-KILOGRAM HUMAN. AND THE RAT, THE MALE RAT  
27 WEIGHED 400 GRAMS OR .4 KILOGRAMS. SO YOU TAKE THE  
28 SCALING FACTOR FOR THE RAT BECOMES 2.27, FOR THE FEMALE

1 RAT, 2.41. FOR THE MALE RAT, 6.89 -- FOR THE MALE MOUSE,  
2 6, 89 AND FOR THE FEMALE MOUSE, 7.27.

3 Q ALL RIGHT. WHAT DID YOU DO NEXT?

4 A WELL, I CONVERTED THE ANIMAL POTENCY SLOPES  
5 TO THE HUMAN CANCER POTENCY SLOPES BY MEANS OF  
6 MULTIPLYING BY THE SCALING FACTOR.

7 Q IS THAT SET FORTH IN TABLE 1.D?

8 A 1.D.1.

9 Q COULD YOU SHOW THAT. ALL RIGHT.

10 SO IN THIS TABLE, YOU HAVE IN THE FIRST  
11 COLUMN THE SEX AND SPECIES, THE SECOND COLUMN THE TUMOR  
12 SITE, OR SITES, AND THEN YOU HAVE THE B.M.D.L. 10, THE  
13 ANIMAL CANCER SLOPE FACTOR, ALL OF THIS IS INFORMATION  
14 THAT YOU PREVIOUSLY --

15 A IT IS REPEATED.

16 Q THEN YOU HAVE THE SCALING FACTOR, WHICH YOU  
17 JUST DERIVED?

18 A YES.

19 Q AND THE LAST COLUMN IS THE HUMAN CANCER  
20 SLOPE FACTOR?

21 A THAT IS CORRECT.

22 Q WHAT DOES THAT ACTUALLY REPRESENT?

23 A THE HUMAN CANCER SLOPE FACTOR?

24 Q YES.

25 A THAT REPRESENTS THE ESTIMATE OF THE HUMAN  
26 RISK, HUMAN POTENCY, THE ACTUAL FIGURE THAT WE ARE  
27 LOOKING FOR IN THE DOSE-RESPONSE ASSESSMENT. I MEAN, ALL  
28 THIS IS WHAT WE HAVE DONE JUST TO ESTIMATE THE HUMAN

1 CANCER SLOPE FACTOR IN THE DOSE-RESPONSE ASSESSMENT.  
2 THAT ONE AREA OF RISK ASSESSMENT.

3 SO THE PERTINENT FIGURES ARE -- FOR THE  
4 HUMAN CANCER SLOPE FACTORS ON THE RIGHT-HAND COLUMN --  
5 ARE COMBINED SITES FOR THE MALE RAT, IT IS 2.62, FOR THE  
6 FEMALE RAT, IT IS 2.69. FOR THE MALE MOUSE, IT IS .0602.

7 AM I RIGHT ON THAT?

8 Q YEAH, IT SAYS "0.602."

9 A I THINK THAT IS WHAT -- I DON'T USE THE  
10 THYROID -- I DON'T USE THE HARDERIAN GLANDS?

11 Q THIS -- DR. BAYARD?

12 A YES, SIR.

13 Q LET ME ASK YOU, FOR THE FIRST SET OF DATA  
14 REGARDING THE MALE MOUSE, WHEN YOU HAVE THE COMBINED SITE  
15 HUMAN CANCER SLOPE FACTOR OF 2.628, HOW IS THAT DERIVED  
16 FROM THE DATA THAT IS ABOVE IT?

17 A I'M SORRY, I COULD NOT SEE YOUR SLIDES. MAY  
18 I GO BACK TO -- BECAUSE I COULD NOT SEE THE SLIDES.

19 THE TOP TWO ARE ACTUALLY FOR THE MALE MOUSE  
20 AND FEMALE MOUSE.

21 Q RIGHT.

22 A SO THOSE FIGURES ARE CORRECT.

23 Q WELL, OKAY. MY QUESTION IS: YOU TOLD US  
24 THAT THE COMBINED SITE, HUMAN CANCER SLOPE FACTOR FOR THE  
25 MALE MOUSE WAS 2.628, WHICH APPEARS THERE. HOW IS THAT  
26 2.628 CALCULATED?

27 A IT IS CALCULATED BY MULTIPLYING THE ANIMAL  
28 CANCER SLOPE, WHICH IS COLUMN 4, TIMES THE SCALING

1 FACTOR, WHICH IS COLUMN 5.

2 Q SO NOW THAT YOU HAVE THESE HUMAN CANCER  
3 SLOPE FACTORS, WHAT DO YOU DO WITH THAT?

4 A WELL, I PUT THEM ASIDE FOR A MOMENT UNTIL I  
5 CAN ESTIMATE WHAT THE EXPOSURE IS BECAUSE NOW I HAVE  
6 ESTABLISHED WHAT THE DOSE-RESPONSE POTENCY IS. SO NOW IT  
7 IS A MATTER OF RISK EQUALS POTENCY TIMES DOSE. I HAVE TO  
8 FIGURE OUT WHAT THE DOSE OF ACRYLAMIDE IN COFFEE IS.

9 Q BEFORE WE LEAVE THIS, YOU HAVE THESE  
10 COMBINED SITE HUMAN CANCER SLOPE FACTORS, AND WHY DO YOU  
11 HAVE THE COMBINED SITES?

12 A BECAUSE I WANT TO ESTABLISH TOTAL -- THE  
13 MOST SENSITIVE STUDY, AND THE MOST SENSITIVE STUDY -- IN  
14 TRYING TO ESTABLISH THE MOST SENSITIVE ANIMAL STUDY, YOU  
15 HAVE TO ACCOUNT THE MULTIPLE TUMORS. MULTIPLE TUMOR  
16 SITES.

17 Q OKAY. THANK YOU. ALL RIGHT.

18 NOW, DR. BAYARD, HOW DOES THIS COMPARE, THIS  
19 ANALYSIS THAT YOU HAVE DONE, COMPARE TO THAT DONE BY  
20 OTHER GOVERNMENTAL AGENCIES?

21 A WELL, I WANTED TO KNOW THAT MYSELF ACTUALLY.  
22 SO I THINK IT IS PROBABLY IN -- IT IS IN ONE OF THE NEXT  
23 TABLES.

24 Q GO ON TO THE NEXT ONE.

25 A ALL THIS DOES IS SHOW THE DIFFERENCE BETWEEN  
26 THE SCALING FACTORS USED BY CALIFORNIA E.P.A. AND THE  
27 SCALING FACTORS THAT I USED. CAL E.P.A. USED SLIGHTLY  
28 LARGER SCALING FACTORS IN 2005. THEY USED SLIGHTLY

1 DIFFERENT STUDIES. THEY USED A DIFFERENT DRINKING WATER  
2 STUDY FOR ACRYLAMIDE ALSO. THEY USED THE EARLIER ONE.

3 Q ALL RIGHT. SO WHAT DID YOU DO WITH THE  
4 HUMAN CANCER SLOPES THAT YOU CALCULATED FOR THE N.T.P.  
5 DATA?

6 A WELL, I COMPARED THEM WITH CAL E.P.A., AND  
7 IF YOU WILL FIND TABLE 1.D.2.

8 Q THAT IS SLIDE 102, I THINK.

9 A IT IS A LITTLE DIFFICULT TO SEE, BUT I THINK  
10 THE IMPORTANT PART OF THIS TABLE IS THAT IT ALLOWED ME TO  
11 COMPARE THE HUMAN SLOPES ESTIMATED BY CAL E.P.A. TO --  
12 WITH THEIR DRINKING WATER STUDIES, TO THE HUMAN SLOPES  
13 THAT I ESTIMATED BASED ON MY RAT STUDIES, NOT MY RAT  
14 STUDIES, THE N.T.P. RAT STUDIES, AND THE HUMAN SLOPES I  
15 ESTIMATED FROM THE N.T.P. MOUSE STUDIES.

16 Q OKAY. AND YOU HAVE A FEW NUMBERS ON THIS  
17 TABLE HIGHLIGHTED IN YELLOW. I ASSUME THAT THOSE HAVE  
18 SOME SIGNIFICANCE. WOULD YOU EXPLAIN THAT?

19 A WELL, I HIGHLIGHTED THEM BECAUSE I WANTED TO  
20 REMIND MYSELF THAT THESE WERE THE ESTIMATES THAT I WAS  
21 USING WITH HARDERIAN GLAND TUMORS. I THINK THE -- AND TO  
22 SHOW THE COMPARATIVELY LARGE EFFECT CAUSED BY THE  
23 HARDERIAN GLAND TUMORS TO THE TOTAL RISK ESTIMATES. BUT  
24 I THINK THE MORE PERTINENT DATA THAT ARE INTERESTING TO  
25 ME ANYWAY ARE THE COMPARISONS OF THE CALIFORNIA E.P.A.  
26 ESTIMATES FOR HUMAN CANCER POTENCY WITH MINE FOR RATS.  
27 OKAY, AND CALIFORNIA -- AND YOU COULD FIND THAT BY  
28 LOOKING ON THE BOTTOM LINE, BOTTOM ROW.

1 Q THE BOTTOM ROW IT SAYS -- THE ROW IS TITLED,  
2 "GEOMETRIC MEAN SLOPE FACTOR," AND YOU HAVE 0.70 FOR CAL  
3 E.P.A. 2005?

4 A YES.

5 Q AND THEN YOU HAVE 0.67 FOR THE N.T.P. 2012  
6 RATS?

7 A THAT IS THE ONE I DID.

8 Q OKAY. THEN YOU HAVE 2.66 FOR THE N.T.P.  
9 2012 MOUSE?

10 A YES.

11 Q SO WHAT DO YOU MAKE OF THOSE DATA?

12 A WELL, THE FIRST THING I MADE WAS THAT  
13 CALIFORNIA E.P.A. AND MY ESTIMATES, BASED ON THE N.T.P.  
14 RATS, WERE VERY, VERY SIMILAR.

15 Q OKAY. WHAT ELSE DID YOU CONCLUDE?

16 A THAT THE N.T.P. MOUSE WAS MORE SENSITIVE  
17 THAN -- THE RISKS BASED ON THE N.T.P. MOUSE WERE MORE  
18 SENSITIVE THAN THE RATS.

19 Q DID YOU PERFORM THE SAME ANALYSIS FOR THE  
20 N.T.P. DATA WITHOUT THE HARDERIAN GLANDS?

21 A YES.

22 Q IS THAT THE NEXT TABLE?

23 A YES.

24 Q TABLE 1.D.2.

25 WHAT DID YOU CONCLUDE FROM THIS ANALYSIS?

26 A IF I LEAVE THE HARDERIAN GLAND TUMORS OUT OF  
27 THE MOUSE ANALYSIS, THEN THE ESTIMATES ARE VERY, VERY  
28 CLOSE.

1 Q OKAY. THAT BEING FOR YOUR -- FOR THE N.T.P.  
2 2012 RATS, THE ESTIMATE IS 0.67, AND FOR THE N.T.P. 2012  
3 MOUSE STUDY WITHOUT THE HARDERIAN TUMORS IS 0.88?

4 A THAT'S CORRECT.

5 Q ALL RIGHT. SO WHAT SEX AND SPECIES DID YOU  
6 CONCLUDE YOU SHOULD USE AS THE MOST SENSITIVE STUDY FOR  
7 YOUR RISK ASSESSMENT?

8 A I CONCLUDED THAT THE N.T.P. MOUSE COMBINED  
9 MALE AND FEMALES SHOULD BE USED.

10 Q WHAT WAS THE BASIS FOR THAT CONCLUSION?

11 A THAT THE N.T.P. MOUSE WAS THE MOST SENSITIVE  
12 STUDY.

13 Q AND IS THERE GUIDANCE THAT SUPPORTED THE USE  
14 OF THAT, OF THE HARDERIAN GLAND TUMOR FOR THIS AS THE  
15 MOST SENSITIVE SITE EVEN THOUGH HUMANS DO NOT HAVE AN  
16 EXACT HARDERIAN GLAND?

17 A THE GUIDANCE -- I THINK THE MOST PERTINENT  
18 GUIDANCE I USED WAS THE E.P.A. CANCER ASSESSMENT  
19 GUIDELINES OF 2005, WHICH SAID SITE CONCORDANCE IS NOT A  
20 PREREQUISITE FOR DOING ANIMAL TO HUMAN COMPARISONS.

21 Q ALL RIGHT. SO ARE WE NOW READY TO PROCEED  
22 TO THE EXPOSURE ASSESSMENT?

23 A I HOPE SO.

24 Q OKAY. YOU HAVE CONCLUDED THE CANCER POTENCY  
25 DOSE-RESPONSE ANALYSIS THAT YOU DID?

26 A YES.

27 Q VERY GOOD. SO HOW DID YOU GO ABOUT YOUR  
28 EXPOSURE ASSESSMENT ANALYSIS?

1           A           WELL, IF YOU CAN MOVE AHEAD, THERE IS JUST  
2 ONE -- IT IS A SLIDE THAT IS PROBABLY BETTER AS A VISUAL.  
3 I HAVE IT AS SLIDE 107.

4           Q           GO AHEAD. WE ARE NOW ON "EXPOSURE  
5 ASSESSMENT."

6           A           THAT'S CORRECT. ONE MORE, PLEASE.  
7 MR. SCHURZ, DO YOU HAVE THIS?

8           Q           GO AHEAD, DR. BAYARD.

9           A           WELL, THE AMOUNT OF ACRYLAMIDE -- WHAT WE  
10 HAVE TO KNOW IS THE AMOUNT OF ACRYLAMIDE CONSUMED PER  
11 DAY. THAT IS WHAT WE ARE LOOKING FOR BECAUSE OUR SLOPE  
12 IS IN TERMS OF DOSE PER DAY. OKAY?

13                       RISK PER DOSE, OKAY, PER DAY.

14                       SO IN ORDER TO FIND OUT HOW MUCH ACRYLAMIDE  
15 IS IN COFFEE, WE HAVE TO GET THE CONCENTRATION OF  
16 ACRYLAMIDE IN COFFEE. YOU KNOW, WHAT IS THE  
17 CONCENTRATION PER OUNCE OF COFFEE, AND THEN WE HAVE TO  
18 GET THE AMOUNT OF COFFEE CONSUMED DAILY.

19           Q           LET ME JUST INTERRUPT YOU A SECOND AND ASK  
20 YOU ABOUT THIS CONCEPT OF THE CONCENTRATION OF ACRYLAMIDE  
21 IN COFFEE. FIRST OF ALL, ARE YOU TALKING THE AMOUNT OF  
22 THE CONCENTRATION OF ACRYLAMIDE IN BREWED COFFEE OR IN  
23 COFFEE BEANS?

24           A           OH, NO, BREWED COFFEE.

25           Q           ALL RIGHT. AND IS THE ACRYLAMIDE  
26 CONCENTRATION IN BREWED COFFEE LESS THAN THE AMOUNT OF  
27 CONCENTRATION OF ACRYLAMIDE IN COFFEE BEANS?

28           A           IT PROBABLY IS. I ASSUME THAT IT IS

1 PRETTY -- COOKED COFFEE BEANS, ANYWAY, THAT IT IS  
2 PROBABLY PRETTY DENSE IN A SMALL BEAN.

3 Q I MEANT ROASTED COFFEE BEANS.  
4 WHEN ACRYLAMIDE FROM THE ROASTED COFFEE  
5 BEANS ENDS UP IN BREWED COFFEE THROUGH THE BREWING  
6 PROCESS, IS THERE DILUTION BECAUSE OF THE WATER THAT IS  
7 USED TO BREW THE COFFEE?

8 A YES, THE CONCENTRATION, OF COURSE, IN WATER  
9 WOULD BE LESS BECAUSE THERE IS SO MUCH MORE WATER.

10 Q SO CONCENTRATION IS RELATED IN PART TO  
11 DILUTION?

12 A YES.

13 Q OKAY. AND IS THAT ACCOUNTED FOR IN THE  
14 QUANTITATIVE -- IN YOUR EXPOSURE ASSESSMENT?

15 A OH, YES.

16 Q ALL RIGHT. SO LET'S PROCEED. HOW DID YOU  
17 DETERMINE THE CONCENTRATION OF ACRYLAMIDE IN BREWED  
18 COFFEE?

19 A I USED THE F.D.A. 2002 AND 2003 INDIVIDUAL  
20 MARKET BASKET STUDY, OR SOMETHING LIKE THAT, WHICH LOOKS  
21 AT 20 SAMPLES OF BREWED -- WHICH ANALYZED THE  
22 CONCENTRATIONS OF ACRYLAMIDE IN 20 SAMPLES OF BREWED  
23 COFFEE.

24 Q OKAY. I THINK THAT DOCUMENT HAS ACTUALLY  
25 BEEN JUDICIALLY NOTICED.

26 DID YOU TAKE THE DATA FROM THAT DOCUMENT AND  
27 COMPILE IT INTO A TABLE?

28 A YES.

1 Q IS THAT TABLE SET FORTH ON SLIDE 109?

2 A YES.

3 Q ALL RIGHT. AND THAT SLIDE IS TITLED, "U.S.  
4 F.D.A. TEST RESULTS FOR ACRYLAMIDE CONCENTRATIONS IN  
5 BREWED COFFEE, 2003 TO 2004"; IS THAT CORRECT?

6 A YES, AND I WOULD LIKE YOU TO KNOW THAT THE  
7 CALIFORNIA E.P.A. IN THEIR 2005 DOCUMENT ON ACRYLAMIDE  
8 INTAKE IN CERTAIN FOODS ALSO USED THE SAME DATA THAT I  
9 DID.

10 Q YOU HAVE GIVEN THAT DATA HERE IN THE SECOND  
11 COLUMN FOR ACRYLAMIDE IN P.P.B.; IS THAT CORRECT?

12 A THAT IS CORRECT.

13 Q WHAT IS "P.P.B."?

14 A PARTS PER BILLION.

15 Q AND THE VALUES THAT APPEAR RANGE FROM THREE  
16 PARTS PER BILLION TO 13 PARTS PER BILLION; IS THAT  
17 CORRECT?

18 A THAT'S CORRECT.

19 Q AND YOU DETERMINED AN AVERAGE CONCENTRATION  
20 OF ACRYLAMIDE IN BREWED COFFEE BASED UPON THIS DATA SET?

21 A THAT IS CORRECT.

22 Q HOW DID YOU DO THAT?

23 WHAT KIND OF AN ESTIMATE OF TENDENCY DID YOU  
24 USE?

25 A ESSENTIAL ESTIMATE, MEAN AVERAGE.

26 Q SO YOU SIMPLY TOTALED THOSE UP AND DIVIDED  
27 BY THE NUMBER OF SAMPLES, DIVIDED BY 20?

28 A THAT'S CORRECT.

1 Q WHAT DID YOU END UP WITH?

2 A 7.35 P.P.B. OR MICROGRAMS PER KILOGRAM.

3 Q IS P.P.B. THE SAME AS MICROGRAMS PER  
4 KILOGRAM?

5 A IN WATER. IT IS WET WEIGHT. IT IS NOT IN  
6 THE AIR, BUT IT IS IN WATER.

7 Q ALL RIGHT. SO NOW, WHAT DID YOU CONCLUDE  
8 FROM THIS EXERCISE?

9 A THAT AN INDEPENDENT AGENCY TOOK 20 SAMPLES,  
10 AND THOSE SAMPLES OF AMERICAN COFFEE -- THOSE ARE ALL  
11 AMERICAN COFFEE, I WANTED GET AMERICAN SAMPLES -- AND  
12 THEY AVERAGED 7.35 MICROGRAMS PER KILOGRAM. THAT IS THE  
13 CONCENTRATION IN COFFEE.

14 Q THAT IS THE CONCENTRATION OF ACRYLAMIDE IN  
15 BREWED COFFEE BASED UPON THE F.D.A. DATA SET?

16 A THAT'S CORRECT.

17 Q WHAT DID YOU DO NEXT IN YOUR EXPOSURE  
18 ASSESSMENT?

19 A I THINK I ESTIMATED THE AMOUNT OF COFFEE  
20 THAT PEOPLE DRANK.

21 Q OKAY. AND HOW DID YOU GO ABOUT DETERMINING  
22 HOW MUCH COFFEE CALIFORNIANS DRANK EVERY DAY?

23 A WELL, I LOOKED IN THE NATIONAL COFFEE  
24 ASSOCIATION TABLE. IT WAS A DATA TABLE. IT WAS AN EXCEL  
25 TABLE THAT YOU GAVE ME. I LOOKED AT -- THERE IS A WHOLE  
26 DATA SET. THE NATIONAL COFFEE ASSOCIATION USES THESE  
27 DATA FOR THEIR ANNUAL REPORT. I LOOKED UNDER THE TITLE,  
28 "PEOPLE WHO DRANK COFFEE YESTERDAY."

1 I TOOK THE NUMBER OF -- IT WOULD PROBABLY  
2 HELP IF YOU COULD MOVE ONE TABLE.

3 THAT WORKS FINE.

4 SO THE FIRST THING I DID WAS USED THE N.C.A.  
5 TABLE, THE EXCEL FILE. THEN I LOOKED IN THE WEST REGION  
6 BECAUSE THE N.C.A. SAMPLED PEOPLE THROUGHOUT THE COUNTRY  
7 BY REGIONS. FOR THE WEST REGION SAMPLE, THEY HAD 653  
8 PEOPLE.

9 Q WHY DID YOU CHOSE THE WEST REGION?

10 A BECAUSE I WAS INTERESTED IN CALIFORNIA.

11 Q WHAT DID YOU FIND?

12 A 533 OF THOSE WERE CALIFORNIANS, 82 PERCENT.  
13 AND OF THOSE, 428 REPLIED THAT THEY HAD DRANK COFFEE  
14 YESTERDAY. SO OUT OF FOUR -- OUT OF 653 PEOPLE IN THE  
15 WEST REGION, 428, WHICH IS 65 PERCENT OF THE SAMPLE SET,  
16 SAID, "YES, I DRANK COFFEE YESTERDAY."

17 Q WHAT DID YOU CONCLUDE FROM THAT?

18 A THAT A LOT OF PEOPLE DRINK COFFEE.

19 Q OKAY.

20 A I'M SORRY. SIXTY-FIVE PERCENT OF THE PEOPLE  
21 WERE REGULAR COFFEE DRINKERS. AND I WANTED TO DETERMINE  
22 REGULAR COFFEE DRINKERS BECAUSE THE CALIFORNIA  
23 REGULATIONS SPECIFY THAT THE RISKS SHOULD APPLY TO PEOPLE  
24 WHO USE THE PRODUCT. OKAY?

25 Q OKAY.

26 A NOW, AND THEN I DID SUBSEQUENT CALCULATIONS  
27 ON THE SAME TABLE AND DETERMINED THAT THESE 428 DRINKERS  
28 DRANK AN AVERAGE OF 3.08 CUPS PER DAY.

1 Q OKAY. ALL RIGHT. SO WE NOW HAVE THE NUMBER  
2 OF -- ALL RIGHT. SO WHAT DID YOU DO NEXT?

3 A WELL, SO NOW I KNEW THE CONCENTRATION IN THE  
4 COFFEE, AND I KNEW THE NUMBER OF CUPS THEY DRANK, BUT I  
5 JUST DID NOT KNOW THE SIZE OF THE CUP. THEY COULD HAVE  
6 BEEN SMALL CUPS.

7 Q HOW DID YOU DETERMINE THAT?

8 A I USED ANOTHER TABLE, BECAUSE THEY ASKED --  
9 THE SURVEY CONTINUED AND SAID, "WELL, WHAT SIZE CUPS DID  
10 YOU DRINK?"

11 Q THIS IS THE SAME N.C.A. SURVEY?

12 A YES.

13 Q WHAT DID YOU FIND?

14 A I THINK -- I DON'T REMEMBER THE NUMBERS, BUT  
15 I AM PRETTY SURE IT IS IN THE SLIDES.

16 SO I LOOKED AND THESE PEOPLE REPLIED ON THE  
17 SIZE OF THE COFFEE CUPS THAT THEY USED. FROM THAT, I  
18 DETERMINED THAT THE AVERAGE OUNCES -- THE AVERAGE SIZE OF  
19 THE CUP WAS 10.66 OUNCES.

20 Q HOW DID YOU REACH THAT CONCLUSION?

21 A WELL, THEY REPLIED THAT THEY DRANK A  
22 TOTAL -- WELL, A CERTAIN NUMBER OF PEOPLE -- CERTAIN --  
23 THEY SAID, "WELL, WHAT SIZE CUP DID YOU DRINK?"

24 "THREE, EIGHT, TWELVE, SIXTEEN OUNCES?"

25 OF THOSE PEOPLE WHO REPLIED, THEY SAID,

26 "THIS IS THE SIZE CUP I USE."

27 SO YOU MULTIPLY THE FREQUENCY OF THE PEOPLE  
28 TIMES THE SIZE OF THE CUP AND DIVIDE BY THE TOTAL NUMBER

1 OF THE PEOPLE, AND YOU COME UP WITH THE TOTAL NUMBER  
2 OUNCES PER CUP, AVERAGE NUMBER OF OUNCES PER CUP.

3 Q SO YOU HAVE -- THE NUMBER OF OUNCES PER CUP  
4 YOU HAVE AS 10.66 OUNCES PER CUP, AND THAT THE PEOPLE IN  
5 THE WEST REGION CONSUMED 3.08 CUPS PER DAY, AND YOU  
6 DETERMINED THE AMOUNT OF ACRYLAMIDE IN THE -- IN COFFEE  
7 FROM THE F.D.A. DATA, AND WHAT DID YOU DO WITH THAT  
8 INFORMATION?

9 A I'M SORRY, I DETERMINED THE CONCENTRATION OF  
10 ACRYLAMIDE IN COFFEE FROM THE F.D.A. DATA, NOT THE  
11 AMOUNT.

12 Q THANK YOU FOR CLARIFYING THAT.

13 A WELL, I WANTED TO FIND OUT HOW MUCH  
14 ACRYLAMIDE WAS IN AN AVERAGE CUP OF COFFEE.

15 Q WHAT IS THE FORMULA FOR THAT?

16 A IT IS A SIMPLE FORMULA, THE CONCENTRATION  
17 TIMES THE SIZE OF THE CUP, AND THEN YOU CONVERT FROM  
18 OUNCES TO MICROGRAMS. THE SIZE OF THE CUP IS IN OUNCES.

19 Q DID YOU MAKE THAT CALCULATION?

20 A YES, IT IS IN THE NEXT SLIDE.

21 Q THAT WAS WHAT?

22 A MY CONVERSION WAS 7.35 MICROGRAMS PER  
23 KILOGRAM, AND IT WAS THE CONCENTRATION IN THE COFFEE,  
24 TIMES THE SIZE OF THE CUP, TIMES THE CONVERSION OF 28  
25 GRAMS PER OUNCE. IT COMES OUT WITH 2.22 MICROGRAMS OF  
26 ACRYLAMIDE PER CUP OF COFFEE. SO THAT IS THE AMOUNT OF  
27 ACRYLAMIDE IN A 10.66 OUNCE CUP OF COFFEE.

28 Q ALL RIGHT. WAS THAT THE END OF YOUR

1 EXPOSURE ANALYSIS OR DID YOU HAVE TO DO SOMETHING  
2 FURTHER?

3 A I DON'T REMEMBER.

4 OH, WHAT I HAD TO DO WAS CONVERT THE 2.22  
5 MICROGRAMS INTO MICROGRAMS PER KILOGRAM BODY WEIGHT OF  
6 HUMAN.

7 Q WHY DID YOU NEED TO DO THAT CONVERSION?

8 A BECAUSE MY POTENCY ESTIMATES ARE IN THOSE  
9 UNITS.

10 Q OKAY, AND WHAT WAS -- HOW DID YOU DO THAT  
11 CONVERSION?

12 A I TOOK 2.22 MICROGRAMS DIVIDED BY 70  
13 KILOGRAMS AND CAME UP WITH A FIGURE OF 0.03 MICROGRAMS OF  
14 ACRYLAMIDE PER KILOGRAM BODY WEIGHT IN ONE CUP OF COFFEE.

15 Q THE 70 KILOGRAMS PER THE HUMAN, WAS THAT  
16 SPECIFIED IN THE CALIFORNIA REGULATIONS?

17 A IT IS SPECIFIED IN CALIFORNIA, AND I THINK I  
18 HAVE ALREADY SAID THAT, AND IT IS ALSO SPECIFIED IN U.S.  
19 E.P.A. GUIDELINES.

20 Q ALL RIGHT. DOES THAT NOW COMPLETE YOUR  
21 EXPOSURE ASSESSMENT?

22 A YES.

23 Q THEN THE LAST COMPONENT OF THE RISK  
24 ASSESSMENT IS THE RISK CHARACTERIZATION; CORRECT?

25 A YES.

26 Q THAT IS -- TO DO THAT, YOU NEED THE  
27 INFORMATION FROM THE EXPOSURE ASSESSMENT AND THE  
28 DOSE-RESPONSE, THE POTENCY?

1 A RISK EQUALS POTENCY TIMES EXPOSURE.

2 Q SO YOU NOW HAVE THE VALUES, YOU CAN DO THAT?

3 A I HAVE POTENCY, I HAVE THE HUMAN CANCER  
4 POTENCY ESTIMATES, AND I HAVE THE EXPOSURE ESTIMATES.

5 Q OKAY.

6 A NOW, I WILL JUST MULTIPLY THE TWO AND GET  
7 RISK.

8 Q ALL RIGHT. DID YOU NEED TO MAKE ANY  
9 ADJUSTMENTS TO THE HUMAN CANCER POTENCY VALUES?

10 A YES, I DID.

11 Q WHY?

12 A BECAUSE PEOPLE -- MY SEARCH THROUGH THE  
13 LITERATURE, AND MY INTUITION, I GUESS, SAID I CAN'T --  
14 PEOPLE UNDER 17 DON'T DRINK COFFEE BASICALLY. THE N.C.A.  
15 STUDIES THAT I LOOKED AT HAD CALCULATIONS FOR AGES 13 AND  
16 ABOVE, AND AGES 18 AND ABOVE. COFFEE CONSUMPTION FIGURES  
17 FOR THOSE AGE GROUPS. I DID NOT SEE MUCH COFFEE DRINKING  
18 AT ALL UNDER THE AGE OF 17 OR SO.

19 SO I SAID, WELL, I CAN'T CALCULATE A RISK  
20 FOR KIDS 14 AND 15 BECAUSE THEY DON'T DRINK COFFEE. SO  
21 IF I AM GOING TO TRY TO BE TRUE TO RISK ASSESSMENT, I CAN  
22 ONLY LOOK AT HOW MUCH COFFEE PEOPLE DRANK FROM THE AGES  
23 OF 17 ON.

24 Q OKAY. SO WHAT -- WHAT TYPE OF ADJUSTMENT  
25 DID YOU MAKE?

26 A WELL, I HAD TO ADJUST THE POTENCY ESTIMATES  
27 DOWN BECAUSE THESE ANIMALS WERE EXPOSED TO THIS THEIR  
28 WHOLE LIFE, AND NOW HUMANS ARE ONLY GOING TO BE EXPOSED

1 TO ACRYLAMIDE IN COFFEE FROM AGES 17 ON. SO I HAD TO  
2 REDUCE THE POTENCY ESTIMATE BY A FACTOR OF 54 OVER 70.

3 THE COURT: LET ME ASK YOU A QUESTION. GOING BACK  
4 TO THE PREVIOUS SLIDE, SO MANY MICROGRAMS PER CUP OF  
5 COFFEE. WHAT DOES THAT TRANSLATE IN TERMS OF PARTS PER  
6 MILLION?

7 THE WITNESS: OH, IT IS MUCH LESS IN PARTS PER  
8 MILLION. CAN I DO IT IN PARTS PER BILLION?

9 THE COURT: YEAH.

10 THE WITNESS: I THINK IT IS ABOUT THREE PARTS PER  
11 BILLION.

12 THE COURT: THREE PARTS ACRYLAMIDE FOR BILLION  
13 PARTS OF COFFEE?

14 THE WITNESS: WELL, IT IS NOT CONCENTRATION, IT  
15 IS -- BECAUSE NOW WE HAVE GONE FROM CONCENTRATION.  
16 ACRYLAMIDE IN THE COFFEE IS SEVEN PARTS PER BILLION,  
17 SEVEN AND A HALF.

18 THE COURT: SEVEN AND A HALF PER BILLION?

19 THE WITNESS: ACRYLAMIDE IN COFFEE IS 7.35 PARTS  
20 PER BILLION, BUT WHEN IT GOES INTO THE HUMAN BODY, IT  
21 IS -- YOU -- ITS CONCENTRATION IN THE HUMAN BODY HAS TO  
22 BE -- YOU HAVE TO GET THE AMOUNT OF ACRYLAMIDE PER BODY  
23 WEIGHT. SO NOW IT IS --

24 THE COURT: I KNOW THAT IS THE FOLLOWING SLIDE. I  
25 AM CURIOUS ABOUT THE EARLIER ONE. NOW YOU ARE  
26 TRANSLATING IT IN TERMS OF BODY WEIGHT?

27 THE WITNESS: THAT'S CORRECT.

28 Q BY MR. METZGER: ARE WE NOW AT THE POINT

1 WHERE YOU ARE GOING TO DETERMINE OR CALCULATE THE EXCESS  
2 CANCER RISK?

3 A YES.

4 Q DID YOU DO THAT FOR DIFFERENT COFFEE  
5 CONSUMPTION LEVELS?

6 A YES.

7 Q AND HOW DID YOU SELECT THE DIFFERENT COFFEE  
8 CONSUMPTION LEVELS?

9 A WELL, I ACTUALLY USED THE SAME NATIONAL  
10 COFFEE ASSOCIATION SLIDE DATA, WHICH ASKED PEOPLE: "HOW  
11 MUCH COFFEE DID YOU DRINK A DAY?"

12 Q OKAY.

13 A IT SEEMS REASONABLE TO ME THAT I WANTED TO  
14 LOOK AT NOT ONLY OF THE AVERAGE DRINKER WHO DRANK 3.1  
15 CUPS A DAY, BUT PEOPLE WHO DRANK FROM ONE TO NINE CUPS A  
16 DAY. WHY DID I PICK THESE, I PICKED THEM TO GET A  
17 SPREAD. I MEAN, ONE CUP OF COFFEE A DAY IS A BASIC  
18 STANDARD.

19 SO IT TURNS OUT THAT OF THE 428 WESTERNERS  
20 WHO SAID THEY DRANK COFFEE, THE NINTH PERCENTILE  
21 REPRESENTED ONE CUP PER DAY. THE AVERAGE DRINKER OF 3.1  
22 COFFEES REPRESENTED THE 48TH PERCENTILE. SO ROUGHLY HALF  
23 THE PEOPLE DRANK THE AVERAGE AMOUNT OF COFFEE OF 3.1 PER  
24 DAY. FIVE CUPS WERE -- IS IT 71 PERCENT PER DAY?

25 Q GO TO SLIDE 122, WHICH, I THINK, HAS THE  
26 DATA THAT YOU --

27 A FIVE CUPS PER DAY -- I WANTED TO GET THE  
28 RISK FOR FIVE CUPS PER DAY, WHICH REPRESENTED THE 71ST

1 PERCENTILE OF THE SAMPLE. SEVEN CUPS PER DAY, THE 85TH.  
2 AND NINE CUPS PER DAY, I CAN'T IMAGINE WHO CAN DRINK NINE  
3 CUPS PER DAY, BUT IT WAS 91ST PERCENTILE.

4 SO I ESTIMATED THE RISK FROM ONE THROUGH  
5 NINE CUPS PER DAY.

6 Q ALL RIGHT. DID YOU DO THAT FOR BOTH THE  
7 DATA INCLUDING THE HARDERIAN GLAND AND FOR THE DATA  
8 EXCLUDING THE HARDERIAN GLAND?

9 A YES.

10 Q GO TO SLIDE 124.

11 IS THIS THE DATA THAT DOES THAT WITH THE  
12 HARDERIAN GLAND TUMORS INCLUDED?

13 A YES.

14 Q ALL RIGHT. SO TELL US, FIRST OF ALL, WHAT I  
15 THINK THIS IS -- THIS IS THE SUMMARY SLIDE?

16 A IT IS ONE OF THEM, YES.

17 Q TELL US WHAT THE COLUMNS REPRESENT AND WE  
18 WILL GO THROUGH THIS.

19 A OKAY. I WANTED TO ESTIMATE THE P.M.I. RISK,  
20 THE RISK THAT I GOT WITH THE CALIFORNIA E.P.A., THE U.S.  
21 E.P.A., AND I HAD BOTH RAT AND MOUSE STUDIES. SO THAT IS  
22 THE FIRST COLUMN.

23 Q ALL RIGHT. AND THE SECOND COLUMN?

24 A THE SECOND COLUMN IS THE ADJUSTED HUMAN  
25 CANCER FACTOR, CANCER POTENCY FACTOR. REMEMBER, I SAID  
26 WE HAD TO ADJUST THE HUMAN CANCER FACTOR POTENCIES DOWN  
27 BECAUSE HUMANS LESS THAN UNDER THE AGE OF 17, I ASSUMED  
28 DRANK NO COFFEE.

1 Q OKAY.

2 A SO THEY DID NOT GET ANY ACRYLAMIDE FROM THAT  
3 COFFEE.

4 Q OKAY. THEN THE NEXT COLUMN IS COFFEE  
5 CONSUMPTION IN CUPS PER DAY FOR THE THREE -- I'M SORRY,  
6 FOR THE FIVE CONSUMPTION LEVELS THAT YOU MENTIONED?

7 A THAT'S CORRECT.

8 Q AND THEN THE LAST COLUMNS ARE WHAT?

9 A THOSE ARE THE NONSIGNIFICANT RISK LEVELS.  
10 NO SIGNIFICANT RISK LEVELS THAT I CALCULATED.

11 Q NO SIGNIFICANT RISK LEVEL?

12 A LEVELS.

13 Q ARE THOSE -- WHAT IS A NO SIGNIFICANT RISK  
14 LEVEL?

15 A IT IS THE AMOUNT OF ACRYLAMIDE PER DAY WHICH  
16 WILL PRODUCE ONE IN 100,000 EXCESS LIFETIME RISK.

17 Q CANCERS?

18 A EXCESS CANCER RISKS IF CONSUMED YOUR WHOLE  
19 LIFETIME.

20 Q IS THAT A STATUTORY DEFINITION?

21 A YES.

22 Q OKAY. ALL RIGHT. I SEE FOR THE LAST COLUMN  
23 THERE IS ACTUALLY A BREAKDOWN FOR ACRYLAMIDE FROM 54  
24 YEARS OF COFFEE CONSUMPTION, WHICH IS NOT INCLUDING THE  
25 CHILDREN; RIGHT?

26 A THAT IS CORRECT.

27 Q AND THEN YOU HAVE IN THE LAST COLUMN FOR 70  
28 YEARS?

1           A           THAT'S CORRECT.

2           Q           WHY DO YOU HAVE THAT COLUMN FOR 70 YEARS?

3           A           WELL, LET'S LOOK AT THE LAST TWO ENTRIES ON  
4 THE BOTTOM ROW.

5           Q           OKAY.

6           A           IT IS JUST EASIER TO SPEAK THAT WAY.  
7 ACCORDING TO THE CALIFORNIA STATUTE, IT SAYS, IF YOU  
8 DRINK THIS EVERY DAY OF YOUR LIFE, AND YOU ONLY GET -- IF  
9 YOU DRINK COFFEE EVERY DAY OF YOUR LIFE BECAUSE WE ARE  
10 DOING ACRYLAMIDE IN COFFEE, THEN THE ACRYLAMIDE YOU GET  
11 CAN TOTAL .26 MICROGRAMS PER DAY.

12          Q           SO ARE YOU SAYING THAT THE REGULATION SAYS  
13 THAT YOU SHOULD ASSUME A 70-YEAR CONSUMPTION?

14          A           THE REGULATION SAYS --

15           MR. SCHURZ: OBJECTION TO THE EXTENT IT CALLS FOR A  
16 LEGAL CONCLUSION AND LEADING.

17           THE COURT: OVERRULED.

18           THE WITNESS: THE REGULATION SAYS, YEAH, YOU CAN  
19 DRINK THIS FOR 70 YEARS. IF YOU DO IT FOR 70 YEARS, THEN  
20 WHAT IS NO SIGNIFICANT RISK LEVEL?

21                    BUT I THOUGHT THAT THAT REALLY WASN'T FAIR  
22 BECAUSE YOU ARE PENALIZING PEOPLE WHO START DRINKING WHEN  
23 THEY ARE 17 ON. SO I AM LITERALLY -- CARRIED TO ITS  
24 LOGICAL CONCLUSION, PEOPLE COULD START DRINKING AT AGE 70  
25 AND STILL ONLY HAVE .26 MICROGRAMS PER DAY. YOU WOULD  
26 STILL HAVE TO CALCULATE THAT ACCORDING TO THE CALIFORNIA  
27 REGULATION.

28                    BUT IF YOU ASSUME THAT PEOPLE START DRINKING

1 AT AGE 17, AND CONTINUE DRINKING THE REST OF THEIR LIFE,  
2 THEN IN ORDER TO REACH THE TOTAL AMOUNT OF ACRYLAMIDE  
3 THAT THEY ABSORB, THEY CAN -- THAT WILL CAUSE ONE IN  
4 100,000 CANCER RISK, THEY CAN DRINK .34 MICROGRAMS PER  
5 DAY OF ACRYLAMIDE IN THE COFFEE.

6 Q OKAY. ALL RIGHT. SO IN THIS TABLE, THE  
7 FIRST ROW THAT YOU HAVE IS FOR THE CAL E.P.A. 2005, AND  
8 WHAT IS THAT?

9 WHAT IS THAT DOCUMENT?

10 A IT IS THE CALIFORNIA DOCUMENT ON THE RISK OF  
11 ACRYLAMIDE.

12 Q WHEN YOU SAY THE RISK OF ACRYLAMIDE, YOU  
13 MEAN WHERE CALIFORNIA IN 2005 DID A CALCULATION TO  
14 ESTABLISH THE NO SIGNIFICANT RISK LEVEL FOR ACRYLAMIDE?

15 A THAT IS CORRECT.

16 MR. SCHURZ: OBJECTION; LACKS FOUNDATION. WE HAVE  
17 SEEN THE DOCUMENT AND IT HAS BEEN MISREPRESENTED. THAT  
18 IS NOT WHAT THAT DOCUMENT SAYS.

19 THE COURT: OVERRULED, SUBJECT TO CROSS-  
20 EXAMINATION.

21 MR. METZGER: OKAY.

22 Q TELL US WHAT THE DATA OR RESULTS WERE FOR  
23 THAT DOCUMENT THAT YOU HAVE LISTED HERE THAT -- GO AHEAD.

24 A WHAT THE -- I USED THE POTENCY ESTIMATE  
25 DEVELOPED BY THE STATE OF CALIFORNIA IN THEIR 2005  
26 ACRYLAMIDE DOCUMENT. I ADJUSTED THAT POTENCY ESTIMATE  
27 DOWNWARD TO ACCOUNT FOR THE FACT THAT COFFEE DRINKING  
28 ONLY BEGAN AT AGE 17. I MULTIPLIED THAT, THAT CALIFORNIA

1 POTENCY ESTIMATE, WHICH WAS .54, BY THE EXPOSURE OF  
2 ACRYLAMIDE IN NUMBERS OF CUPS OF COFFEE, RANGING FROM ONE  
3 TO NINE TO DETERMINE TOTAL LIFETIME EXCESS CANCER RISK.

4 Q SO IF WE LOOK FOR ONE CUP OF COFFEE PER DAY,  
5 THE ACRYLAMIDE CONTENT OF 2.22 FOR USING THE CAL E.P.A.  
6 2005, YOU HAVE "1.72 E MINUS 5."

7 WHAT DOES "E MINUS 5" MEAN?

8 A THAT IS COMPUTER NOTATION FOR 10 TO THE  
9 MINUS 5. THAT IS EQUAL TO 1.72 TIMES 10 TO THE MINUS 5  
10 OR 1.7 CASES INCREASED CANCERS PER 100,000.

11 Q OKAY. SO FOR THE AVERAGE COFFEE DRINKER WHO  
12 DRINKS ABOUT 3.1 CUPS PER DAY, WHAT IS THE EXCESS CANCER  
13 RISK BASED UPON THE CAL E.P.A. 2005?

14 MR. SCHURZ: OBJECTION; LACKS FOUNDATION. COUNSEL  
15 IS TESTIFYING.

16 THE COURT: OVERRULED.

17 THE WITNESS: IT IS THE CALIFORNIA E.P.A. 2005  
18 CANCER POTENCY ESTIMATE ADJUSTED DOWNWARD. FOR 3.1 CUPS  
19 PER DAY, I -- MY ESTIMATE OF INCREASED CANCER RISK ARE  
20 FIVE PER 100,000 LIFETIME.

21 Q BY MR. METZGER: FOR CONSUMPTION OF FIVE  
22 CUPS PER DAY?

23 A 8.6 CANCER, EXCESS CANCERS LIFETIME PER  
24 100,000.

25 Q FOR CONSUMPTION OF SEVEN CUPS OF COFFEE A  
26 DAY?

27 A 12 CANCERS PER 100,000.

28 Q AND FOR CONSUMPTION OF NINE CUPS OF COFFEE

1 PER DAY?

2 A 15.4 EXCESS CANCERS PER 100,000.

3 Q ALL RIGHT. AND WHAT IS THE NEXT COLUMN THAT  
4 YOU HAVE?

5 A THE NEXT COLUMN IS NONSIGNIFICANT RISK LEVEL  
6 BASED ON 54 YEARS OF COFFEE DRINKING, NOT 70 YEARS.

7 Q AND YOU HAVE THE VALUE THERE BASED ON CAL  
8 E.P.A. 2005 OF 1.30 MICROGRAMS PER DAY?

9 A CAL E.P.A. DID NOT ESTIMATE THAT NUMBER.  
10 THEY ESTIMATED 1.0 MICROGRAMS PER DAY BECAUSE THEY  
11 ESTIMATED THE N.S.R.L. FOR 70 YEARS. THE REASON I  
12 ADJUSTED IT TO -- FROM 1.0 TO 1.3 WAS TO ACCOUNT THAT --  
13 THAT THIS QUANTITATIVE CANCER RISK ASSESSMENT IS FOR  
14 ACRYLAMIDE IN COFFEE.

15 Q SO YOU EXCLUDED THE CHILDREN?

16 A EXACTLY.

17 Q GOT IT. ALL RIGHT. THE NEXT ROW THAT YOU  
18 HAVE IS DENOMINATED "U.S. E.P.A. 2010." WHAT IS THAT  
19 DOCUMENT?

20 A THAT IS THE U.S. E.P.A. RISK ASSESSMENT  
21 DOCUMENT FOR ACRYLAMIDE.

22 Q FROM 2010?

23 A YES.

24 Q ALL RIGHT. AND WHAT DID YOU CONCLUDE FROM  
25 THAT?

26 A I CONCLUDED THAT THEIR RISK ESTIMATES FOR  
27 ONE CUP OF COFFEE PER DAY WAS 1.2 PER 100,000 EXCESS  
28 CANCERS OVER A LIFETIME DRINKING.

1 Q FOR THREE CUPS PER DAY?

2 A 3.8 PER 100,000.

3 Q AND FOR FIVE CUPS PER DAY?

4 A 6.1 PER 100,000.

5 Q FOR SEVEN CUPS PER DAY?

6 A 8.6 PER 100,000.

7 Q AND FOR NINE CUPS PER DAY?

8 A 11 PER 100,000.

9 Q ALL RIGHT. AND WHAT DID YOU CONCLUDE WAS  
10 THE N.S.R.L. FOR -- BASED UPON THE E.P.A. DOCUMENT FOR 70  
11 YEARS VERSUS 54 YEARS OF CONSUMPTION?

12 A WELL, IT WAS 1.4 FOR 70 YEARS, BUT IF YOU  
13 ONLY DRANK IT FOR 54 YEARS, IT WAS 1.8.

14 Q OKAY.

15 A MICROGRAMS PER DAY.

16 Q I THINK YOU INDICATED THAT THE LAST TWO ROWS  
17 IN THIS TABLE ARE YOUR ANALYSES BASED UPON THE N.T.P. RAT  
18 AND MOUSE DATA; IS THAT CORRECT?

19 A THAT IS CORRECT.

20 Q ALL RIGHT. TELL US FIRST REGARDING THE RAT  
21 DATA, WHAT YOU CONCLUDED?

22 A THE ESTIMATES WERE ALL VERY SIMILAR TO THOSE  
23 OF CAL E.P.A. AND U.S. E.P.A. BASED ON THE RAT ALSO.

24 Q ALL RIGHT. AND SO FOR THE ADJUSTED HUMAN  
25 CANCER FACTOR, YOU HAVE 0.517?

26 A THAT'S CORRECT.

27 Q AND THAT IS -- THAT NUMBER WAS PREVIOUSLY  
28 PROVIDED IN THE EARLIER TABLE; IS THAT CORRECT?

1           A           THAT'S CORRECT. NO. NO, IT WASN'T PROVIDED  
2 IN THE EARLIER TABLES. THE HIGHER POTENCY ESTIMATE WAS  
3 PROVIDED IN THE EARLIER TABLE. HERE I ADJUSTED FOR NO  
4 COFFEE DRINKING UP TO 17.

5           Q           ALL RIGHT. AND BASED UPON THE N.T.P. RAT  
6 STUDY, TELL US WHAT THE EXCESS CANCER RISK PER 100,000  
7 WERE FOR ONE, THREE, FIVE, SEVEN AND NINE CUPS OF COFFEE  
8 CONSUMED A DAY?

9           A           FOR ONE CUP OF COFFEE, THE EXCESS RISK PER  
10 100,000 WAS 1.4 PER 100,000.

11          Q           IS THAT 1.64?

12          A           1.64 PER 100,000. I APOLOGIZE.  
13                      FOR THREE CUPS, IT WAS 5.1 PER 100,000.  
14                      FOR FIVE CUPS PER DAY, IT WAS 8.2 PER  
15 100,000.

16                      FOR SEVEN CUPS PER DAY, IT IS ONE POINT --  
17 IT IS 11.5 PER 100,000.

18                      AND FOR NINE CUPS PER DAY, IT IS 14.8 PER  
19 100,000.

20          MR. SCHURZ: YOUR HONOR, WE WILL OBJECT TO THE --  
21 THIS HAS HAPPENED ON A NUMBER OF OCCASIONS, THE WITNESS  
22 HAS TESTIFIED WITH RESPECT TO VALUES THAT ARE NOT  
23 REPRESENTED ON THE TABLE AND DIFFER SUBSTANTIALLY FROM  
24 THE TABLE. SO SOMEBODY IS WRONG. EITHER THE TABLE HAS  
25 MULTIPLE ERRORS, AND ALTHOUGH REVISED SUBSEQUENTLY TO THE  
26 DEPOSITION, IS ERRONEOUS, OR DR. BAYARD HAS NOT GIVEN  
27 TRUTHFUL AND ACCURATE REFLECTIONS WITH RESPECT TO THE  
28 VALUES THAT ARE SUPPOSED TO BE REFLECTED HERE.

1 THE COURT: ALL RIGHT. WELL, YOU CAN GO INTO THAT  
2 IN CROSS-EXAMINATION. AT THIS TIME, WE ARE GOING TO  
3 RECESS FOR 15 MINUTES.

4  
5 (RECESS TAKEN.)

6  
7 THE COURT: COUNSEL IN CERT, READY TO RESUME TRIAL?  
8 BACK ON THE RECORD IN THE CASE OF CERT  
9 VERSUS STARBUCKS. ALL COUNSEL ARE PRESENT. DR. BAYARD  
10 IS ON THE STAND.

11 DR. BAYARD, YOU UNDERSTAND YOU ARE STILL  
12 UNDER OATH?

13 THE WITNESS: YES, SIR.

14 THE COURT: PLEASE REPEAT YOUR NAME FOR THE RECORD.

15 THE WITNESS: STEVEN BAYARD.

16 THE COURT: THANK YOU.

17 MR. METZGER, YOU MAY PROCEED.

18 MR. METZGER: THANK YOU, YOUR HONOR.

19 Q WE WERE ON YOUR TABLE 4.A.1, REGARDING THE  
20 INCREASED RISK OF CANCER FROM ACRYLAMIDE IN COFFEE AND  
21 THE NO SIGNIFICANT RISK LEVEL. THIS TABLE INCLUDING THE  
22 HARDERIAN GLANDS TUMORS.

23 DR. BAYARD, WHAT I WOULD LIKE YOU TO DO IS  
24 TO TAKE A LOOK AT EXHIBIT 130.

25 A YES, SIR.

26 Q SPECIFICALLY ON THE LAST PAGE OF THAT  
27 EXHIBIT, WHICH IS YOUR DEPOSITION CORRECTIONS, YOU HAVE A  
28 TABLE 4.A.1. I WOULD LIKE YOU TO TELL THE COURT WHETHER

1 THE DATA THAT IS IN -- WHAT YOU HAVE NOW PRESENTED, WHICH  
2 IS IN SLIDE 124, IS THE DATA THAT IS IN THAT TABLE THAT  
3 YOU PROVIDED AS A CORRECTION TO YOUR DEPOSITION?

4 A THOSE ARE THE RESULTS. THEY ARE NOT DATA.

5 Q I'M SORRY. I WAS IMPRECISE.

6 ARE THOSE RESULTS THE SAME THAT YOU  
7 PRESENTED HERE IN COURT AS WHAT WAS IN THIS TABLE THAT  
8 YOU PROVIDED AS A CORRECTION TO YOUR DEPOSITION?

9 A THAT'S CORRECT. YES, THEY ARE.

10 Q NOW, I JUST WANT TO MAKE SURE SOMETHING IS  
11 CLEAR, IF WE LOOK AT THE LAST ROW WHERE YOU HAVE FOR THE  
12 N.T.P. MOUSE, FOR ONE CUP OF COFFEE CONSUMED PER DAY, YOU  
13 HAVE "6.52 E TO THE MINUS 5," AND THAT REPRESENTS EXACTLY  
14 WHAT?

15 A 6.52 CANCER -- EXCESS CANCER CASES PER  
16 100,000.

17 Q ALL RIGHT. THE NEXT COLUMN, FOR CONSUMPTION  
18 OF 3.1 CUPS OF COFFEE PER DAY, THE AVERAGE, YOU HAVE  
19 "2.01 E TO THE MINUS 4." WHAT DOES THAT REPRESENT?

20 A 20.1 EXCESS CANCERS PER 100,000 OR AS  
21 WRITTEN, 2.01 PER 10,000.

22 Q "E TO THE MINUS 4" IS?

23 A TEN TO THE MINUS 4 IS ONE PER 10,000.

24 Q THANK YOU FOR CLARIFYING THAT. ALL RIGHT.

25 SO WHAT DID YOU CONCLUDE, BASED UPON YOUR  
26 ANALYSIS, WAS THE ACRYLAMIDE CONSUMPTION IN MICROGRAMS  
27 PER DAY THAT PRODUCED AN EXCESS CANCER RISK OF ONE IN  
28 100,000?

1           A           IF WE USE THE CALIFORNIA STANDARD FOR ONE  
2 FOR EVERY DAY FOR A 70-YEAR LIFETIME, MY ESTIMATE WOULD  
3 BE .26 MICROGRAMS PER DAY WOULD PRODUCE -- FOR A  
4 LIFETIME, WOULD PRODUCE A ONE IN 100,000 RISK, EXCESS  
5 RISK. IF WE USE MY STANDARD WHERE PEOPLE DON'T START  
6 DRINKING COFFEE UNTIL THEY ARE 17, THEN THEY CAN  
7 ACCUMULATE .34 MICROGRAMS PER DAY FOR ONE IN 100,000  
8 EXCESS CANCER RISK.

9           Q           NOW, I THINK YOU TOLD THE COURT THAT THERE  
10 WERE APPROXIMATELY SEVEN POINT SOMETHING PARTS PER  
11 BILLION OF ACRYLAMIDE?

12          A           7.35 PARTS PER BILLION ACRYLAMIDE IN BREWED  
13 COFFEE, AVERAGE.

14          Q           DOES THAT -- DOES THAT, IN THE ANALYSIS THAT  
15 YOU HAVE DONE, TELL YOU ANYTHING ABOUT THE CANCER  
16 POTENCY -- WHETHER ACRYLAMIDE IS A POTENT CARCINOGEN?

17          A           IF -- YES.

18          Q           WHAT DOES IT TELL YOU?

19          A           THAT IF ACRYLAMIDE IN MICROGRAMS PER DAY  
20 PRODUCES THIS MUCH EXCESS CANCER, IT IS POTENT, NOT ONLY  
21 THAT, BUT ACRYLAMIDE ITSELF HAS A LOT OF EVIDENCE THAT  
22 MAKES ONE REALLY BE CONCERNED ABOUT IT. THAT IS THAT  
23 EVERY SPECIES THAT HAS BEEN TESTED EVERY TIME CAUSES  
24 EXCESS CANCERS AT A HIGH RATE, IN MULTIPLE SITES, BOTH  
25 SEXES. IT CAN AFFECT THE TESTES IN RATS. IT IS THE  
26 MAMMARY GLAND IN FEMALES. THOSE ARE SITES WHERE YOU CAN  
27 EXPECT EXCESS CANCERS.

28                    YOU KNOW, THERE ARE NO FALSE POSITIVES HERE.

1 WHEN YOU TEST THIS MANY SPECIES AND YOU GET MULTIPLE  
2 TUMOR SITES ALL OVER THE PLACE, IT IS PRETTY POTENT.

3 Q OKAY. THANK YOU. ALL RIGHT. NOW, DID YOU  
4 ALSO DO A SUMMARY TABLE COMPARABLE TO TABLE 4.A.1, BUT  
5 EXCLUDING THE HARDERIAN GLAND TUMORS?

6 A YES, I DID.

7 Q IS THAT WHAT IS SLIDE 127?

8 A YES.

9 Q OKAY. YOU KNOW, BEFORE WE DO THIS, I WANT  
10 TO GO BACK A MINUTE. GO BACK TO THE LAST TABLE.

11 WHAT DO YOU CONCLUDE FROM THE NUMBERS THAT  
12 YOU HAVE HERE, THE RESULTS THAT YOU HAVE HERE, FOR EXCESS  
13 CANCERS PER 100,000 FOR THE DIFFERENT COFFEE CONSUMPTION  
14 GROUPS?

15 A FOR ANYONE THAT DRINKS MORE THAN ONE CUP OF  
16 COFFEE PER DAY, STARTING AT AGE 17, ON A CONTINUOUS  
17 BASIS, THEIR WHOLE LIFETIME, UP TO 70 YEARS, ALL AGENCIES  
18 WOULD PREDICT, USING MY FIGURES AND THOSE AGENCIES' HUMAN  
19 CANCER POTENCY VALUES, ALL AGENCIES WOULD PREDICT GREATER  
20 THAN ONE IN 100,000 RISK.

21 Q FROM ALL CONSUMPTION LEVELS, FROM ONE UP TO  
22 NINE?

23 A AT LEAST ONE, YES.

24 MR. SCHURZ: I WILL INTERPOSE AN OBJECTION.  
25 CALLING FOR SPECULATION. OF COURSE, CALLING FOR  
26 SPECULATION AND LACKS FOUNDATION BECAUSE NONE OF THESE  
27 REGULATORY AGENCIES HAVE EVER SAID THAT COFFEE CAUSES  
28 CANCER AT ONE IN 100,000 LEVEL RISK.

1 THE COURT: OBJECTION OVERRULED.

2 Q BY MR. METZGER: FIRST OF ALL, ARE WE  
3 TALKING ABOUT COFFEE CAUSING CANCER?

4 A NO.

5 Q WHAT ARE WE TALKING ABOUT?

6 A ACRYLAMIDE IN COFFEE.

7 Q ALL RIGHT. HOW MUCH COFFEE DOES ONE HAVE TO  
8 CONSUME TO GET MORE THAN ONE EXCESS CANCER PER 100,000?

9 A WELL, LET'S TAKE A LOOK AT -- I THINK AN  
10 EASY WAY TO GIVE YOU THIS ANSWER, MAYBE A MORE  
11 DESCRIPTIVE WAY, WOULD BE TO LOOK AT THE NO SIGNIFICANT  
12 RISK LEVEL. THAT IS THE LEVEL OF COFFEE PER DAY --  
13 ACRYLAMIDE IN COFFEE PER DAY WHICH WILL CAUSE ONE IN  
14 100,000 RISK. FOR THE MICE WITH HARDERIAN GLAND TUMORS,  
15 THAT IS 0.26 MICROGRAMS PER DAY. BUT ONE CUP OF COFFEE  
16 CONTAINS 2.2 MICROGRAMS OF ACRYLAMIDE. SO THAT IS ABOUT  
17 EIGHT OR NINE TIMES THE BACKGROUND -- THE NO SIGNIFICANT  
18 RISK LEVEL.

19 SO UNDER MY ESTIMATIONS, ANYTHING --  
20 ANYTHING MORE THAN A CUP OF COFFEE IS -- A HALF A CUP OF  
21 COFFEE IS GOING TO GIVE MORE THAN ONE IN 100,000 LIFETIME  
22 RISK.

23 Q MORE THAN ONE IN 100,000 EXCESS LIFETIME  
24 CANCER RISK?

25 A THAT'S CORRECT.

26 Q THANK YOU, DR. BAYARD. ALL RIGHT.

27 AND YOU DID AN ANALYSIS LIKE THIS WITHOUT  
28 THE HARDERIAN GLAND TUMORS, AND WITHOUT GOING THROUGH ALL

1 THE RESULTS, COULD YOU TELL US -- WELL, FIRST OF ALL,  
2 WHAT HAPPENED WHEN YOU EXCLUDED THE HARDERIAN GLANDS?

3 A WELL, THE MOUSE WAS NO LONGER THE MOST  
4 SENSITIVE STUDY.

5 Q WHAT WAS THE MOST SENSITIVE STUDY?

6 A THE N.T.P. RAT. AT LEAST IT WAS IN MY  
7 ANALYSIS.

8 Q WHAT DID YOU FIND REGARDING THE EXCESS  
9 LIFETIME CANCER RISK WHEN YOU EXCLUDED THE HARDERIAN  
10 GLAND TUMORS FROM THE ANALYSIS?

11 A WELL, IF YOU USE THE N.T.P. RAT, THE RISK  
12 PROJECTIONS WHICH I DID, BECAUSE IT WAS NOW THE MOST  
13 SENSITIVE STUDY, THEN FOR ONE CUP OF COFFEE PER DAY, THE  
14 EXCESS RISK WAS 1.6 PER 100,000 EXCESS LIFETIME CANCERS.  
15 IF YOU USE THE AVERAGE, THE AVERAGE OF 3.1 CUPS OF COFFEE  
16 PER DAY, WHICH IS WHAT THE AVERAGE DRINKER DRINKS  
17 ACCORDING TO MY ESTIMATES, THEN THE RISK IS FIVE EXCESS  
18 CANCERS PER 100,000 PER YEAR -- PER LIFETIME.

19 Q DOES THAT CONCLUDE YOUR QUANTITATIVE CANCER  
20 RISK ASSESSMENT OF THE EXCESS CANCER RISK FROM ACRYLAMIDE  
21 IN COFFEE?

22 A YES, IT DOES.

23 Q ALL RIGHT. THANK YOU, DR. BAYARD.

24 NOW, CHANGING TOPICS IF WE MAY. DID I  
25 INFORM YOU THAT THE DEFENDANTS IN THIS CASE WERE  
26 CONTENDING THAT THERE IS NO EXCESS RISK OF CANCER FROM --  
27 FOR ANY CANCER FROM CONSUMPTION OF COFFEE?

28 A YES, YOU DID.

1 Q DID I ASK YOU TO DO A QUANTITATIVE RISK  
2 ASSESSMENT TO DETERMINE WHETHER THERE WAS AN EXCESS RISK  
3 OF CANCER, SPECIFICALLY CHILDHOOD LEUKEMIA, FROM MATERNAL  
4 CONSUMPTION OF COFFEE DURING PREGNANCY?

5 A YES, YOU DID.

6 Q DID YOU DO SUCH AN ANALYSIS?

7 A I DID A QUANTITATIVE RISK ASSESSMENT.

8 Q WAS THAT QUANTITATIVE RISK ASSESSMENT FOR  
9 CONSUMPTION OF COFFEE AS OPPOSED TO ACRYLAMIDE IN COFFEE?

10 A YES, IT WAS.

11 Q DID YOU DO THAT BASED UPON MY REQUEST FOR  
12 YOU TO ASSUME A CANCER RISK FROM COFFEE?

13 A YES.

14 Q ALL RIGHT. NOW, WOULD YOU TELL US -- FIRST  
15 OF ALL, TO DO THAT ANALYSIS, DID YOU SELECT -- HOW DID  
16 YOU GO ABOUT SELECTING A STUDY TO SERVE AS THE BASIS FOR  
17 THAT ANALYSIS?

18 A I MOSTLY RELIED ON THE STATEMENT TO ME BY  
19 DR. SMITH, DR. MARTIN SMITH, THAT THE BONAVENTURE STUDY  
20 WAS A GOOD STUDY TO USE FOR THE QUANTITATIVE RISK  
21 ASSESSMENT.

22 Q ALL RIGHT. SO YOU USED THE BONAVENTURE 2013  
23 STUDY?

24 A YES.

25 Q THAT WAS AN EPIDEMIOLOGIC STUDY; CORRECT?

26 A YES.

27 Q SO FOR THIS ANALYSIS OF THE EXCESS RISK OF  
28 CONSUMPTION -- EXCESS RISK OF CHILDHOOD LEUKEMIA FROM

1 MATERNAL CONSUMPTION OF COFFEE, YOU BASED THAT ON HUMAN  
2 DATA?

3 A YES.

4 Q AND WHY WERE YOU ABLE TO BASE THAT ANALYSIS  
5 ON HUMAN DATA RATHER THAN ANIMAL DATA?

6 A BECAUSE I ASSUMED THAT COFFEE WAS A  
7 CARCINOGEN SOLELY FOR THE PURPOSE OF THIS ANALYSIS, AND  
8 BECAUSE YOU TOLD ME TO.

9 Q DID YOU -- WERE YOU ABLE TO DERIVE A SLOPE  
10 FACTOR BASED UPON THE HUMAN EPIDEMIOLOGIC DATA?

11 MR. SCHURZ: YOUR HONOR, WE ARE GOING TO OBJECT AND  
12 MOVE TO STRIKE. WHAT THE WITNESS HAS JUST TESTIFIED IS  
13 THAT HE HAS MADE NO DETERMINATION WITH RESPECT TO WHETHER  
14 COFFEE -- MATERNAL CONSUMPTION OF COFFEE RESULTS IN AN  
15 INCREASED RISK OF CANCER. WE HAVE JUST BEEN FURTHER TOLD  
16 THAT HE WAS DIRECTED TO DO THIS BY COUNSEL. WE HAVE BEEN  
17 FURTHER TOLD THAT HE WAS TOLD TO ASSUME THAT COFFEE WAS A  
18 CARCINOGEN, AND JUST RUN A NUMBER. THAT IS NOT A  
19 PREDICATE FOR ANY SORT OF RELIABLE EXPERT TESTIMONY. WE  
20 WOULD MOVE TO STRIKE AS LACKS FOUNDATION AS TO ANY OF  
21 DR. BAYARD'S CALCULATIONS OF THE RESULTS HERE.

22 THE COURT: OBJECTION OVERRULED. YOU MAY  
23 CROSS-EXAMINE.

24 Q BY MR. METZGER: ALL RIGHT. SO DID YOU NEED  
25 TO DERIVE A SLOPE FACTOR TO DO THE ANALYSIS OR A  
26 DOSE-RESPONSE?

27 A IT WAS VERY SIMILAR TO A DOSE-RESPONSE  
28 EXCEPT THAT I ONLY USED ONE DOSE.

1 Q TELL US HOW YOU WENT ABOUT DOING THIS  
2 ANALYSIS TO DETERMINE WHAT THE CANCER RISK FOR CHILDHOOD  
3 LEUKEMIA WAS FOR MATERNAL CONSUMPTION OF COFFEE DURING  
4 PREGNANCY?

5 MR. SCHURZ: OBJECTION; LACKS FOUNDATION.

6 THE COURT: OVERRULED.

7 THE WITNESS: OKAY, FOR THEIR STUDY ON CHILDHOOD  
8 LEUKEMIA DUE TO MATERNAL DRINKING OF COFFEE, IT IS  
9 PROBABLY BETTER IF WE CAN SHOW THE TABLE, THE BONAVENTURE  
10 TABLE SO THAT I CAN EXPLAIN HOW I DID THE RISK ESTIMATE.

11 Q BY MR. METZGER: I BELIEVE THAT THAT IS  
12 SLIDE 143.

13 IS THAT WHAT YOU ARE REFERRING TO?

14 A YES, SIR.

15 Q SO THIS IS TABLE 2 TAKEN DIRECTLY FROM THE  
16 BONAVENTURE STUDY. THE TABLE TITLED, "ASSOCIATIONS  
17 BETWEEN CHILDHOOD LEUKEMIA AND SELF-REPORTED MATERNAL  
18 CONSUMPTION OF CAFFEINATED ALCOHOLIC BEVERAGES DURING  
19 PREGNANCY."

20 MR. SCHURZ: OBJECTION; LACKS FOUNDATION. THIS IS  
21 NOT TABLE NO. 2. IT IS A FRACTION OF TABLE NO. 2.

22 THE COURT: ALL RIGHT, YOU MAY CROSS-EXAMINE HIM ON  
23 THAT.

24 Q BY MR. METZGER: SO THIS THE DATA THAT YOU  
25 ARE REFERRING TO, DR. BAYARD?

26 A YES, IT IS.

27 Q ALL RIGHT. THIS IS SPECIFICALLY THE DATA  
28 FROM THAT TABLE REGARDING COFFEE CONSUMPTION DURING

1 PREGNANCY; CORRECT?

2 A ONLY THE POPULATING TO COFFEE CONSUMPTION,  
3 THAT'S CORRECT.

4 Q HOW DID YOU USE THIS DATA FOR YOUR ANALYSIS?

5 A WELL, I FIRST SAW THAT IN THE FIRST SET  
6 WHERE WE ARE TALKING ABOUT ALL ACUTE LEUKEMIA, THAT IS  
7 "ALL A.L.," THE CATEGORIES WERE "REGULAR," "NEVER, NEVER"  
8 VERSUS "EVER" TYPICALLY. AND SO THERE WERE  
9 NEVER-DRINKERS, AND THESE WERE MOTHERS WHO NEVER DRANK  
10 AND THEIR CHILDREN HAD -- COMPARED TO THE CHILDREN WHO  
11 HAD CHILDHOOD LEUKEMIA. OKAY?

12 Q OKAY.

13 A AND SO THE NEVER-DRINKERS SERVED AS A  
14 CONTROL OR A REFERENCE GROUP. AND THE REGULAR COFFEE  
15 DRINKERS THEN SERVED AS THE EXPOSURE GROUP. AND THE  
16 EXPOSURE GROUP HAD A RELATIVE RISK OR AN ODDS RATIO OF  
17 1.2 COMPARED TO THE NEVER-EXPOSED. NEVER-DRINKERS.  
18 OKAY?

19 Q HOW IS REGULAR EXPOSURE DEFINED IN THIS  
20 TABLE?

21 A AT LEAST ONE CUP PER WEEK. THEN IT WAS  
22 FURTHER SUBDIVIDED TO LESS THAN ONE CUP PER DAY, ONE TO  
23 TWO CUPS PER DAY, AND GREATER THAN TWO CUPS PER DAY.

24 SO OF THE 1008 CONTROLS WHO WERE REGULAR  
25 COFFEE DRINKERS, 503, 259 AND 246 CONSTITUTED THE THREE  
26 CATEGORIES OF LESS THAN ONE CUP, ONE TO TWO CUPS, AND  
27 GREATER THAN TWO CUPS PER DAY.

28 THE ODDS RATIOS FOR THOSE WERE 1.0 FOR THE

1 LESS THAN ONE CUP PER DAY, 1.3 FOR THE ONE TO TWO CUPS  
2 PER DAY, AND 1.6 FOR THE GREATER THAN TWO CUPS PER DAY.

3 I HAD TO ASSUME WHAT WAS A REASONABLE AMOUNT  
4 OF COFFEE DRINKING FOR PREGNANT WOMEN. I ASSUMED THAT IT  
5 WOULD BE ONE TO TWO CUPS PER DAY AMONG THE AVERAGE  
6 DRINKERS. THE ODDS RATIO FOR THE ONE TO TWO CUPS PER DAY  
7 VERSUS THE CONTROLS WERE 1.3.

8 Q OKAY.

9 A WELL, TO ESTIMATE THE INCREASED RISK OF  
10 CHILDHOOD LEUKEMIA, ONE NEEDS TO KNOW THE ODDS RATIO AND  
11 ESTIMATE THE BACKGROUND RATE FOR LEUKEMIA IN THE U.S.

12 OKAY?

13 SO THE ODDS RATIO IS GOING TO BE A SURROGATE  
14 FOR THE RELATIVE RISK. IT IS FAIRLY CLOSE. YOU MULTIPLY  
15 THE INCREASED RISK, WHICH IS .3, TIMES THE BACKGROUND  
16 RATE.

17 Q HOW DID YOU DETERMINE THE BACKGROUND RATE OF  
18 LEUKEMIA, CHILDHOOD LEUKEMIA?

19 A I LOOKED FOR THE UNITED STATES STATISTICS ON  
20 CHILDHOOD LEUKEMIAS.

21 Q WHERE DID YOU FIND THOSE STATISTICS?

22 A THE NATIONAL CANCER INSTITUTE MAINTAINS A  
23 DATABASE OF INCIDENCE OF CHILDHOOD LEUKEMIAS FROM 13  
24 DIFFERENT SITES IN THIS COUNTRY, AREAS INCLUDING  
25 SAN FRANCISCO AND LOS ANGELES, I BELIEVE.

26 Q OKAY.

27 A AND THOSE -- THEY CALCULATE THOSE SITES  
28 IN -- THEY CALCULATE THE INCIDENCE RATES IN -- IT IS WHAT

1 IS CALLED A STATISTICAL EPIDEMIOLOGY END RESULTS, OR  
2 STATISTICS AND EPIDEMIOLOGY END RESULTS SECTION. THAT IS  
3 CALLED THE SEER RESULTS.

4 SO THESE TABLES ARE IN THE N.C.I. DATABASE  
5 AND THEY ARE ON THE WEB. IN MY REPORT, I SHOW WHERE ONE  
6 CAN DERIVE -- GET THESE TABLES FOR BACKGROUND RATES.

7 SO I CALCULATED THE -- GO AHEAD.

8 Q WHAT DID YOU DETERMINE WAS THE BACKGROUND  
9 RATE FOR CHILDHOOD LEUKEMIA?

10 A BACKGROUND RATE FOR CHILDHOOD LEUKEMIA IS --  
11 I CAN GO THROUGH ALL THE CALCULATIONS, BUT THE BACKGROUND  
12 RATE FOR CHILDHOOD LEUKEMIA IS 73 PER 100,000 IN THIS  
13 COUNTRY.

14 Q 73 CASES OF CHILDHOOD LEUKEMIA PER 100,000?

15 A PER 100,000, YES.

16 Q AND WELL, I ACTUALLY WOULD LIKE TO KNOW HOW  
17 YOU DETERMINED THAT. CAN YOU TAKE US THROUGH THAT?

18 A YES. I LOOKED UP THE -- I LOOKED UP THE  
19 AGE-SPECIFIC INCIDENCE RATES FOR CHILDHOOD LEUKEMIA FOR  
20 ACUTE LYMPHOBLASTIC LEUKEMIA AND ACUTE MYELOBLASTIC  
21 LEUKEMIA. I -- THE INCIDENCE RATES IN THESE TABLES,  
22 TABLE 13.12 IN THE N.C.I. TABLES AND 13.13, THEY ARE  
23 BROKEN DOWN FOR AGE GROUPS LESS THAN ONE, ONE TO FOUR,  
24 FIVE TO NINE, AND 10 TO 14.

25 SO IN ORDER TO CALCULATE THE INCIDENCE  
26 RATES, YOU TAKE THE AGE-SPECIFIC INCIDENCE RATE FOR AGE  
27 LESS THAN ONE, IT WAS 3.5. FOR AGES ONE TO FOUR, IT WAS  
28 8.9 PER 100,000. FOR AGES FIVE TO NINE, IT WAS FIVE PER

1 100,000. AND FOR AGES 10 TO 14, IT WAS FIVE -- I'M  
2 SORRY. I'M SORRY. I GOT THAT WRONG.

3 FOR AGES LESS THAN ONE, THE INCIDENCE RATE  
4 FOR CHILDHOOD LEUKEMIA, INFANT LEUKEMIA WAS 3.5. FOR  
5 AGES ONE TO FIVE, THE AGE-SPECIFIC INCIDENCE RATES WERE  
6 8.9 PER 100,000. FOR AGES FIVE TO NINE, THE AGE  
7 SPECIFIC-INCIDENCE RATES WAS 4.1 PER 100,000. AND FOR  
8 AGE 10 TO 14, THE AGE-SPECIFIC INCIDENCE RATES IS 2.7 PER  
9 100,000. BY MULTIPLYING THE NUMBER OF YEARS TIMES THE  
10 AGE-SPECIFIC RATES, AGE-SPECIFIC INCIDENCE RATES FOR EACH  
11 AGE GROUP AND SUMMING THE FOUR DIFFERENT AGE GROUPS  
12 REPORTED ON, ONE GETS AN ESTIMATE FOR TOTAL AGE-SPECIFIC  
13 INCIDENCE OF CHILDHOOD LEUKEMIA, TOTAL INCIDENCE OF  
14 CHILDHOOD LEUKEMIA OF 73 PER 100,000.

15 Q WOULD YOU GO TO SLIDE 147.

16 DOES THIS REPRESENT THE MATH THAT YOU DID?

17 A THAT IS IT.

18 Q ONE TIMES 3.5, PLUS 4 TIMES 8.9, PLUS 5  
19 TIMES 4.1, PLUS 5 TIMES 2.7 EQUALS 73 PER 100,000?

20 A THAT'S CORRECT. I ALSO CHECKED THESE  
21 RESULTS, BY THE WAY, USING A LIFE TABLE ANALYSIS PROGRAM  
22 AND I CAME UP WITH THE SAME RESULTS.

23 Q SO YOU WERE CONFIDENT THAT THIS WAS THE  
24 ACCURATE NUMBER, THE CORRECT NUMBER?

25 A YES, FOR BACKGROUND RATE.

26 Q SO NOW THAT YOU HAD THE BACKGROUND RATE FOR  
27 CHILDHOOD ACUTE LEUKEMIA IN THE UNITED STATES, WHAT DID  
28 YOU DO WITH THAT?

1           A           WELL, I WOULD LIKE TO JUST MULTIPLY IT BY  
2 1.3, BUT YOU CAN'T.

3           Q           WHY NOT?

4           A           BECAUSE SOME OF THE BACKGROUND RATE I HAVE  
5 TO ASSUME IS CAUSED BY MOTHERS DRINKING COFFEE. SO YOU  
6 HAVE TO DETERMINE WHAT THE RISK -- WHAT THE BACKGROUND  
7 RATE WOULD BE FOR THE MOTHERS WHO DID NOT DRINK COFFEE.

8           Q           OKAY.

9           A           THAT IS A SIMPLE ALGEBRA I PROBLEM.

10          Q           TELL US THAT.

11          A           WELL, THAT WOULD BE IN THE NEXT SLIDE.

12          Q           OKAY.

13          A           NO, IT IS NOT IN THAT SLIDE. NO.

14                    NOW, IN ORDER TO -- SO YOU HAVE TO GET THE  
15 RISK TO PEOPLE WHO ARE EXPOSED AND THE RISK TO PEOPLE WHO  
16 AREN'T EXPOSED, AND YOU HAVE TO GET THE -- AND YOU HAVE  
17 TO KNOW THE PROPORTION EXPOSED AND THE PROPORTION OF  
18 MOTHERS NOT EXPOSED. IN OTHER WORDS, YOU HAVE TO KNOW  
19 THE PROPORTION OF CALIFORNIANS WHO DRINK COFFEE; OKAY?

20                    FROM OUR SURVEY, N.C.A. SURVEY, WE -- I  
21 ESTIMATED THAT 65 PERCENT OF CALIFORNIANS DRINK COFFEE.  
22 I ASSUMED THAT THAT AVERAGE WAS ONE TO TWO PER DAY FOR  
23 PREGNANT MOTHERS. SO 35 PERCENT DID NOT DRINK COFFEE.

24                    SO THE CALCULATION BECOMES, AND I THINK YOU  
25 FIND IT IN THE TABLE BEFORE THIS, OR THE TABLE  
26 AFTERWARDS.

27          Q           OKAY. SLIDE 149.

28          A           149.

1                   SO THE CALCULATION IS 35 PERCENT TIMES THE  
2 RISK OF NO COFFEE, PLUS 65 PERCENT, WHICH IS THE RISK OF  
3 1.3, THE RISK OF NO COFFEE, SO 65 PERCENT OF THE  
4 POPULATION HAVE THE INCREASED RISK OF 1.3, AND 35 PERCENT  
5 OF THE POPULATION DON'T HAVE THE INCREASED RISK. THAT --  
6 THOSE TWO SUBPOPULATIONS COMPRISE THE GROUP THAT CAUSES A  
7 73 PER 100,000 RISK. THEN IT IS PRETTY EASY TO CALCULATE  
8 THAT THE BACKGROUND RISK OF CANCER FROM NO COFFEE IS  
9 .00065 OR 65 PER 100,000, AND THEN TO ESTIMATE THE  
10 INCREASED RISK, WHICH IS THE RELATIVE RISK MINUS ONE  
11 TIMES THE UNEXPOSED GROUP, AS .3 TIMES 65 PER 100,000, OR  
12 19.5 PER 100,000. SO THAT IS THE WAY YOU DO THE  
13 CALCULATION.

14                   SO WHAT YOU CALCULATE IS -- I'M SORRY, I AM  
15 FINISHED WITH MY ANSWER.

16           Q           SO WHAT DID YOU CONCLUDE -- ASSUMING THAT  
17 THE ASSOCIATION BETWEEN MATERNAL CONSUMPTION OF COFFEE  
18 AND CHILDHOOD LEUKEMIA IS GENUINE, WHAT DID YOU CONCLUDE  
19 WAS THE EXCESS RISK OF CANCER PER 100,000 CHILDREN FROM  
20 MATERNAL CONSUMPTION OF COFFEE DURING PREGNANCY?

21           A           FOR MATERNAL CONSUMPTION OF COFFEE DURING  
22 PREGNANCY OF ONE TO TWO CUPS PER DAY, I CONCLUDED THAT  
23 THE INCREASED CANCER RISK WAS 19.5 PER 100,000 UNDER THE  
24 ASSUMPTIONS THAT YOU JUST STATED.

25           Q           SO THAT IS 19.5 TIMES WHAT IS ALLOWED?

26           A           THAT IS NOT MY -- THAT IS NOT MY BAILIWICK,  
27 WHAT IS ALLOWED AND WHAT IS NOT.

28           Q           19.5 TIMES THE STANDARD OF ONE EXCESS CANCER

1 PER 100,000?

2 A THAT CALIFORNIA USES.

3 Q ALL RIGHT. NOW, YOU RELIED ON THE  
4 BONAVENTURE STUDY FOR THIS AND THE SEER DATA; CORRECT?

5 A YES.

6 Q WERE THERE ANY OTHER DATA SOURCES THAT YOU  
7 RELIED ON FOR THIS ANALYSIS?

8 A NO, I DID LOOK AT THE CHENG ANALYSIS AND HIS  
9 META-ANALYSIS, AND I HAVE CONFIDENCE IN META-ANALYSES, SO  
10 I WAS ABLE TO LOOK AT HIS -- THE SEVEN STUDIES THAT HE  
11 ANALYZED TO MAKE THE CONCLUSION THAT THERE WAS A HIGH  
12 ASSOCIATION BETWEEN MATERNAL COFFEE DRINKING AND  
13 CHILDHOOD LEUKEMIA.

14 SO FROM THE CHENG STUDY, I EXTRACTED THE  
15 BONAVENTURE STUDY AS THE STUDY WHICH PROVIDED THE MOST  
16 CASES AND THE MOST WEIGHT TO HIS META-ANALYSIS.

17 Q NOW, ARE YOU AN EPIDEMIOLOGIST?

18 A NO.

19 Q ARE YOU AN EXPERT SPECIFICALLY IN THE  
20 MECHANISMS OF CHILDHOOD LEUKEMIA?

21 A NO. NO.

22 Q ARE YOU AN EXPERT IN THE CAUSES OF CHILDHOOD  
23 LEUKEMIA?

24 A NO.

25 Q SO WHAT YOU WERE DOING WAS THE CALCULATION;  
26 IS THAT CORRECT?

27 A I WAS DOING THE QUANTITATIVE RISK  
28 ASSESSMENT.

1 Q AND WERE YOU LEAVING TO THE OTHER EXPERTS IN  
2 THIS CASE TO DETERMINE WHETHER THE EPIDEMIOLOGY SUPPORTS  
3 THIS AND WHETHER THE -- THERE IS A BIOLOGICALLY-PLAUSIBLE  
4 MECHANISM FOR MATERNAL CONSUMPTION OF COFFEE DURING  
5 PREGNANCY CAUSING CHILDHOOD LEUKEMIA?

6 A YES.

7 Q SO DID YOU DO THIS ANALYSIS PURELY BASED  
8 UPON MY REQUEST TO YOU BASICALLY TO DO AN ANALYSIS OF  
9 WHETHER THERE WAS A CANCER RISK BASED UPON THE  
10 DEFENDANT'S VIEW OF THE CASE?

11 A I DID THIS PURELY ON YOUR REQUEST.

12 Q ALL RIGHT. DOES THAT CONCLUDE YOUR  
13 QUANTITATIVE RISK ASSESSMENT OF THE RISK OF CHILDHOOD  
14 LEUKEMIA FROM MATERNAL CONSUMPTION OF COFFEE DURING  
15 PREGNANCY, DR. BAYARD?

16 A YES, IT DOES.

17 MR. METZGER: THANK YOU VERY MUCH. I HAVE NO  
18 FURTHER QUESTIONS.

19 THE COURT: ALL RIGHT. WHY DON'T -- MR. SCHURZ, DO  
20 YOU WANT TO GET STARTED TODAY?

21 MR. SCHURZ: WHATEVER YOU WOULD LIKE, YOUR HONOR.

22 THE COURT: ALL RIGHT. LET'S GO FOR FIVE, TEN  
23 MINUTES.

24 MR. SCHURZ: OKAY.

25

26 CROSS-EXAMINATION

27 BY MR. SCHURZ:

28 Q GOOD AFTERNOON, DR. BAYARD.

1           A           GOOD AFTERNOON, MR. SCHURZ.

2           Q           NOW, I WOULD LIKE TO PICK UP WITH THE  
3 DISCUSSION THAT YOU WERE HAVING WITH MR. METZGER RELATING  
4 TO THE CHILDHOOD LEUKEMIA QUANTITATIVE RISK ASSESSMENT  
5 THAT YOU PERFORMED. NOW, IN THIS CASE, FOR THE  
6 PERFORMING A QUANTITATIVE RISK ASSESSMENT OF COFFEE, YOU  
7 CHOSE TO DO A MIXTURE ANALYSIS; CORRECT?

8           A           I CHOSE TO DO -- THE MIXTURE BEING COFFEE?

9           Q           YES.

10          A           THAT'S CORRECT. I DID NOT CHOOSE IT, I WAS  
11 ASKED TO DO IT.

12          Q           AND YOU LOOKED AT THE DATA ON COFFEE  
13 CONSUMPTION TO PERFORM YOUR RISK ASSESSMENT REGARDING  
14 CHILDHOOD LEUKEMIA; CORRECT?

15          A           YES.

16          Q           YOU LOOKED AT THE BONAVENTURE STUDY AND YOU  
17 LOOKED AT THE CHENG STUDY; CORRECT?

18          A           YES.

19          Q           YOU ARE FAMILIAR WITH PERFORMING RISK  
20 ASSESSMENTS FOR CHEMICAL MIXTURES; ARE YOU NOT?

21          A           YES.

22          Q           BECAUSE YOU WERE THE PROJECT MANAGER FOR THE  
23 OFFICE OF HEALTH AND ENVIRONMENTAL ASSESSMENT RISK  
24 ASSESSMENT FOR SECONDHAND SMOKE; CORRECT?

25          A           YES.

26          Q           AND WERE YOU THE CO-EDITOR OF THAT DOCUMENT;  
27 CORRECT?

28          A           THE DOCUMENT WAS THE NATIONAL CANCER

1 INSTITUTE DOCUMENT.

2 Q AND WITH RESPECT TO THE RESPIRATORY HEALTH  
3 EFFECTS OF PASSIVE SMOKING, YOU HAD OVERALL  
4 RESPONSIBILITY FOR THE CONTENTS OF THE REPORT AND ITS  
5 CONCLUSIONS; CORRECT?

6 A THAT'S CORRECT.

7 Q NOW, IN THIS CASE, DR. BAYARD, WOULD YOU  
8 AGREE THAT COFFEE IS A COMPLEX MIXTURE?

9 A YES.

10 Q AND WOULD YOU FURTHER AGREE THAT UNDER  
11 E.P.A.'S GUIDELINES FOR A HEALTH RISK ASSESSMENT OF  
12 CHEMICAL MIXTURES, A DOCUMENT YOU DISCUSSED WITH  
13 MR. METZGER, THAT THE DIRECTION FROM E.P.A. IS THAT  
14 WHENEVER POSSIBLE, THE PREFERRED APPROACH TO THE HEALTH  
15 RISK EVALUATION OF CHEMICAL MIXTURES IS TO PERFORM THE  
16 ASSESSMENT USING HEALTH AND EXPOSURE DATA ON THE WHOLE  
17 MIXTURE?

18 A WHICH ASSESSMENT DO YOU MEAN?

19 Q I AM TALKING ABOUT A RISK ASSESSMENT WITH  
20 RESPECT TO BEING PRESENTED AS TO A WHOLE MIXTURE, THAT  
21 THE PREFERENCE IS TO DO AN ANALYSIS ON THE WHOLE MIXTURE  
22 WHENEVER POSSIBLE.

23 A ARE YOU TALKING ABOUT A HAZARD ASSESSMENT?

24 A DOSE-RESPONSE ASSESSMENT?

25 Q I AM TALKING ABOUT A RISK ASSESSMENT, AND  
26 PARTICULARLY AT THE HAZARD IDENTIFICATION STAGE, BUT ALL  
27 FOUR STAGES.

28 MR. METZGER: WELL, I THINK I NEED TO OBJECT. IT

1 IS VAGUE AS TO WHETHER IT IS QUALITATIVE RISK ASSESSMENT  
2 OR QUANTITATIVE THAT HE IS ASKING.

3 THE COURT: ALL RIGHT. PLEASE REPHRASE THE  
4 QUESTION.

5 Q BY MR. SCHURZ: IS IT THE CASE, DR. BAYARD,  
6 THAT E.P.A.'S GUIDELINES FOR HEALTH RISK ASSESSMENT FOR  
7 CHEMICAL MIXTURES ADVISES THAT WHENEVER POSSIBLE, THE  
8 PREFERRED APPROACH TO THE HEALTH RISK EVALUATION OF  
9 CHEMICAL MIXTURES IS TO PERFORM AN ASSESSMENT OF THE  
10 MIXTURE ITSELF?

11 MR. METZGER: SAME OBJECTIONS.

12 THE COURT: OVERRULED.

13 THE WITNESS: WOULD YOU SHOW ME THE STATEMENT,  
14 PLEASE.

15 Q BY MR. SCHURZ: ARE YOU FAMILIAR WITH THE  
16 CHEMICAL MIXTURES GUIDELINES?

17 A I AM FAMILIAR WITH IT, BUT I -- IT IS  
18 SEVERAL HUNDRED PAGES. IF YOU PULL SOMETHING OUT OF  
19 CONTEXT, I THINK I HAVE -- I WOULD LIKE TO SEE WHERE YOU  
20 ARE MAKING YOUR STATEMENT FROM.

21 Q ARE YOU AWARE OF WHETHER E.P.A.'S  
22 SUPPLEMENTARY GUIDANCE FOR CONDUCTING HEALTH RISK  
23 ASSESSMENT OF CHEMICAL MIXTURES HAS A PREFERENCE FOR  
24 CONDUCTING ASSESSMENTS OF CHEMICAL MIXTURES ON THE  
25 MIXTURE ITSELF WHENEVER POSSIBLE?

26 A THE WAY I READ IT, IT IS NOT WHENEVER  
27 POSSIBLE. IT IS THEY DO NOT MAKE A CHOICE BETWEEN DOING  
28 AN ASSESSMENT AS A MIXTURE OR A -- OR BY CONSTITUENTS

1 WITHIN THE MIXTURE. THAT IS WHY I ASKED YOU IF YOU WOULD  
2 SHOW ME THE STATEMENT.

3 Q OF COURSE. TAKE A LOOK, IF YOU WOULD, AT  
4 DX-10255, WHICH YOU DISCUSSED WITH MR. METZGER EARLIER  
5 TODAY.

6 I DIRECT YOUR ATTENTION TO PAGE 0038,  
7 SECTION 2.5.

8 DO YOU HAVE THAT IN FRONT OF YOU?

9 MR. METZGER: COULD YOU WAIT JUST A MOMENT UNTIL I  
10 GET THE DOCUMENT, COUNSEL.

11 WHAT PAGE ARE YOU AT?

12 MR. SCHURZ: 00038. AS IT INDICATES ON THE  
13 MONITOR.

14 MR. METZGER: THANK YOU.

15 THE WITNESS: COULD YOU PLEASE HELP ME.

16 Q BY MR. SCHURZ: IF YOU GO TO THE VERY BOTTOM  
17 OF THE PAGE, DR. BAYARD, YOU WILL SEE A STAMP THAT SAYS  
18 "DX-10255."

19 A YES.

20 Q IF YOU GO TO 0038, IT IS ALSO PAGE 23 IN THE  
21 PAGINATION OF THE DOCUMENT, THE BATES NUMBER THAT  
22 APPEARS IS 0038.

23 A I UNDERSTAND.

24 THE WAY I READ THIS, IT TALKS ABOUT DOING  
25 THE EXPOSURE ASSESSMENT AND THE QUALITATIVE RISK  
26 ASSESSMENT FIRST. IT IS A LITTLE BIT MORE VAGUE TO ME ON  
27 WHETHER OR NOT TO DO THE QUANTITATIVE RISK ASSESSMENT ON  
28 THE WHOLE MIXTURE.

1 Q WHAT THE CHEMICAL -- EXCUSE ME, THE CHEMICAL  
2 MIXTURE GUIDANCE DOCUMENT FURTHER INDICATES IS THAT ONE  
3 IS TO DO AN ANALYSIS OF THE HUMAN EPIDEMIOLOGIC, CLINIC  
4 OR OCCUPATIONAL STUDIES, THE ANIMAL STUDIES ON THE  
5 COMPLEX MIXTURE, OR IN VITRO DATA ON THE COMPLEX MIXTURE;  
6 CORRECT?

7 A IT SAYS WHAT THE DATA ARE THAT YOU SHOULD  
8 ASSESS, THAT'S CORRECT.

9 Q AND THE ASSESSMENT OF THE ADEQUACY OF THE  
10 UNDERLYING DATA INFORMS WHETHER YOU PERFORM A MIXTURE  
11 ANALYSIS FOR COFFEE AS A SINGLE CHEMICAL MIXTURE, OR  
12 ALTERNATIVELY, WHETHER ONE DOES EITHER A CONSTITUENT  
13 ANALYSIS OR YOU DO AN ANALYSIS OF A SIMILAR OR  
14 SUFFICIENTLY SIMILAR MIXTURE; CORRECT?

15 A THE WAY I READ THIS IS IF YOU CAN DO AN  
16 ANALYSIS ON THE MIXTURE, DO IT, BUT THERE ARE OTHER WAYS  
17 OF DOING IT ALSO. I GET THAT ACTUALLY FROM THE FIRST  
18 PART OF THE DOCUMENT, NOT FROM THE PART THAT YOU POINTED  
19 OUT.

20 IF A MIXTURE IS CARCINOGENIC, I WOULD HAVE  
21 NO TROUBLE DOING IT ON THE MIXTURE.

22 Q ALL RIGHT. BUT I WANT TO FOCUS FIRST ON THE  
23 DETERMINATION AS TO WHETHER ONE DOES A MIXTURE ANALYSIS  
24 OR WHETHER ONE DOES A CONSTITUENT ANALYSIS AS YOU HAVE  
25 DONE HERE; IS THAT CORRECT?

26 A IT IS CORRECT THAT YOU WANT TO FOCUS ON  
27 THIS. THAT IS CORRECT.

28 Q ALL RIGHT.

1 THE COURT: WE WILL STOP AT THIS TIME. THAT WAS  
2 THE SIGN.

3 WE HAVE TO RECESS FOR THE DAY. WE WILL  
4 RESUME THE TRIAL TOMORROW MORNING AT 9:00 O'CLOCK.

5 DR. BAYARD, YOU ARE ORDERED TO RETURN  
6 TOMORROW MORNING AT 9:00 O'CLOCK.

7 MR. METZGER: YOUR HONOR, I OWE YOU AN APOLOGY.  
8 ONCE AGAIN, I OVERESTIMATED MY TIME.

9 THE COURT: THANK YOU.

10 HOW LONG ARE YOU GOING TO BE ON  
11 CROSS-EXAMINATION?

12 MR. SCHURZ: CONSISTENT WITH OUR EARLIER ESTIMATE,  
13 YOUR HONOR, WE THINK A HALF A DAY.

14 THE COURT: WE WILL RECESS UNTIL TOMORROW MORNING.

15 MR. METZGER: THANK YOU, YOUR HONOR.

16

17 (THE MATTER WAS ADJOURNED AT 4:19 P.M.)

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**EXHIBIT “C”**

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## EDUCATION

B.S. Mathematics, Tufts University, 1965. Minors - Biology, Chemistry.  
Ph.D. Biostatistics, Johns Hopkins University, 1971. Minor - Biochemistry.  
30 Graduate Credits, Toxicology, Mass. Institute of Technology, 1979, Non Degree program.

## AREAS OF EXPERTISE.

I have over 30 years of experience in health and quantitative risk assessment of toxic chemical hazards. This experience includes work both as a private consultant and employee of the U.S. Government. I have experience as an employee with three Federal regulatory agencies - CPSC, EPA, and OSHA - I initiated and developed the quantitative risk assessment process for cancer risk modeling at CPSC. At EPA I authored published cancer risk assessments for over 20 chemicals, including Dioxin, asbestos, methylene chloride, nickel and nickel compounds, 1,3-butadiene, vinyl and vinylidene chloride, ethylene oxide, and cadmium. At OSHA, I authored a risk assessment for tuberculosis infection and contraction of active disease due to U.S. worker exposure to the TB bacillus. I have more than 6 years of U.S. Government supervising experience of scientific experts in risk assessment. My teaching experience includes graduate courses in statistics, biostatistics, epidemiology and demography. International experience includes teaching risk assessment for the Pan American Health Organization (1988), and co-authorship of a World Health Organization report on health hazards of environmental tobacco smoke. Referee for several journals for articles submitted for publication in these fields. I also have graduate training in toxicology and biochemistry.

I also have extensive experience in contract and project management. Since retiring from the U.S. OSHA in 2007, as a consultant I have managed U.S.CDC contracts designing, conducting and analyzing studies on Iowa private well water quality, and on Illinois air pollution and respiratory health. Prior to that, at OSHA I managed OSHA's Technical Data Center and Library operations. Also, at OSHA, I was Director of OSHA's Office of Risk Assessment. While at EPA I was project manager and co-author of EPA's 1992 report on the "Health Effects of Passive Smoking: Lung Cancer and Other Disorders." I provided the statistical risk analyses for children's respiratory effects and authored the chapter on "Assessment of Increased Risk for Respiratory Illnesses in Children from Environmental Tobacco Smoke." I was also a scientific editor for that report, as well as for the NCI Monograph 4 on the same topic in NCI's Smoking and Tobacco Control series. I also worked with EPA's Indoor Air Division to develop EPA smoking policy, control measures and outreach. The week the EPA report was released, both McDonald's and Burger King went smoke-free nationwide, as did many U.S. airports and other facilities.

## **WORK EXPERIENCE**

2016. Consultant for Statistics and Risk Assessment on Glyphosate and Roundup. Lundy, Lundy, Soileau & South. Lake Charles, La.
- 2013-2014; 2017. Consultant and Expert Witness for Statistics and Risk Assessments on Acrylamide in Coffee. Metzger Law Group, Long Beach, California.
- 2009-2010. Consultant for Statistics and Risk Assessments on Beryllium and Silica. OSHA, Washington, D.C.
- 2007-2009. Senior Statistician and Project Manager for both Water quality and Air Pollution and Health studies. Raleigh, N.C.
2007. Consultant and Expert Witness, Tobacco Litigation Case, Chicago, IL.
- 2005- 2006. Health Scientist, Directorate of Science, Technology and Medicine, U.S. Occupational Safety and Health Administration. Supervisory Health Scientist GS-601-15.
- 2003- 2004. Acting Director, Technical Data Center, Directorate of Science, Technology and Medicine, U.S. Occupational Safety and Health Administration. Information Program Manager, GS-340-15.
- 2003- 2004. Health Scientist, Directorate of Science, Technology and Medicine, U.S. Occupational Safety and Health Administration. Supervisory Health Scientist GS-601-15.
- 2002-2003. Health Scientist (Epidemiologist/Toxicologist), Directorate of Standards and Guidance,. Health Scientist GS-601-15.
- 1997-2002. Director, Office of Risk Assessment, Directorate of Health Standards Programs, U.S. Occupational Safety and Health Administration. Supervisory Health Scientist GS-601-15.
- 1995-1997. (Detail from U.S. EPA) Senior Science Advisor to the Director, Directorate of Health Standards Programs, U.S. Occupational Safety and Health Administration.
- 1979-1995. Health Statistician, Office of Research and Development, U.S. Environmental Protection Agency. GS-1530-14
- 1978-1979. Mathematical Statistician, Office of Program Planning and Evaluation, U.S. EPA. GS-1529-14
- 1974-1978. Statistician/Acting Branch Chief, Biometrics Branch, U.S. Consumer Product Safety

Commission. GS-1529-13

1970-1974. Assistant Professor of Public Health (Biometry), Yale University.  
Taught graduate level courses in biostatistics, epidemiology, and demography; thesis advisor for M.S. and Ph.D. students

## **AWARDS**

1965-1970. *U.S. Public Health Service Fellowship* for Graduate training.

1978. *Chairman's Special Achievement Award* for achievements in risk assessment, U.S. Consumer Product Safety Commission.

1985. *Bronze Medal* for participation in Carcinogen Assessment Guidelines writing team. U.S.EPA.

1991. *Peer Review Award for Scientific Achievement* for EPA's Risk Assessment of Passive Smoking.. Office of Health and Environmental Assessment, U.S. EPA.

1992. *EPA Individual Gold Medal* for Science Leadership for work as project officer and co-author of EPA's Passive Smoking Report. This is EPA's highest award.

1995. *EPA Statisticians Award* for contributions to the field of Environmental Statistics.

2001. Dept. of Labor *Secretary's Exceptional Achievement Award (Team)* for work on reinvention in the development of safety and health standards.

2001. Dept. of Labor *Secretary's Exceptional Achievement Award (Team)* for work on OSHA's Ergonomics standard.

## **Publications Since 1992.**

Bayard S.

EPA's Report on the Respiratory Health Effects of Passive Smoking.  
1993, Proceedings of Indoor Environment '93. pgs. 297-300.

Jinot J, Bayard, S.

Respiratory Health Effects of Passive Smoking: EPA's Weight-of-Evidence Analysis. 1994, Journal of Clinical Epidemiology, Vol.47, pgs. 339-349.

Farland, W.; Bayard, S.; Jinot, J.

Environmental Tobacco Smoke: A Public Health Conspiracy? A Dissenting View. 1994, *Journal of Clinical Epidemiology*, Vol. 47, No. 4, pgs. 335-337.

Bayard, S.; Jinot, J.

Environmental Tobacco Smoke: Industry's Lawsuit. *EPA Journal*. Vol. 19. No. 4. pg. 20.

Axelrad, R.; Bayard, S.; Jinot, J.

Setting the Record Straight: Secondhand Smoke is a Preventable Health Risk. *Tobacco Control*. 1994. Vol. 3, No. 2. pgs. 263-267.

Bayard S, Jinot J, Brown ,K.

Passive Smoking and Lung Cancer: The U.S. EPA's Weight-of-Evidence Analysis, With Emphasis on the Epidemiology Studies. *American Statistical Association 1994 Proceedings of the Section on Statistics and the Environment*. 1995. Pgs. 56-61.

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Environmental Tobacco Smoke and Lung Cancer: Uncertainties in the Population Estimates but not in the Causal Association. *Environmetrics*. 1995. Vol. 6, pgs. 413-418.

Jinot J, Bayard S.

Environmental Tobacco Smoke: Science vs. Rhetoric. *Risk Analysis*. 1995. Vol.15, No. 1, pgs 91-96.

Bayard S. Jinot J.

Response to Dr. Barry's Article on ETS/Regarding Mr. Wilson's Letter. Letter to the Editor. *Risk Analysis*. 1996. Vol.16, No.3, pgs. 303-304.

Jinot J, Bayard S.

Respiratory Health Effects of Exposure to ETS. *Reviews on Environmental Health*. 1997. Vol.11, No. 3.

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Air Nicotine and Saliva Cotinine as Indicators of Workplace Passive Smoking Exposure and Risk. *Risk Analysis*. 1998. Vol. 18, No. 1, pgs. 71-83.

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Toraason M, Albertini R, Bayard S, et al

Applying New Biotechnologies to the Study of Occupational Cancer. A Workshop Summary. *Env. Health Perspect*. 2004 Mar; 112(4): 413-6.

### **Invited Presentations Since 1992**

Over 20 invited presentations to scientific meetings, symposiums, and seminars including:

Indoor Environment '93, Baltimore, Md.  
8th Annual American Industrial Hygiene Association Toxicology Symposium, 1993, Asheville, N.C.  
Toxicology Forum, 1993, Aspen, Colo.  
American Public Health Association, 1993, San Francisco, Calif.  
23rd International Symposium on Environmental Analytical Chemistry, 1993, Jekyl Island, Georgia (keynote speaker)  
EPA/DOD Conference on Risk Assessment, 1993, Dayton, Ohio  
American Industrial Hygiene Conference, 1993, New Orleans, La.  
National Cancer Institute Seminar Series, 1993, Bethesda, Md.  
Governor's Cancer Summit, 1993, Baltimore, Md.  
EPA Regional Risk Assessors Annual Meeting, 1994, Boston, Mass.  
National Conference of State Legislators, 1994, New Orleans, La.  
Texas Health Department Smoke-Free Conference, 1994, Lubbock, Tex.  
American Statistical Association, 1994, Toronto, Ontario  
Environmetrics Society, 1994, Burlington, Ontario  
ASA-NISS 1994 Conference RTP, N.C.  
Cato Institute, 1994, Washington, D.C.  
Environmental Tobacco Smoke Science and Policy Conference, 1994, Marlboro, Mass.  
Wake Forrester University, 1995, Winston-Salem, N.C.  
EPA Statisticians' Conference, 1995, Williamsburg, Va.  
Harvard University, 1995, Cambridge, Massachusetts  
Society for Risk Analysis, 1997, Monterey, California  
American Cancer Society, 1998, Washington, D.C.  
Eurotox 2000, 2000. London, England.  
National Occupational Research Agenda Conference, 2002. Washington, D.C.

## **PUBLICATIONS AND PRESENTATIONS: 1971-1992.**

- Bayard SP. An Age-Time Parametric Model for Follow-up Studies. Ph.D. Thesis. Dissertation Abstracts, 1971. P.2550B.
- Bayard SP. Another Look at the Statistical Analysis of Changes during Storage of Serum Specimens. Health Laboratory Science, Jan. 1974, Vol. 11; No. 1, 45-49.
- Bayard SP. Views on the Understanding of Statistical Techniques in Laboratory Research. Health Laboratory Science, Jan. 1974.
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- Hehir RM, Bayard SP, Thompson, E.  
CPSC Regulation of Non-Occupational Exposure to Asbestos in Consumer Products Presented at the National Workshop on Asbestos. July 1977. Paper published in the *Proceedings*.
- Bayard SP. A Time to Tumor Model for Risk Assessment of Asbestos Induced Death from Respiratory Cancer. Presented at the Third Annual FDA Office of Science Symposium, Colorado Springs, Colorado. February 1978. Paper published in the *Proceedings*.
- Ehrlich AM, Bayard SP, Thompson EJ.  
Consumer Protection and the Consumer Product Safety Commission. Presented at the Society for Occupational and Environmental Health Symposium on Occupational Exposure to Fibrous and Particulate Dust. Washington, D.C., December 1977. Paper published in the *Proceedings*.
- Bayard SP. Multistage Models in Carcinogenesis. Presented at the EPA Statisticians' Conference, RTP, N.C., April 1984.
- Hiremath C, Bayliss D, Bayard S.  
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(FRG). September 1985. Paper published in Chemosphere. 1986. Vol. 15, Nos. 9-12, pp. 1815-1823.

Koppikar A, Bayard SP.

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Thorslund T, Bayard SP, Brown R.

Quantitative Model for the Tumor Promoting Activity of 2,3,7,8-TCDD. Presented at the 7th International Symposium on Chlorinated Dioxins and Related Compounds. Las Vegas, Nev., Oct. 1987.

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Bayard SP.

Pharmacokinetics in the Risk Assessment of 1,3-Butadiene. Presented at the International. Workshop on Biological Data for Pharmacokinetic Modeling and Risk Assessment. Asheville, NC, May, 1988.

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Cancer Risk Assessment of 1,3-Butadiene. Environmental Health Perspectives. 1990, Vol. 86, pages 149-153

Bayard SP, Brown K, Thorslund T.

Risk Assessment of Passive Smoking. Presented at the Biometric Society/Eastern North American Region Meeting. Houston, Texas, March, 1991.

Bayard SP. Epidemiology of Environmental Tobacco Smoke and Lung Cancer. Presented at the Organization Resource Counsellors Meeting. Washington, D.C., August, 1991.

Bayard SP. Quantitative Implications of the Use of Different Extrapolation Procedures for Low-Dose Cancer Risk Estimates from Exposure to 2,3,7,8 - TCDD. In Dioxin Perspectives. Bretthauer, Kraus and di Domanico editors. Plenum Press, 1991. pages 205-247.

## Other

Adjunct Faculty at Uniformed Services University of Health Sciences, Graduate School of Nursing. Taught Biostatistics and Epidemiology in the Ph.D. Nursing Program. 2004-2005.

EPA expert witness for Dioxin, testified in Vietnam Veteran's Agent Orange Lawsuit.

EPA expert witness for Environmental Tobacco Smoke, testified at hearings before U.S. Congress, U.S. OSHA, Maryland OSHA, State legislatures of Maryland, Virginia, and Pennsylvania, and several local jurisdictions; participated in press conferences and radio programs. 1993-1995

DOJ fact witness in the Tobacco Litigation Lawsuit. 2002.

Graduate of U.S. Office of Personnel Management's Executive Development Program, 1989.

Course in Probabilistic Risk Assessment, Harvard University, 2002. (2.5 continuing Education Units).

Course in Physiologically-Based-Pharmacokinetic Modeling, Colorado State University, 1992.

OSHA Representative to National Academy of Sciences Federal Regulatory Liaison Committee. 2003.