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

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CERT’s Submission No. 2 regarding the Quantitative Risk Assessment for Acrylamide in Coffee of Dr. Steven P. Bayard.

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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. 2

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 Acrylamide in Coffee of Dr. Steven P. Bayard:

1. Exhibit A - Statement of Steven P. Bayard (April 22, 2014).

2. Exhibit B - Addendum to Statement of Steven P. Bayard: Recalculations for Revised Exhibit #3, Corrected 5/24/2014: Cancer Risk Assessment of Acrylamide in Coffee Not Including Harderian Tumors in NTP Mice

3. Exhibit C - Statement of Steven P. Bayard - Errata to Cancer Risk Assessment of Acrylamide in Coffee (April 28, 2014)

4. Exhibit D - Addendum to Statement of Steven P. Bayard: Recalculations for Revised Exhibit #3, Corrected 5/25/2014, Cancer Risk Assessment of Acrylamide in Coffee Not Including Harderian Tumors in NTP Mice(June 2, 2014)

5. Exhibit E - Statement of Steven Bayard Regarding: Calculation of the No Significant Risk Level (NSRL) for Acrylamide; Calculations of Cancer Potency Slopes Using the Animal-to-Human Species Conversion Factor; and Number of Previous Depositions

6. Exhibit F - Corrections to April 28, 2014 Deposition: Exhibits #1 and #3 (June 12, 2014)

7. Exhibit G - Direct Testimony of Dr. Steven Bayard in *CERT v. Starbucks* (October 27, 2014 a.m.)

8. Exhibit H - Direct Testimony of Dr. Steven Bayard in *CERT v. Starbucks* (October 28, 2014 p.m.)

9. Exhibit I - 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, Ph.D.

Introduction.

I am Steven Bayard. I reside at [redacted], MD., [redacted] I am retired (2007) from a 32 year career with the U.S. Government. Nearly thirty of those years, I was working on various areas of quantitative and scientific risk assessment of toxic chemicals, for the U.S. Consumer Product Safety Commission, the U.S. Environmental Protection Agency, or the U. S. Occupational Safety and Health agency. I have a B.S. in Mathematics from Tufts University, 1965, and a Ph.D. in Biostatistics from Johns Hopkins University in 1971.

My purpose in this statement is to provide a quantitative risk analysis for the risk of cancer from drinking coffee, due to the acrylamide content in coffee. To estimate the cancer risk from acrylamide (AA) in coffee, we need to estimate three components:

I. The cancer potency per amount of acrylamide ingested on a daily basis;
II. The amount of acrylamide in coffee; and
III. The amount of coffee consumed on a daily basis.

This analysis develops estimates of these three components and then combines these to estimate the lifetime risk of cancer to the coffee drinker due to its acrylamide contaminant. Final risk estimates will be presented as both cancer risk from coffee consumption, and whether the amount of AA in coffee is above or below California's No Significant Risk Level (NSRL) for the Proposition 65 Carcinogen AA. The results of this risk assessment will also be compared with those on AA of the Cal. EPA and the U.S. EPA

I. The cancer potency of ingested acrylamide.

First, the carcinogenic potential of AA has been studied by several government and other health agencies worldwide, and, to my knowledge, has been declared to be a presumptive human carcinogen by all those who have made formal statements on their analyses. In its most basic form the logic is that AA 1) causes cancers in multiple sites in both sexes of rats and mice in multiple lifetime bioassay tests, 2) is bioavailable from ingestion, and 3) is metabolized, in animals and humans, to a bioavailable epoxide that forms DNA adducts, and is mutagenic. Although there is little direct evidence of carcinogenicity in humans, the evidence in test animals, and other tests, provide sufficient concern that humans, too, are susceptible to cancer from ingestion of acrylamide.

Two of the lead government regulatory agencies in quantitative risk assessments are the U.S. EPA and the California EPA, and both have produced comprehensive reports on AA, including estimates of the carcinogenic potency from ingestion (U.S. EPA, 2010, Plaintiff's exhibit #48631; and Cal EPA, 2005, Plaintiff's Exhibit #23930). I will use results from both these reports to provide estimates of the cancer potency of ingested AA. I will also use some of their methods in my analysis. Although their methods are quite similar and they both use the same rat drinking water studies as the basis for their quantitative estimates (Johnson et al. (1986) Plaintiff's Exhibit # 23171; and Friedman et al. (Plaintiff's Exhibit #15159), there are differences that do have an effect on their final estimates. The analysis below will present and compare the results of these two reports.

A third governmental agency whose report is important to my analysis is the National Toxicology Program, (NTP, part of the U.S. National institutes of Health), which conducts much of the animal toxicology testing. Specifically, I will be using NTP's recent report on their bioassay results for AA (NTP TR 575, Plaintiff's Exhibit #48449). I will also be referring to a recent publication by some of the study's investigators (Beland et. al., (2013), Plaintiff's Exhibit #48992) which presents the major results of that study. The NTP TR 575 provides the basic data for the dose-response part of this risk assessment, specifically the multiple tumors in both rats and mice of both sexes. These NTP results have not been reported on by either the U.S. EPA or Cal EPA, although some foreign and international agencies have done analyses on these data.

As stated, both Cal. EPA and U.S. EPA use the results from two earlier studies on rats (Johnson et. al., 1986; and Friedman et al., 1995) to perform their dose-response analyses, and these analyses will not be repeated here. Instead, my analysis will use the new NTP data and I compare those results with those from the earlier analyses. These comparisons start with calculating dose-response models for the NTP data.

I.A. Dose-Response Modelling of the NTP Rat and Mouse Cancer Data for AA.

The NTP results show statistically significant dose-related tumors in multiple sites of both sexes of the tested rats and mice. As a result of their investigation, the NTP "conclude(s) that acrylamide in the drinking water caused cancer in several different tissues in male and female rats and mice (NTP, 2012, Summary page 6). More specifically, their larger statement is shown in Figure 1, below.

CONCLUSIONS

Under the conditions of these 2-year drinking water studies, there was *clear evidence of carcinogenic activity* of acrylamide in male F344/N rats based on increased incidences of malignant mesothelioma of the epididymis and testis tunica, malignant schwannoma of the heart, and follicular cell adenoma or carcinoma of the thyroid gland. An increased incidence of pancreatic islet adenoma was also considered related to acrylamide exposure.

There was *clear evidence of carcinogenic activity* of acrylamide in female F344/N rats based on increased incidences of fibroadenoma of the mammary gland, squamous cell neoplasms (primarily papilloma) of the oral cavity (mucosa or tongue), mesenchymal neoplasms (fibroma, fibrosarcoma, or sarcoma) of the skin, and follicular cell neoplasms (adenoma or carcinoma) of the thyroid gland. Increased incidences of hepatocellular adenoma of the liver and carcinoma of the clitoral gland were also considered to be related to acrylamide exposure. The occurrence of malignant schwannoma of the heart may have been related to acrylamide exposure.

There was *clear evidence of carcinogenic activity* of acrylamide in male B6C3F1 mice based on increased incidences of neoplasms (primarily adenoma) of the harderian gland, alveolar/bronchiolar neoplasms (primarily adenoma) of the lung and squamous cell neoplasms (primarily papilloma) of the forestomach.

There was *clear evidence of carcinogenic activity* of acrylamide in female B6C3F1 mice based on increased incidences of harderian gland adenoma, alveolar/ bronchiolar adenoma of the lung, adenoacanthoma and adenocarcinoma of the mammary gland, benign granulosa cell neoplasms of the ovary, and malignant mesenchymal neoplasms of the skin. Increased incidences of squamous cell papilloma of the forestomach were also considered to be related to acrylamide exposure.

Figure 1. Statement of the NTP (2013) on the Results of their Acrylamide Bioassay

I have calculated dose-response models for most of the tumors sites for which the NTP declared either "Clear evidence of carcinogenic activity" or "considered to be related to acrylamide exposure." To do this modeling, I have used the publically available free BMDS

software used by the U.S. EPA, provided so that anyone could use the same software that it uses in its calculations. (U. S. EPA. BMDS version 2.4, and BMDS Wizard Version 1.8, available at http://www.epa.gov/ncea/bmds/. I have also used EPA's Benchmark Dose Technical Guidance. (EPA/100R-12/001. June 2012; http://www.epa.gov/raf/publications/pdfs/benchmark_dose_guidance.pdf) , and specific information on reporting and their cancer example from their training modules. http://www.epa.gov/ncea/bmds/training/ . For guidance on which models and procedures to use, I also use EPA's Carcinogen Risk Assessment Guidelines. EPA/630/P-03/001F, Mar 2005.

http://www.epa.gov/raf/publications/pdfs/CANCER_GUIDELINES_FINAL_3-25-05.PDF

I.B Animal Dose-Response Models for Individual Timor Sites. (All calculated using U.S. EPA's BMDS Version 2.4 Software)

The models under consideration are termed "multistage models," or "cancer-multistage models. They are basically linear (in the parameters) polynomial dose-response models of varying degree, with all the parameter values restricted to be non-negative, and the response a dichotomous "yes" or "no" cancer response. Because there are five dose groups, including the control, I allowed models with polynomials up to degree 4, plus a parameter estimate for background response. I also ran BMDS for other "best fitting" models, but this was strictly for comparing how the different models fit the data. Because of the overwhelming conclusion among so many government and health bodies, including U.S. and Cal EPA, that have considered that AA exerts its carcinogenic effects through a genotoxic mechanism of "mode of action," agencies that produce dose-response quantitative cancer risk assessments for AA would use the family of multistage models for their analysis. Often, models with only the linear term included might be considered. However, U.S. EPA's BMDS provides guidance and calculations to help choose the model that provides the "best fit" of the data from those of the multistage family. In our case, there are four models from which to choose.

For their cancer potency estimates, the U.S. EPA and the BMDS software basically use a modification of full model low-dose extrapolation called a "point of departure (POD) approach. This is all more fully explained in the BMDS training modules, but , essentially, the approach is to model the full data set, then use the best fitting model to select a lower confidence limit on the dose, $BMDL_{10}$ that will produce a 10% extra risk in cancer. From that $BMDL_{10}$, a "cancer slope factor," i.e. a linear dose-response slope, is created by taking risk/dose, or 0.1/ $BMDL_{10}$. This linear low dose model approach is used for carcinogens that are active through a genotoxic "mode of action" (MOA), which is the case for AA.

The BMDS software is used for estimating dose-response for both cancer and non-cancer risks, so it includes models not typically used in cancer modeling. However, examining some of these non-linear best fitting models can give a relative perspective on how well the multistage family fits the data.

Using the BMDS software, I fit the suite of models to the NTP tumor data for the rats and mice of both sexes. I report these results in Table 1.1 A-T. To determine the "best fitting" model(s), I relied on the BMDS Technical Guidance. The Technical Guidance suggests 3 ways to determine the best fitting model, and the statistics to evaluate these are presented in each portion of Table 1. The first is the well-known Chi square goodness of Fit test, which determines how well the model fits the data, overall at all the data points. The Guidance suggests that for the total suite of models, each model should fit with a probability, P>0.05. However, for the cancer multistage, the fit need only attain P>0.10, and that value is used here. The second criteria examines more closely the model fit at each data point, especially at the BMD_{10}; this is called the scaled residual and the suggested level of concern is the absolute value of this scaled residual is >2 . The third criterion is the Akaike information criterion, a value close to the statistical likelihood, but one that also incorporates the number of parameters estimated. The lower the Akaike score the better. If a model fits the data for these criteria, the BMDS software will declare the model "viable." Finally, the BMDS has an algorithm which will compare these models and choose one as "recommended" because it may have the "lowest Akaike score" or lowest $BMDL_{10}$. ' If several models provide adequate acceptance statistic the BMDS classifies these as "alternate."

The results of my 17 individual tumor sites' modeling of the NTP rats and mice tumor data are presented in **Table I.A-U**. I was unable to obtain model results for the female rat clitoral carcinoma and female rat liver hepatocellular adenoma, and I, therefore, eliminated these from further analyses. Most of my results match those shown in Beland et al (2013), although there are a few exceptions. By far the most notable are my model choices for male and female mouse Harderian Gland tumors. Beland's results suggest either log-logistic or log-probit models for this tumor site in both male and female mice. My results suggest that a one stage model will fit the both data sets barely adequately (0.05 <P <0.10). As a result, my $BMDl_{10}$ are higher than his for the mouse, making my cancer potency slopes over 40% lower for the female mouse (See Tables 1.L and I.M, which include BMDS results of fitting the data with and without the high dose groups.

Table I.2 presents the chosen animal cancer slope factors from the results of the modeling shown in Table I. A-U.

I.C. Summing Risks from Individual Tumor Sites to Estimate Total Cancer Risks from Multiple Tumor Sites.

Since AA causes multiple site tumors in the animal bioassays, risks from total cancer must be considered. In their respective quantitative risk analyses for AA, both U.S. EPA and Cal. EPA

estimated the combined risks by summing risks for each tumor site. Although their methods were slightly different, they both included summing central estimates of individual cancer slope factors, and factoring in the standard errors of these central estimates to derive a an upper bound on the combined summed cancer potency estimate. Based on my knowledge of these methods, I conclude that they are appropriate for this assessment of AA, and, using the same central estimates and their standard errors, will give near identical results to within round-off error. To do my calculations for summed cancer risks for the NTP rat and mice data, I have used U.S. EPA's method, detailed in their assessment, Appendix D, Table D-8 and Table D-10 (Plaintiff's Exhibit #48631). My results are presented here in four separate Tables I.B.1 – i.B.4, for male and female rats, and for male and female mice. They show results based on summing risks for different combinations of sites in each separate sex-species. The final animal cancer slope factors are:

Male Rats (4 sites): 0.2617 $(mg/kg\text{-}day)^{-1}$
Female Rats (4 sites): 0.3092 $(mg/kg\text{-}day)^{-1}$
Male Mice (3 sites): 0.3813 $(mg/kg\text{-}day)^{-1}$
Female Mice (6 sites): 0.3701 $(mg/kg\text{-}day)^{-1}$

These results suggest that the AA is about 30% more potent as a carcinogen in the mice than in the rats. These results are affected by the calculated slopes for Harderian Gland tumors in both the male and female mice.

1.D. Animal-to-Human Scaling or interspecies Conversion Factors.

In extrapolating cancer risks from animals to humans, both the U.S. EPA and Cal. EPA use what is variously called an "animal-to-human scaling factor" or "interspecies scaling factor" or "interspecies conversion factor." Cal. EPA notes that "the default interspecies factor assumes that dose in amount per surface area produces the same cancer incidence in different species. The factor is described as:

Cancer potency (human) = cancer potency (animal) x interspecies factor

For Cal. EPA, its default "interspecies conversion factor is based on surface area scaling, i.e., (human body weight/animal body weight)$^{1/3}$ [Title, California Code of Regulation, section 12703 (a)(6)." For its interspecies conversion factors for rats-to-humans in the Friedman (1995) and Johnson (1986) drinking water bioassays, these factors (for a 70 kg human) are:

Interspecies Factor male rat to human: $(70/0.35)^{1/3} = 5.85$
Female rat to human: $(70/0.20)^{1/3} = 7.05$

However, in its AA assessment, CAL EPA (page 24) further divides this interspecies factor into a pharmacokinetic(PK, differences in internal dose) and pharmacodynamic (PD, differences in response to internal dose) part:

Cancer potency (human) = cancer potency (animal) x PK factor x PD factor

For their PK factor, Cal. EPA derived a factor of 1.2 "as the ratio of the human to rat internal dose of glycidamide, normalized by the applied dose" (see their Table 9, page 21). Then their "assessment chose to proportion equally (page 24)" the PK and PD portions. The resulting animal-to-human conversions for male and female rats were:

Cancer potency (human) = cancer potency (male rat) x 1.2 x $(5.85)^{1/2}$
Cancer potency (human) = cancer potency (FEmale rat) x 1.2 x $(7.05)^{1/2}$

For Cal. EPA, their final rat-to-human conversion factors are 3.19 for the female rat and 2.90 for the male rat. For my analysis of the NTP (2012) data, I substitute the NTP male rat= 418.2 gm, and female rat= 262.0 gm weights and calculate factors of 2.82 for the male rat and 3.05 for the female rat. Based on these above scaling factors I calculate the human potency estimates based on the tumor sites in the NTP rats. These results are shown in Table I.D.1.

Also shown in Table I.D.1 are my results for the Human Cancer Potency Factors for the risk assessment based on the NTP mice. For the mice, I do not have an estimate of the PK factor in mice relative to humans, and choose to use the default interspecies scaling factor. However, instead of the $(W_h/W_a)^{1/3}$ factor used by Cal EPA, I choose the alternative of $(W_h/W_a)^{1/4}$ as discussed in U.S. EPA's Publication, "Recommended Use of Default Body Weight$^{3/4}$ as the Default Method in Derivation of the Oral Reference Dose." (EPA/100/R11/0001 Final) http://www.epa.gov/raf/publications/pdfs/recommended-use-of-bw34.pdf EPA also recommends use of this default animal-to-human scaling factor in its 2005 Cancer Guidelines. (http://www.epa.gov/raf/publications/pdfs/CANCER_GUIDELINES_FINAL_3-25-05.PDF)

The default scaling factors for the mouse then become: male mouse = $(70/0.031)^{1/4}$ = 6.89, and for the female mouse it is $(70/0.0252)^{1/4}$ = 7.27. As can be seen in the **Table I.D.1,** the overall risk estimates based on combined tumor sites in the mouse are about three times as high as those based on the rat, but a large portion of the mouse-to-human risk estimates are due to the contribution of Harderian Gland tumors.

Finally, this section concludes with a comparison of the human cancer potency estimates for rat tumors in the Johnson (1986), Friedman (1995), and NTP (2012) AA bioassays for both sexes. Both Cal. EPA and U.S.EPA reported on the two earlier AA bioassays, but I will show the Cal. EPA

factors for comparisons, because I used their interspecies conversion factors. These comparisons are shown in **Table I.D.2.** Although some of the tumor sites are different in the same sex in the different studies, the results for the combined human cancer potency estimates based on multiple tumor sites are quite close quantitatively in all three male and three female rat bioassays. Human cancer slope factors based on the NTP mouse study are about 3 times as high as those of the NTP rat. This factor is a combination of both the increased mouse cancer potency estimates, but more the animal-to-human species conversion factor that I used.

II. Acrylamide Content in Coffee

In order to estimate the risk of AA from drinking coffee, I need to know both the concentration of AA in coffee and the amount of coffee consumed per day. My estimates of AA concentration in Coffee come from the U.S. FDA survey. Dybing (2005, Plaintiff's Exhibit #23810), and Cal. EPA's "Characterization of Acrylamide Intake from Certain Foods." (2005. http://oehha.ca.gov/prop65/law/pdf_zip/acrylamideintakeReport.pdf) In 2003-2004 the FDA analyzed twenty samples of U.S. brewed coffee for acrylamide, The average concentration was 7.35 ppb or 7.35 ug/kg, standard deviation= 2.37. with a minimum of 3 ug/kg and a maximum of 13 ug/kg.

III. Amount of Coffee Consumed Per Day

The amount of coffee consumed/day will be estimated in two parts: 1) Number of cups/day, and 2) average size of a cup. Together, they will allow an estimate of the amount of coffee/day.

I estimated 1) the number of cups/day from the NCA Survey Table excel file: 0416_Age_18_Plus_banner1(Region,Gender,Age,Income,Kids).xslm I used the Table Q20, the WEST Region (column F). There were 653 (cell F858) people in the West region, of whom 428 (cell F861) replied they had drunk coffee "yesterday". The total number of cups they drank was 1318 (cell F910) for a mean number of cups = 3.08 (cell F904).

I estimated 2) the amount of ounces/cup from the same Excel file, but used Table Q21, % Drinking by Size of Cup. Again I used the West Region, Column F, summing the frequencies of those who reported drinking 3 oz. (expresso), 8 oz., 12 oz., and 16 oz., cups. – (cells F1849, F1852, F1855, and F1858). The total oz. of the 509 cups was 5,426, or 10.66 oz./cup.

IV. Estimates of Lifetime Cancer Risk from Acrylamide in Coffee.

Based on the concentration of AA in coffee (7.35 □g/kg coffee), the amount of coffee consumed by coffee drinkers in the West, (average 3.1 cups/day, and 10.66 oz./cup), we can calculate the amount of AA per cup of coffee as:

7.35 μg/kg x 10.66 oz./cup x 28.375 gm/oz. x 1 kg/1000 gm = 2.22 μg AA/cup.

For a 70 kg human, one cup/day yields 2.22 μg/70 kg = 0.0317 AA μg/kg-day. I use these values to estimate the lifetime cancer risk from AA in coffee based on my analyses above. I compare these results with those from both Cal EPA (2005) and U.S. EPA (2010), for daily coffee consumption of 1, 3.1, 5. 7. And 9 cups/day. All these human cancer potency factors must first be adjusted to account for little coffee drinking under the age of 18. Based on figures in the NCA Tables for coffee drinkers ages 13-17, (0416_Age_13_Plus_banner1(Region,Gender,Age,Income,Kids).xslm), I note some coffee drinkers between these ages, and I concluded that using 17 as a cut-off age for these purposes is reasonable. Thus, the results presented assume that there is no coffee drinking below age 17. (See also, Branum et. al.; 2014.) To the extent that there is, these risk estimates will be too low.

These sets of cancer increased risk estimates from AA in coffee, both from my analyses and those of the Cal. EPA and U.S. EPA are shown in **Table IV.A.1**. I use the amount of AA (2.22 μg AA/cup) from one cup per day (9th percentile of coffee drinkers) to extrapolate to amount of AA in 3.1 cups/day, the average consumption of adult coffee drinkers in the West. I also estimate the cancer risk to those who drink 5 (71st percentile), 7 (85th percentile), and 9 (91st percentile) cups per day. I note that for **all** those who drink at least one cup per day, **all** the cancer risk estimates predict greater than 1 in 100,000 increased risk of cancer. For the 20 estimates I calculated in the table, these increased risks range from a low of 1.2 x 10^{-5} for 1 cup/day to nearly 1 per 1000 for the 9 cup/day drinker.

Corresponding to cancer risk estimates, California often describes risks in terms of the **NSRL** (No Significant Risk Levels), the level of exposure below which excess lifetime cancer risk will be less than 1 per 100000. In its March 2005 risk assessment for AA, Cal. EPA (Plaintiff's Exhibit #23930) estimated the NSRL for all sources of AA, from birth, as 1.0 μg/day. As seen above and in Table IV.A.1, this level of AA is exceeded in just ½ cup of coffee per day.

V. Estimates of Margin-Of-Exposure Ratios (MOE) From AA in Coffee.

Some European commissions and health Agencies prefer the MOE approach over dose- response and animal-to-human extrapolation procedures. (See e.g. Plaintiff's Exhibit #41795), and I have provided an MOE analysis for cancer potency in the NTP 2 year rat and mouse bioassays. The analysis, presented in

Table V.A.1, provides the same estimates of the NTP animal cancer slope factors $BMDL_{10}$'s, amount of AA in coffee, and amount of human coffee consumption used in the quantitative risk assessment above, but it does not extrapolate down to lower levels of risk. Instead, the MOEs in the table provides ratios of the oral dose levels causing 10% extra risk of cancer in animals, to the amount of AA ingested from different levels of coffee. Note that the ratios are pure numbers, but the units are in (mg/kg –b.w.) animals/mg/kg –b.w. humans.

The estimates in Table V.A.1 show that the MOE's for the single coffee cup/day drinker (< 10% of coffee drinkers) are in the 8,000 – 12,000 range. For these 52% who drink more than the average 3.1 cups/day the MOE's vary between 900 and 3900, for a 10% excess cancer response.

VI. Conclusions and Opinion.

According to a recent National Coffee Association annual survey of 653 Western adults, the 65% who said they were coffee drinkers drank an average of 3.1 cups/day. For these drinkers, I estimated that their average excess lifetime cancer risks from the acrylamide in that coffee is between 5 and 20 per 100000, well above the California NSRL.

I believe that these are real risks. Even though the specifics of the extrapolations from animals to humans can show variations, the consistency of animal cancer responses, with multiple tumors in both sexes of all three AA rat drinking water bioassays and in the AA NTP mice bioassay, plus other positive AA cancer results in mice, provides strong evidence that AA causes cancers in humans.

I have presented four different estimates of the excess risk levels for human cancer, one each by the Cal. EPA and the U.S. EPA, and my two on the NTP (2012) AA bioassays. They differ by as much as a factor of 3.8 - my NTP combined sex mouse based analysis vs. the U.S. EPA rat based analysis. If one specific study must be cited, I believe the higher risk NTP mouse based analysis should be used as the final estimate, because:

1) California Code of Regulations 27 CCR Section 25703 (3) states:

   "Risk analysis shall be based on the most sensitive study deemed to be of sufficient quality."

I note that when Cal EPA (2005) and the U.S. EPA (2010) did their quantitative risk assessments, the NTP results were not available, so the rat

would have been the most sensitive species. However, in the NTP study, my analysis based on the animal cancer potency factors for combined tumor sites, Table I.D.1 shows that the mouse is 20% (for the female mouse vs. the female rat) to 45% (for the male mouse vs. the male rat) more sensitive than is the rat to the cancer effects of AA.

2) The Male and Female mouse had nearly identical combined tumor sites animal cancer slope factors of 0.381 $(mg/kg\text{-}d)^{-1}$ and 0.371 $(mg/kg\text{-}d)^{-1}$, respectively. Thus, I conclude that the male and female mice are equally sensitive.

Thus, I believe that the risks based on the pooled estimates I calculated for the NTP mice, Table IV.A.1, should be used for California's risk assessment purposes.

Additional References.

Branum A, Rossen LM, and Schoendorf KC. (2014). Trends in Caffeine Intake Among U.S. Children and Adolescents. Pediatrics. 2014; 133:386-393.

U.S. EPA. Recommended Use of Body Weight$^{3/4}$ as the Default Method in the derivation of the Oral Reference Dose. EPA/100/R11/0001 Final. http://www.epa.gov/raf/publications/pdfs/recommended-use-of-bw34.pdf

Table I.1. Results of U.S.EPA's (Benchmark Dose Software) BMDS 2.4, and BMDS Wizard Version 1.8 on NTP Acrylamide 2-Year Rat and Mice Bioassay studies TR 575 (2012). Calculation of BMD_{10}, $BMDL_{10}$, and Cancer Slope Factor for all tumors sites the NTP reported as Clear or Related evidence of acrylamide carcinogenicity. Results Calculated for EPA's Cancer Dichotomous Multistage Models with Number of dose parameters from 1-4. Recommended Selection of Model to be Used for the Combined Tumor Calculations, based on Goodness-of Fit (GOF; P>.0.05) of Model to Data, Akaike Information Criteria (AIC), Scaled Residuals at Low doses, and Lowest $BMDL_{10}$ for Models meeting the Recommended Criteria.

A. **Male Rat**: Tumor Site: Thyroid Follicular Cell Adenoma or Carcinoma

Dose mg/kg-d	**0**	**0.33**	**0.66**	**1.32**	**2.71**
Response	**1/47**	**3/48**	**4/47**	**6/48**	**0/48**

Results/Models	Multistage -1	Multistage -2	Multistage -3	Multistage -4	**Log-Probit**
BMD_{10}	1.45	1.45	1.45	1.45	1.17
$BMDL_{10}$	0.889	0.889	0.889	0.889	0.336
Cancer Slope Factor	0.113	0.113	0.113	0.113	0.2398
AIC	146.35	146.35	146.35	146.35	147.91
P-value G.O.F	0.947	0.947	0.947	0.947	0.994
Scaled Residuals at administered doses levels: Z= Nearest BMD.					
Dose : mg/kg-day	Scaled Residuals				
0	-0.347	-0.347	-0.347	-0.347	-0.010
0.33	0.297	Z0.297	Z0.297	Z0.297	0.072
0.66	0.253	0.253	0.253	0.253	-0.053
1.32	0.134^{Z}	0.134^{Z}	0.134^{Z}	0.134^{Z}	-0.051
2.71	-0.275	-0.275	-0.275	-0.275	0.041
Recommend?	Viable/alternate	Viable/alternate	Viable/alternate	Viable/alt.	
Comment	Chosen				Low $BMDL_{10}$

B. **Male Rat**: Tumor Site: Heart Schwannoma

Dose mg/kg -d	0	0.33	0.66	1.32	2.71
Response	1/48	2/48	3/48	4/48	6/48

Results/Models	Multistage -1	Multistage -2	Multistage -3	Multistage -4	**Other: Log-Probit**
BMD_{10}	2.43	2.43	2.43	2.43	2.41
$BMDL_{10}$	1.30	1.30	1.30	1.30	1.25
Cancer Slope Factor	0.0767	0.0767	0.0767	0.0767	0.0415
AIC	116.67	116.67	116.67	116.67	116.64
P-value G.O.F	0.982	0.982	0.982	0.982	
Scaled Residuals at administered doses levels: Z= Nearest BMD.					
Dose : mg/kg-day	Scaled Residuals				
0	-0.218	-0.218	-0.218	-0.218	-0.191
0.33	0.070	0.070	0.070	0.070	0.068
0.66	0.282	0.282	0.282	0.282	0.263
1.32	0.082	0.082	0.082	0.082	0.057
2.71	-0.183	-0.183	-0.183	-0.183	-0/172
Recommend?	Viable/alternate	Viable/alternate	Viable/alternate	Viable/alt.	Via/Recomm
Comment	Chosen				Low $BMDL_{10}$

C. Male Rat: Tumor Site: Epididymis or Testis Mesotheliomas

Dose	0	0.33	0.66	1.32	2.71
Response	2/48	2/48	1/48	5/48	8/48

Results/Models	Multistage -1	Multistage -2	Multistage -3	Multistage -4	Probit
BMD_{10}	2.09	2.18	2.18	2.18	2.16
$BMDL_{10}$	1.21	1.29	1.29	1.29	1.60
Cancer Slope Factor	0.0826	0.0776	0.0776	0.0776	0.0625
AIC	124.49	125.88	125.88	125.88	124.00
P-value G.O.F	0.613	0.487	0.487	0.487	0.685
Scaled Residuals at administered doses levels: Z= Nearest BMD. Dose : mg/kg-day Scaled Residuals					
0	0.558	0.232	0.232	0.232	0.378
0.33	-0.090	0.080	0.080	0.080	0.030
0.66	-1.144	-0.866	-0.866	-0.866	-0.959
1.32	0.322	0.768	0.768	0.768	0.644
2.71	0.278^{Z}	$--0.193^{Z}$	$--0.193^{Z}$	$--0.193^{Z}$	-0.091^{z}
Recommended Model	Viable/alternate	Via/alt.	Via/alt.	Via/alt.	Viable/ ”Recommended”
Comment:	Chosen/lowest BMD_{10}				Lowest AIC

D. Male Rat: Tumor Site: Pancreatic Islet Adenoma

Dose	0	0.33	0.66	1.32	2.71
Response	1/46	2/48	4/48	1/48	6/48

Results/Models	Multistage -1	Multistage -2	Multistage -3	Multistage -4	Other: Gamma
BMD_{10}	3.37	3.093	2.92	2.84	2.77
$BMDL_{10}$	1.59	1.603	1.65	1.68	1.73
Cancer Slope Factor	0.063	0.062	0.061	0.060	0.058
AIC	107.25	109.16	108.92	106.73	106.51
P-value G.O.F	0.366	0.191	0.191	0.360	0.386
Scaled Residuals at administered doses levels: Z= Nearest BMD. Dose : mg/kg-day: Scaled Residuals					
0	-0.293	-0.472	-0.629	-0.678	-0.688
0.33	0.097	0.087	0.029	-0.002	-0.015
0.66	1.109	1.286	1.399	1.427	1.422
1.32	-1.303	-1.160	-0.966	-0.842	-0.736
2.71	0.381	0.258	0.140	0.071	0.002
Recommended Model	Viable/Altern.	Viable/Altern.	Viable/Altern.	Viable/Altern.	Viable/ ”recommended”
Comment:	**Chosen/** BMD>max. dose	BMD>max. dose	BMD>maximum dose	BMD>max. dose	Lowest AIC BMD>max. dose

E. FEMale Rat: Thyroid Follicular cell Adenoma or CARCINOMA

Dose	0	0.44	0.86	1.84	4.02
Response	1/46	2/48	4/48	1/48	6/48

Results/Models	Multistage -1	Multistage -2	Multistage -3	Multistage -4	Other: Gamma
BMD_{10}	3.849	3.849	3.849	3.849	3.849
$BMDL_{10}$	2.328	2.328	2.328	2.328	2.328
Cancer Slope Factor	0.0430	0.0430	0.0430	0.0430	0.0430
AIC	70.5	70.5	70.5	70.5	70.5
P-value G.O.F	0.807	0.807	0.807	0.807	0.807
Scaled Residuals at administered doses levels: Z= Nearest BMD. Dose : mg/kg-day: Scaled Residuals					
0	0	0	0	0	0
0.44	-0.763	-0.763	-0.763	-0.763	-0.763
0.88	0.812	0.812	0.812	0.812	0.812
1.84	0.429	0.429	0.429	0.429	0.429
4.02	-0.429^{Z}	-0.429^{Z}	-0.429^{Z}	-0.429^{Z}	-0.429^{Z}
Recommended Model	Viable/Altern.	Viable/Altern.	Viable/Altern.	Viable/Altern.	Viable/ ”recommended”
Comment:	**Chosen/** Lowest AIC ; BMD>max. dose	BMD>max. dose	BMD>maximum dose	BMD>max. dose	Lowest AIC BMD>max. dose

F. FEMale Rat: Mammary Gland Fibroadenoma

Dose	0	0.44	0.88	1.84	4.02
Response	16/48	18/48	24/46	22/47	31/48

Results/Models	Multistage -1	Multistage -2	Multistage -3	Multistage -4	Other: Dich-Hill
BMD_{10}	0.706	0.706	0.706	0.706	0.499
$BMDL_{10}$	0.441	0.441	0.441	0.441	0.0921
Cancer Slope Factor	0.227	0.227	0.227	0.227	1.086
AIC	321.59	321.59	321.59	321.59	323.48
P-value G.O.F	0.584	0.584	0.584	0.584	0.401
Scaled Residuals at administered doses levels: Z= Nearest BMD. Dose : mg/kg-day: Scaled Residuals					
0	-0.312	-0.312	-0.312	-0.312	-0.085
0.44	-0.295	-0.295	-0.295	-0.295	-0.329^{Z}
0.88	1.198^{Z}	1.198^{Z}	1.198^{Z}	1.198^{Z}	1.029
1.84	-0.571	-0.571	-0.571	-0.571	-0.786
4.02	0.001	0.001	0.001	0.001	0.184
Recommended Model	Viable/Altern.	Viable/Altern.	Viable/Altern.	Viable/Altern.	Viable/ ”recommended”
Comment:	**Chosen/** Low AIC ; Good fit.				Lowest $BMDL_{10-}$

G. FEMale Rat: Oral Mucosa/Tongue, Sq. Cell Pappilloma or Carcinoma

Dose	0	0.44	0.88	1.84	4.02
Response	0/48	2/48	1/48	3/48	5/48

Results/Models	Multistage -1	Multistage -2	Multistage -3	Multistage -4	Other: Log-Probit
BMD_{10}	3.518	3.518	3.518	3.518	4.40
$BMDL_{10}$	2.019	2.019	2.019	2.019	1.86
Cancer Slope Factor	0.0495	0.0495	0.0495	0.0495	0.0538
AIC	86.7	86.7	86.7	86.7	85.880
P-value G.O.F	0.539	0.539	0.539	0.539	0.816
Scaled Residuals at administered doses levels: Z= Nearest BMD. Dose : mg/kg-day: Scaled Residuals					
0	-0.474	-0.474	-0.474	-0.474	0
0.44	1.260	1.260	1.260	1.260	0.600
0.88	-0.391	-0.391	-0.391	-0.391	-0.733
1.84	0.133	0.133	0.133	0.133	-0.039
4.02	-0.288^{Z}	-0.288^{Z}	-0.288^{Z}	-0.288^{Z}	0.200^{Z}
Recommended Model	Viable/Altern.	Viable/Altern.	Viable/Altern.	Viable/Altern.	Viable/ ”recommended”
Comment:	**Chosen/**				

H. FEMale Rat: Skin Fibroma, Fibrosarcoma or sarcoma[F]

Dose	0	0.44	0.88	1.84	4.02
Response	1/48	0/48	0/48	1/48	5/48

Results/Models	Multistage	Multistage -2	Multistage -3	Multistage -4	Other: Log-Probit
BMD_{10}	4.286				4.042
$BMDL_{10}$	3.084				3.010
Cancer Slope Factor	0.032				0.033
AIC	58.4				59.8
P-value G.O.F	0.486				0.373
Scaled Residuals at administered doses levels: Z= Nearest BMD. Dose : mg/kg-day: Scaled Residuals					
0					
0.44					
0.88					
1.84					
4.02					
Recommended Model					
Comment:	**Chosen/** Lowest AIC				Lowest $BMDL_{10}$

F. My BMD calculations for this data set failed. These are from Beland (2013).

I. Male Mouse: Harderian Gland Tumors – All Doses

Dose	0	1.04	2.2	4.11	8.93
Response	2/46	13/46	27/47	36/47	39/47

Results/Models	Multistage -1	Multistage -2	Multistage -3	Multistage -4	Other: Log-Probit
BMD_{10}	0.386	0.386	0.386	0.386	0.383
$BMDL_{10}$	0.320	0.320	0.320	0.320	0.159
Cancer Slope Factor	0.312	0.312	0.312	0.312	0.628
AIC	240.13	240.13	240.13	240.13	237.5
P-value G.O.F	0.053	0.053	0.053	0.053	0.342
Scaled Residuals at administered doses levels: Z= Nearest BMD.					
Dose : mg/kg-day:			Scaled Residuals		
0	-0.313	-0.313	-0.313	-0.313	
1.04	-0.075^{Z}	-0.075^{Z}	-0.075^{Z}	-0.075^{Z}	
2.20	1.284	1.284	1.284	1.284	
4.11	1.102	1.102	1.102	1.102	
8.93	-2.176	-2.176	-2.176	-2.176	
Recommended Model	Viable/Alternate				
Comment:	**Chosen/** Multistage				Lowest $BMDL_{10}$ low AIC

J. Male Mouse: Lung Alveolar/Bronchiolar Adenoma

Dose	0	1.04	2.2	4.11	8.93
Response	5/47	6/46	13/47	10/45	19/48

Results/Models	Multistage -1	Multistage -2	Multistage -3	Multistage -4	Other: Log-Probit
BMD_{10}	2.41	2.41	2.41	2.41	1.77
$BMDL_{10}$	1.57	1.57	1.57	1.57	0.275
Cancer Slope Factor	0.0556	0.0556	0.0556	0.0556	0.3636
AIC	241.40	241.40	241.40	241.40	243.17
P-value G.O.F	0.475	0.475	0.475	0.475	0.338
Scaled Residuals at administered doses levels: Z= Nearest BMD. Dose : mg/kg-day: Scaled Residuals					
0	-0.214	-0.214	-0.214	-0.214	0.055^{Z}
1.04	-0.470	-0.470	-0.470	-0.470	-0.530
2.20	-1.367^{Z}	-1.367^{Z}	-1.367^{Z}	-1.367^{Z}	1.095
4.11	-0.599	-0.599	-0.599	-0.599	-0.812
8.93	-0.081	-0.081	-0.081	-0.081	0.168
Recommended Model	Viable/Altern.	Viable/Altern.	Viable/Altern.	Viable/Altern.	"recomm."
Comment:	**Chosen/** Multistage				Lowest $BMDL_{10}$ low AIC

K. Male Mouse: Stomach (fore), Squamous Cell Pappilloma

Dose	0	1.04	2.2	4.11	8.93
Response	0/46	2/45	2/46	6/47	6/44

Results/Models	Multistage -1	Multistage -2	Multistage -3	Multistage -4	Other: Dich-Hill
BMD_{10}	4.548	4.548	4.548	4.548	3.91
$BMDL_{10}$	3.094	3.094	3.094	3.094	1.10
Cancer Slope Factor	0.0323	0.0323	0.0323	0.0323	0.091
AIC	107.96	107.96	107.96	107.96	110.67
P-value G.O.F	0.667	0.667	0.667	0.667	0.647
Scaled Residuals at administered doses levels: Z= Nearest BMD. Dose : mg/kg-day: Scaled Residuals					
0	0.000	0.000	0.000	0.000	0
1.04	0.908	0.908	0.908	0.908	0.281
2.20	-0.194	-0.194	-0.194	-0.194	-0.670
4.11	0.879^{Z}	0.879^{Z}	0.879^{Z}	0.879^{Z}	0.559^{Z}
8.93	-0.860	-0.860	-0.860	-0.860	-0.159
Recommended Model	Viable/Altern.	Viable/Altern.	Viable/Altern.	Viable/Altern.	“recomm.”
Comment:	**Chosen/** near low AIC				Lowest $BMDL_{10}$

L. FEMale Mouse: Harderian Gland Adenoma. All Doses

Dose	0	1.10	2.23	4.65	1.96
Response	0/45	8/44	20/48	32/47	31/43

Results/Models	Multistage -1	Multistage -2	Multistage -3	Multistage -4	Other: Dich-Hill
BMD_{10}	0.567	0.567	0.567	0.567	0.808
$BMDL_{10}$	0.474	0.474	0.474	0.474	0.385
Cancer Slope Factor	0.211	0.211	0.211	0.211	0.260
AIC	226.13	226.13	226.13	226.13	222.97
P-value G.O.F	0.0867	0.0867	0.0867	0.0867	0.880
Scaled Residuals at administered doses levels: Z= Nearest BMD. Dose : mg/kg-day: Scaled Residuals					
0	0	0	0	0	0
1.10	-0.054^{Z}	-0.054^{Z}	-0.054^{Z}	-0.054^{Z}	0.140
2.23	1.130	1.130	1.130	1.130	-0.273
4.68	1.417	1.417	1.417	1.417	0.352
9.96	-2.202	-2.202	-2.202	-2.202	-0.192
Recommended Model	Viable/Altern.	Viable/Altern.	Viable/Altern.	Viable/Altern.	Viable "recomm."
Comment:	**Chosen/** poor G.O.F.	poor G.O.F.	poor G.O.F.	poor G.O.F.	Lowest AIC

M. FEMale Mouse: Harderian Gland Adenoma. High dose Dropped

Dose	0	1.10	2.23	4.65	
Response	0/45	8/44	20/48	32/47	Dropped

Results/Models	Multistage -1	Multistage -2	Multistage -3	Multistage -4	Other: Log-Logistic
BMD_{10}	0.452	0.527	0.527	0.527	0.692
$BMDL_{10}$	0.366	0.371	0.371	0.371	0.339
Cancer Slope Factor	0.273	0.270	0.270	0.270	0.295
AIC	168.42	170.09	170.09	170.09	169.82
P-value G.O.F	0.897	0.662	0.662	0.662	0.988
Scaled Residuals at administered doses levels: Z= Nearest BMD. Dose : mg/kg-day: Scaled Residuals					
0	0	0	0	0	0
1.10	-0.701Z	-0.359	-0.359	-0.359	-0.078Z
2.23	0.163	0.393	0.393	0.393	0.116
4.65	0.281	-0.113	-0.113	-0.113	-0.061
Dropped					
Recommended Model	Viable/ ”Recommend”	Viable/Altern.	Viable/Altern.	Viable/Altern.	Viable “recomm.”
Comment:	**Chosen Low A.I.C.**				Lowest BMDL

N. FEMale Mouse: Lung Alveolar Bronchiolar Adenoma.

Dose	0	1.10	2.23	4.65	9.96
Response	1/47	4/47	6/48	11/45	19/45

Results/Models	Multistage -1	Multistage -2	Multistage -3	Multistage -4	Other: Log-Logistic
BMD_{10}	1.97	1.97	1.97	1.97	1.91
$BMDL_{10}$	1.47	1.47	1.47	1.47	0.912
Cancer Slope Factor	0.0680	0.0680	0.0680	0.0680	0.11
AIC	188.62	188.62	188.62	188.62	190.74
P-value G.O.F	0.995	0.995	0.995	0.995	.910
Scaled Residuals at administered doses levels: Z= Nearest BMD. Dose : mg/kg-day: Scaled Residuals					
0	-0.050	-0.050	-0.050	-0.050	-0.062
1.10	0.175	0.175	0.175	0.175	0.306
2.23	-0.151^{Z}	-0.151^{Z}	-0.151	-0.151^{Z}	-0.259^{Z}
4.65	0.105	0.105	0.105	0.105	-0.-096
9.96	-0.057	-0.057	-0.057	-0.057	0.119
Recommended Model	Viable/altern.	Viable/Altern.	Viable/Altern.	Viable/Altern.	Viable "recomm."
Comment:	**Chosen Low A.I.C.**				Lowest BMDL

O. FEMale Mouse: Lung Alveolar Bronchiolar Adenoma.

Dose	0	1.10	2.23	4.65	9.96
Response	1/47	4/47	6/48	11/45	19/45

Results/Models	Multistage -1	Multistage -2	Multistage -3	Multistage -4	Other: Log-Logistic
BMD_{10}	1.97	1.97	1.97	1.97	1.91
$BMDL_{10}$	1.47	1.47	1.47	1.47	0.912
Cancer Slope Factor	0.0680	0.0680	0.0680	0.0680	0.11
AIC	188.62	188.62	188.62	188.62	190.74
P-value G.O.F	0.995	0.995	0.995	0.995	.910
Scaled Residuals at administered doses levels: Z= Nearest BMD. Dose : mg/kg-day: Scaled Residuals					
0	-0.050	-0.050	-0.050	-0.050	-0.062
1.10	0.175	0.175	0.175	0.175	0.306
2.23	-0.151^{Z}	-0.151^{Z}	-0.151	-0.151^{Z}	-0.259^{Z}
4.65	0.105	0.105	0.105	0.105	-0.-096
9.96	-0.057	-0.057	-0.057	-0.057	0.119
Recommended Model	Viable/altern.	Viable/Altern.	Viable/Altern.	Viable/Altern.	Viable "recomm."
Comment:	**Chosen Low A.I.C.**				Lowest BMDL

P. FEMale Mouse: Stomach (Fore) Squamous Cell Pappilloma.

Dose	0	1.10	2.23	4.65	9.96
Response					

Results/Models	Multistage -1	Multistage -2	Multistage -3	Multistage -4	Other:
BMD_{10}	7.66	7.62	7.62	7.62	
$BMDL_{10}$	4.29	5.05	5.05	5.05	
Cancer Slope Factor	0.0233	0.0198	0.0198	0.0198	
AIC	128.19	126.69	126.69	126.69	
P-value G.O.F	0.107	0.183	0.183	0.183	
Scaled Residuals at administered doses levels: Z= Nearest BMD. Dose : mg/kg-day: Scaled Residuals					
0	1.605	1.373	1.373	1.373	
1.10	-1.633	-1.508	-1.508	-1.508	
2.23	-0.755	-0.366	-0.366	-0.366	
4.65	0.252	0.720	0.720	0.720	
9.96	0.477	-0.189	-0.189	-0.189	
Recommended Model	Viable/altern.	Viable/Recomm	Viable/Recomm	Viable/Recomm	
Comment:		**Chosen Lowest A.I.C.**			

Q. FEMale Mouse: Stomach (Fore) Squamous Cell Pappilloma.

Dose	0	1.10	2.23	4.65	9.96
Response	4/46	0/46	2/46	5/45	8/42

Results/Models	Multistage -1	Multistage -2	Multistage -3	Multistage -4	Other:
BMD_{10}	7.66	7.62	7.62	7.62	
$BMDL_{10}$	4.29	5.05	5.05	5.05	
Cancer Slope Factor	0.0233	0.0198	0.0198	0.0198	
AIC	128.19	126.69	126.69	126.69	
P-value G.O.F	0.107	0.183	0.183	0.183	
Scaled Residuals at administered doses levels: Z= Nearest BMD. Dose : mg/kg-day: Scaled Residuals					
0	1.605	1.373	1.373	1.373	
1.10	-1.633	-1.508	-1.508	-1.508	
2.23	-0.755	-0.366	-0.366	-0.366	
4.65	0.252	0.720	0.720	0.720	
9.96	0.47^{Z}	-0.189^{Z}	-0.189^{Z}	-0.189^{Z}	
Recommended Model	Viable/altern.	Viable/Recomm	Viable/Recomm	Viable/Recomm	
Comment:		**Chosen Lowest A.I.C.**			

R. FEMale Mouse Mammary Gland Adenocanthoma or Adenocarcinoma.

Dose	0	1.10	2.23	4.65	9.96
Response	0/47	4/46	7/48	4/45	17/42

Results/Models	Multistage -1	Multistage -2	Multistage -3	Multistage -4	Other:
BMD_{10}	2.219	2.22	2.27	2.37	1.72
$BMDL_{10}$	1.679	1.68	1.68	1.69	0.691
Cancer Slope Factor	0.0595	0.0595	0.0595	0.0592	0.1447
AIC	158.95	158.85	160.93	160.83	161.96
P-value G.O.F	0.211	0.211	0.114	0.111	
Scaled Residuals at administered doses levels: Z= Nearest BMD.					
Dose : mg/kg-day:		Scaled Residuals			
0	0	0	0	0	0
1.10	1.184	1.114	1.184	1.252	0.520
2.23	1.13^{Z}	1.046^{Z}	1.130^{Z}	1.220^{Z}	0.478^{Z}
4.65	01.788	-1.838	-1.788	-1.712	-1.973
9.96	0.265	0.375	0.265	0.132	1.096
Recommended Model	Viable/altern.	Viable/altern	Viable/altern	Viable/altern	
Comment:	Chosen				

S. FEMale Mouse Ovary Benign Granulosa Cell Tumor

Dose	0	1.10	2.23	4.65	9.96
Response	0/46	1/45	0/48	1/45	5/42

Results/Models	Multistage -1	Multistage -2	Multistage -3	Multistage -4	Other: Dichotomous-Hill
BMD_{10}	11.4	9.71	9.51	9.51	5.78
$BMDL_{10}$	6.48	6.66	6.80	6.83	4.90
Cancer Slope Factor	0.0154	0.0150	0.0147	0.0146	0.0146
AIC	55.302	56.517	58.213	58.156	58.11
P-value G.O.F	0.626	0.481	0.342	0.348	0.349
Scaled Residuals at administered doses levels: Z= Nearest BMD.					
Dose : mg/kg-day:			Scaled Residuals		
0	0	-0.504	-0.513	-0.452	-0.475
1.10	0.810	1.293	1.189	1.185	1.223
2.23	01.001	-0.731	-0.685	-0.721	-0.702
4.65	-0.665	-0.279	-0.000	0.019	-0.010 [Z]
9.96	0.707 [Z]	0.191 [Z]	0.023 [Z]	0.004 [Z]	0.018
Recommended Model	Viable/Recomm Lowest A.I.C.	Viable/altern	Viable/altern	Viable/altern	Viable/alt.
Comment:	Chosen				Lowest BMDL

T. FEMale Mouse Skin Fibrocarcoma, Sarcoma, Others

Dose	0	1.10	2.23	4.65	9.96
Response	0/48	0/46	3/48	10/45	6/43

Results/Models	Multistage -1	Multistage -2	Multistage -3	Multistage -4	Other: Dichotomous-Hill
BMD_{10}	4.09	4.09	4.09	4.09	2.34
$BMDL_{10}$	2.86	2.86	2.86	2.86	2.10
Cancer Slope Factor	0.034	0.034	0.034	0.034	.0476
AIC	115.92	115.92	115.92	115.92	
P-value G.O.F	0.0726	0.0726	0.0726	0.0726	0.799
Scaled Residuals at administered doses levels: Z= Nearest BMD.					
Dose : mg/kg-day:			Scaled Residuals		
0	0	0	0	0	
1.10	-1.150	-1.150	-1.150	-1.150	
2.23	0.200	0.200	0.200	0.200	
4.65	2.315^{Z}	2.315^{Z}	2.315^{Z}	2.315^{Z}	
9.96	-1.362	-1.362	-1.362	-1.362	
Recommended Model	Questionable/ residual>2	Questionable	Questionable	Questionable	Recommended.
Comment:	Poor GOF				Lowest BMDL

U. FEMale Mouse Skin Fibrocarcoma, Sarcoma, Others

Dose	0	1.10	2.23	4.65	9.96
Response	0/48	0/46	3/48	10/45	Dropped

Results/Models	Multistage -1	Multistage -2	Multistage -3	Multistage -4	Other:
BMD_{10}	2.75	3.05	3.17		
$BMDL_{10}$	1.80	2.41	2.42		
Cancer Slope Factor	0.0556	0.0415	0.0413		
AIC	77.309	73.438	75.319		
P-value G.O.F	0.338	0.874	0.685		
Scaled Residuals at administered doses levels: Z= Nearest BMD. Dose : mg/kg-day: Scaled Residuals					
0	0	0	0		
1.10	-1.407	-0.797	-0.688		
2.23	-0.489^{Z}	0.235^{Z}	0.524^{Z}		
4.65	1.074	0.080	-0.097		
Dropped					
Recommended Model	Viable	Viable/Recomm Chosen	Viable		
Comment:	Lowest BMDL	Lowest A.I.C.			

Table I.2. Animal Oral Cancer Slope (Potency) Estimates for Acrylamide based on Tumor Results in the NTP (2012) Rat and Mouse 2-Year Study Bioassay. Estimates for those Tumor sites NTP judged as "Clear Evidence of Carcingenic Activity" or "Related." Estimates calculated using U.S. EPA's BMDS Version 2.4, and BMDS Wizard Ver 1.8, plus EPA's method for calculating Combined Risks.

Sex/Species	ORGAN	Evidence	MODEL	BMD10	BMDL10	S.E. BMD	Animal Cancer Slope factors
M/mouse	Harderian Adenoma	Clear	Multi1	0.386	0.32	0.040122	0.312500
M/mouse	Lung Adeno	Clear	Multi1	2.414	1.57	0.51307	0.063694
M/mouse	Stomach	Clear	Multi2	4.548	3.094	0.883891	0.032321
F/mouse	Harderian Adeno	clear	Multi1	0.567	0.474	0.056535	0.210970
F/mouse	Lung Adeno	clear	multi1	1.968	1.471	0.302128	0.067981
F/mouse	Stomach	related	multi2	7.623	5.045	1.567173	0.019822
F/mouse	Mammary Adcarc/acnatho	clear	multi1	2.219	1.679	0.328267	0.059559
F/mouse	Ovary granulosa	clear	Multi1	11.4	6.48	2.990881	0.015432
F/mouse	Skin fibroma/sarcoma	clear	multi2 Hi D	3.05	2.41	0.389058	0.041494
M/Rat	Thyroid Gland foll. Ad/Ca.	clear	Multi1	1.448	0.889	0.339818	0.112486
M/Rat	Heart Schwannoma	clear	Multi1	2.426	1.304	0.682067	0.076687
M/Rat	Epidy testis/mesothel.	clear	Multi1	2.09	1.21	0.534954	0.082645
M/Rat	Pancreas Islets Adeno.	related	Multi1	3.37	1.59	1.082067	0.062893
F/Rat	Thyroid Gland foll. Ad/Ca.	clear	Multi1	3.849	2.328	0.92462	0.042955
F/Rat	Mammary Fibroadenoma	clear	Multi1	0.706	0.441	0.161094	0.226757
F/Rat	Oral Mucosa/tongue	clear	Multi1	3.518	2.019	0.911246	0.049529
F/Rat	Skin fibroma/sarcoma	clear	Multi	4.286	3.084	0.730699	0.032425
F/Rat	Clitoral gland carcinoma	related	not calc..				
F/Rat	Liver Hepato Adenoma	related	not calc..				

TABLE I.B.1. Calculation of Summed Risks for tumors at Several Sites in **MALE F344 Rats** exposed to AA in Drinking Water in the NTP Bioassay (2012; Plaintiff's Exhibit 48449). Doses Groups: 0, 0.33, 0.66, 1.32, and2.71 mg/kg/day Using BMD10 and BMDL10 output from EPA's BMDS Software 2.4 and BMDS Wizard 1.8. Calculations for Combined Risks for Multiple Tumor Sites based on method in U.S. EPA Tox. Review of Acrylamide, Table D8 and Table D-10, in Appendix D. (U.S. EPA; 2010; Plaintiff's Exhibit 48631).

TUMOR SITE	**BMD10**	**BMDL10**	RAT Oral Cancer slope factor (central tendency) (per mg/kg-day)	RAT Oral Cancer slope factor (upper-bound) (per mg/kg-day)	t-statistic	s.d.	Variance
	mg/kg-day	**mg/kg-day**					
Thyroid Gland, Foll.Adeno & Carc.	**1.448**	**0.889**	0.069061	0.112486	1.645	0.02640	0.000697
Heart Schwannoma	**2.426**	**1.304**	0.04122	0.076687	1.645	0.02156	0.000465
Cumulative Variance =	0.00116						
Cumulative s.d. =	0.03408						
Sum of Central tendency Risks=	0.11028	(mg/kg-day)-1					
Upper-bound on cum risk=	**0.16635**	(mg/kg-day)-1	**CANCER SLOPE FACTOR** FOR TWO Combined SITES				
Thyroid Gland, a&c	**1.448**	**0.889**	0.069061	0.112486	1.645	0.026398	0.000697
Heart Schwannoma	**2.426**	**1.304**	0.04122	0.076687	1.645	0.02156	0.000465
Epididymis testis/mesothelioma	**2.09**	**1.21**	0.047847	0.082645	1.645	0.021154	0.000447
Cumulative Variance =	0.001609						
Cumulative s.d. =	0.040115						
Sum of Central tendency Risks=	0.158128						
Upper-bound on cum risk=	**0.224117**	(mg/kg-day)-1	**CANCER SLOPE FACTOR** FOR THREE Combined SITES				
Thyroid Gland, a&c	**1.448**	**0.889**	0.069061	0.112486	1.645	0.026398	0.000697
Heart Schwannoma	**2.426**	**1.304**	0.04122	0.076687	1.645	0.02156	0.000465
Epididymis testis/mesothelioma	**2.09**	**1.21**	0.047847	0.082645	1.645	0.021154	0.000447
Pancreatic Islets Adenoma	**3.37**	**1.59**	0.029674	0.062893	1.645	0.020194	0.000408
Cumulative Variance =	0.002017						
Cumulative s.d. =	0.044911						
Sum of Central tendency Risks=	0.187801						
Upper-bound on cum risk=	**0.261680**	(mg/kg-day)-1	**CANCER SLOPE FACTOR** FOR FOUR Combined SITES				

TABLE I.B.2. Calculation of Summed Risks for tumors at Several Sites in **FEMALE F344 Rats** exposed to AA in Drinking Water in the NTP Bioassay (2012; Plaintiff's Exhibit 48449). Dose Groups: 0, 0.44, 0.88, 1.84, and 4.02 mg/kg/day. Using BMD10 and BMDL10 output from EPA's BMDS Software 2.4 and BMDS Wizard 1.8. Calculations for Combined Risks for Multiple Tumor Sites based on method in U.S. EPA Tox. Review of AA, Table D-8 and Table D-10 in Appendix D. (U.S. EPA; 2010; Plaintiff's Exhibit 48631).

TUMOR SITE	**BMD10**	**BMDL10**	Oral slope factor (central tendency)	Oral slope factor (upper-bound)	t-statistic	s.d.	Variance
	mg/kg-day	**mg/kg-day**	per (mg/kg-day)	per (mg/kg-day)			
Thyroid Gland, Foll.Adeno & Carc.	**3.849**	**2.328**	0.025981	0.042955	1.645	0.010319	0.000106
Mammary Fibroadenoma	**0.706**	**0.441**	0.141643	0.226757	1.645	0.051741	0.002677
Cumulative Variance =	0.00278						
Cumulative s.d. =	0.05276						
Sum of Central tendency Risks=	0.16762	(mg/kg-day)-1					
Upperbound on cum risk=	**0.25441**	(mg/kg-day)-1	**CANCER SLOPE FACTOR** FOR TWO Combined SITES				
Thyroid Gland, a&c	**3.848**	**2.328**	0.025988	0.042955	1.645	0.010315	0.000106
Mammary Fibroadenoma	**0.706**	**0.441**	0.141643	0.226757	1.645	0.051741	0.002677
Oral Mucosa/Tongue	**3.518**	**2.019**	0.028425	0.049529	1.645	0.012829	0.000165
Cumulative Variance =	0.002948						
Cumulative s.d. =	0.054297						
Sum of Central tendency Risks=	0.196056						
Upperbound on cum risk=	**0.285374**	(mg/kg-day)-1	**CANCER SLOPE FACTOR** FOR THREECombined SITES				
Thyroid Gland, a&c	**3.848**	**2.328**	0.025988	0.042955	1.645	0.010315	0.000106
Mammary Fibroadenoma	**0.706**	**0.441**	0.141643	0.226757	1.645	0.051741	0.002677
Oral Mucosa/Tongue	**3.518**	**2.019**	0.028425	0.049529	1.645	0.012829	0.000165
Skin Fibrosar/heman/ sarc,etc	**4.286**	**3.084**	0.023332	0.032425	1.645	0.005528	3.06E-05
Cumulative Variance =	0.002979						
Cumulative s.d. =	0.054577						
Sum of Central tendency Risks=	0.219388						
Upperbound on cum risk=	**0.309168**	(mg/kg-day)-1	**CANCER SLOPE FACTOR** FOR FOUR Combined SITES				

TABLE I.B.3. Calculation of Summed Risks for tumors at Several Sites in **MALE B6C3F1 MICE** exposed to AA in Drinking Water in the NTP Bioassay (2012; Plaintiff's Exhibit 48449). Dose Groups: 0, 1.04, 2.20, 4.11, and 8.93 mg/kg/day. Using BMD10 and BMDL10 output from EPA's BMDS Software 2.4 and BMDS Wizard 1.8. Calculations for for Combined Risks for Multiple Tumor Sites based on method in U.S. EPA Tox. Review of Acrylamide, Table D8 in Appendix D. (U.S. EPA; 2010; Plaintiff's Exhibit 48631).

TUMOR SITE	**BMD10**	**BMDL10**	MOUSE Oral Cancer slope factor (central tendency)	MOUSE Oral Cancer slope factor (upper-bound)	t-statistic	s.d.	Variance
	mg/kg-day	**mg/kg-day**	(per mg/kg-day)	(per mg/kg-day)			
Harderian Gland Adenoma	**0.386**	**0.32**	0.259067	0.3125	1.645	0.03248	0.001055071
Lung Alv./Bronch Adenoma	**2.414**	**1.57**	0.041425	0.063694	1.645	0.013538	0.000183265
Cumulative Variance =	0.00124						
Cumulative s.d. =	0.03519						
Sum of Central tendency Risks=	0.30049	(mg/kg-day)-1					
Upper-bound on cum risk=	**0.35838**	(mg/kg-day)-1	**CANCER SLOPE FACTOR** FOR TWO Combined SITES				
Harderian Gland Adenoma	**0.386**	**0.32**	0.259067	0.3125	1.645	0.032482	0.001055071
Lung Alv./Bronch Adenoma	**2.414**	**1.57**	0.041425	0.063694	1.645	0.013538	0.000183265
Stomach (fore) SQ. cell Pappilloma	**4.548**	**3.094**	0.021988	0.032321	1.645	0.006281	3.94562E-05
Cumulative Variance =	0.001278						
Cumulative s.d. =	0.035746						
Sum of Central tendency Risks=	0.322480						
Upper-bound on cum risk=	**0.381283**	(mg/kg-day)-1	**CANCER SLOPE FACTOR** FOR THREE Combined SITES				

TABLE I.B.4. Calculation of Summed Risks for tumors at Several Sites in **FEMALE B6C3F1 MICE** exposed to AA in Drinking Water in the NTP Bioassay (2012; Plaintiff's Exhibit 48449). Doses Groups: 0, 1.10, 2.23, 4.65, and 9.96 mg/kg-da. Using BMD10 and BMDL10 output from EPA's BMDS Software 2.4 and BMDS Wizard 1.8. Calculations for Combined Risks for Multiple Tumor Sites based on method in U.S. EPA Tox. Review of Acrylamide, Tables D-8 and D-10 in Appendix D. (U.S. EPA; 2010; Plaintiff's Exhibit 48631).

TUMOR SITE	**BMD10**	**BMDL10**	MOUSE Oral Cancer slope factor (central tendency)	MOUSE Oral Cancer slope factor (upper-bound)	t-statistic	s.d.	Variance
	mg/kg-day	**mg/kg-day**	(per mg/kg-day)	(per mg/kg-day)			
Harderian Gland Adenoma	**0.568**	**0.474**	0.176056	0.21097	1.645	0.02122	0.000450475
Lung Alv./Bronch Adenoma	**1.968**	**1.471**	0.050813	0.067981	1.645	0.010436	0.000108919
Cumulative Variance =	0.00056						
Cumulative s.d. =	0.02365						
Sum of Central tendency Risks=	0.22687	(mg/kg-day)-1					
Upper-bound on cum risk=	**0.26578**	(mg/kg-day)-1	**CANCER SLOPE FACTOR** FOR **TWO** Combined SITES				
Harderian Gland Adenoma	**0.568**	**0.474**	0.176056	0.21097	1.645	0.021224	0.000450475
Lung Alv./Bronch Adenoma	**1.968**	**1.471**	0.050813	0.067981	1.645	0.010436	0.000108919
Mammary Adenocarc/acanth	**2.219**	**1.679**	0.045065	0.059559	1.645	0.008811	7.76318E-05
Cumulative Variance =	0.000637						
Cumulative s.d. =	0.025239						
Sum of Central tendency Risks=	0.271935						
Upper-bound on cum risk=	**0.313453**	(mg/kg-day)-1	**CANCER SLOPE FACTOR** FOR **THREE** Combined SITES				
Harderian Gland Adenoma	**0.568**	**0.474**	0.176056	0.21097	1.645	0.021224	0.000450475
Lung Alv./Bronch Adenoma	**1.968**	**1.471**	0.050813	0.067981	1.645	0.010436	0.000108919
Mammary Adenocarc/acanth	**2.219**	**1.679**	0.045065	0.059559	1.645	0.008811	7.76318E-05
Ovary benign Granulosa Cell Tumor	**11.4**	**6.48**	0.008772	0.015432	1.645	0.004049	1.63923E-05
Cumulative Variance =	0.000653						
Cumulative s.d. =	0.025562						
Sum of Central tendency Risks=	0.280707						
Upper-bound on cum risk=	**0.322756**	(mg/kg-day)-1	**CANCER SLOPE FACTOR** FOR **FOUR** Combined SITES				
Harderian Gland Adenoma	**0.568**	**0.474**	0.176056	0.21097	1.645	0.021224	0.000450475
Lung Alv./Bronch Adenoma	**1.968**	**1.471**	0.050813	0.067981	1.645	0.010436	0.000108919
Mammary Adenocarc/acanth	**2.219**	**1.679**	0.045065	0.059559	1.645	0.008811	7.76318E-05
Ovary benign Granulosa Cell Tumor	**11.4**	**6.48**	0.008772	0.015432	1.645	0.004049	1.63923E-05
Skin Fibroma/Sarcoma	**3.05**	**2.41**	0.032787	0.041494	1.645	0.005293	2.80152E-05
Cumulative Variance =	0.000681						
Cumulative s.d. =	0.026104						
Sum of Central tendency Risks=	0.313494						
Upper-bound on cum risk=	**0.356435**	(mg/kg-day)-1	**CANCER SLOPE FACTOR** FOR **FIVE** Combined SITES				
Harderian Gland Adenoma	**0.568**	**0.474**	0.176056	0.21097	1.645	0.021224	0.000450475
Lung Alv./Bronch Adenoma	**1.968**	**1.471**	0.050813	0.067981	1.645	0.010436	0.000108919
Mammary Adenocarc/acanth	**2.219**	**1.679**	0.045065	0.059559	1.645	0.008811	7.76318E-05
Ovary benign Granulosa Cell Tumor	**11.4**	**6.48**	0.008772	0.015432	1.645	0.004049	1.63923E-05
Skin Fibroma/Sarcoma	**3.05**	**2.41**	0.032787	0.041494	1.645	0.005293	2.80152E-05
Stomach (fore) Squam. Cell Pappil.	**7.623**	**5.045**	0.013118	0.019822	1.645	0.004075	1.66058E-05
Cumulative Variance =	0.000698						
Cumulative s.d. =	0.026420						
Sum of Central tendency Risks=	0.326612						
Upper-bound on cum risk=	**0.370073**	(mg/kg-day)-1	**CANCER SLOPE FACTOR** FOR **SIX** Combined SITES				

Table I.D.1. Animal and Human Oral Cancer Slope (Potency) Estimates for Acrylamide based on Tumor Results in the NTP (2012) Rat and Mouse 2-Year Study Bioassay. Estimates for those Tumor sites NTP judged as "Clear Evidence of Carcingenic Activity" or "Related." Animal Cancer slope calculated using U.S. EPA's BMDS Version 2.4, plus EPA's method for calculating Combined Risks. Human Cancer slope factors use adaption of Cal EPA's animal-to-human scaling factors for rats, and U.S. EPA's default procedures for the mouse.

Sex/Species	ORGAN	BMDL	Animal Canc Slope factor	Scaling Factor	Human Cancer Slope Factor
M/mouse	Harderian Adenoma	0.32	0.313	6.9	**2.154**
M/mouse	Lung Adeno	1.57	0.064	6.9	**0.439**
M/mouse	Stomach	3.094	0.032	6.9	**0.223**
M/mouse	**Combined Sites**		0.381	6.9	**2.628**
F/mouse	Harderian Adeno	0.474	0.211	7.3	**1.535**
F/mouse	Lung Adeno	1.471	0.068	7.3	**0.495**
F/mouse	Stomach	5.045	0.020	7.3	**0.144**
F/mouse	Mammary Adcarc/acnatho	1.679	0.060	7.3	**0.433**
F/mouse	Ovary granulosa	6.48	0.015	7.3	**0.112**
F/mouse	Skin fibroma/sarcoma	2.41	0.041	7.3	**0.302**
F/mouse	**Combined Sites**		0.370	7.3	**2.692**
M/Rat	Thyroid Gland foll. Ad/Ca.	0.889	0.112	2.8	**0.315**
M/Rat	Heart Schwannoma	1.304	0.077	2.8	**0.215**
M/Rat	Epidy testis/mesothel.	1.21	0.083	2.8	**0.231**
M/Rat	Pancreas Islets Adeno.	1.59	0.063	2.8	**0.176**
M/Rat	**Combined Sites**		0.262	2.8	**0.733**
F/Rat	Thyroid Gland foll. Ad/Ca.	2.328	0.043	3.0	**0.129**
F/Rat	Mammary Fibroadenoma	0.441	0.227	3.0	**0.680**
F/Rat	Oral Mucosa/tongue	2.019	0.050	3.0	**0.149**
F/Rat	Skin fibroma/sarcoma	3.084	0.032	3.0	**0.097**
F/Rat	Clitoral gland carcinoma				
F/Rat	**Combined Sites**		0.309189	3.0	**0.928**

Table I.D.2. Estimates of Human Cancer Slope Risk Factors From Daily Oral Exposure to Acrylamide in Coffee, based on Individual Tumor Sites and Summed Risks from Multiple Sites Combined for Three Rat Bioassays and One Mouse Bioassay, both Sexes separately. Estimates for NTP Rat study based on CAL EPA animal-to-human weight and dose scaling factors.

	Johnson. (1986) **RATS**[A]		Friedman (1995) **Rats**[A]		NTP (2012) **RATS**		NTP (2012) **MOUSE**		
Human Cancer Potency Factors - per (mg/kg b.w.-day).									
Tumor Site Rats	**Females**	**Males**	**Females**	**Males**	**Females**	**Males**	**Tumor Sites Mice**	**Females**	**Males**
Mammary	1.0	--	0.4	--	0.68	--	Mammary	0.43	--
Cent. Nervous. Sys.	0.14	--	0.71	0.13	--	--	Harderian	1.53	2.15
Thyroid follicular	0.24	0.32	0.33	0.44	0.13	0.31	Fore-stomach	0.14	0.22
Testis	--	0.58	--	0.40	--	0.23	Lung alv/br. A/C	0.99	0.44
Heart Schwannoma Ppancreas Islet Tumors	--	--	--	--	--	0.21 0.18	--	--	--
Oral Cavity	0.31	--	--	--	0.15	--	--	--	--
Uterus	0.15	--	--	--	--	--	Ovary	0.11	--
Clitoral Gland	0.26	--	--	--	--	--	Clitoral Gland	Not calc.	--
skin	--	--	--		0.09		Skin	0.30	
Mult. Sites Combined.	1.5	0.75	0.69	0.77	0.93	0.73	Multi. Sties Comb.	2.69	2.63
Geo. Mean Risk Factor	0.70 (CAL/OEHHA, 2005)				0.82			2.66	

A. Human Cancer Potency Factors based on the Johnson (1986) and Friedman (1995) studies are from Cal OEHHA (2005, Exhibit #23930. Cal EPA and U.S. EPA methods for Estimating risks from Combined tumor sites judged quantitatively similar..

Table IV.A.1 Estimates of **Lifetime Increased Risk of Cancer from consuming Acrylamide in Coffee**, based on Extrapolation of Tumor Results in the NTP Rat and Mice Bioassays, and consumption of from 1-9 Cups of Coffee/day from age 16+. Comparison of my (S.B.) Estimates with Those of the U.S. EPA (2010; Plaintiff's Exhibit #48631) and CAL. EPA (2005; Plaintiff's Exhibit #23930). All Human Cancer Slope Factors first adjusted downward (by 54/70 years) to account for little coffee drinking during < 16 years. Estimates of the **NSRL** (amount AA from Coffee/day for a 1 in 100,000 risk of cancer.) [A].

Human Cancer Potency Est. from:	ADJ. Human Cancer Factor	Coffee Consumption (Cups/day)[B] 1	3.1	5	7	9	**NSR**L (1.0E-5 risk) Based on Study Cancer Potency Estimates
	(mg/kg-day)$^{-1}$	Acrylamide Content (μg/day) 2.22	6.85	11.11	15.56	20.01	
Cal.EPA	0.540	1.72E-5	5.28E-5	8.58E-5	1.2E-4	1.54E-4	1.30 μg/day
U.S. EPA	0.386	1.23E-5	3.77E-5	6.13E-5	8.58E-5	1.10E-4	1.81 μg/day
NTP RAT (S. B.)	0.633	2.01E-5	6.19E-5	1.00E-4	1.41E-4	1.81E-4	1.11 μg/day
NTP MOUSE (S.B.)[C]	**2.05**	**6.52E-5**	**2.01E-4**	**3.26E-4**	**4.56E-4**	**8.07E-4**	**0.34 μg/day**

A. All Estimates of the NSRL are less than the amount of AA in 1 cup of coffee. Higher increased cancer risks are seen from more coffee consumption per day.

B. Estimated Percentiles based on the NCA's Survey of 428 coffee drinkers age 18+ who lived in the "West". 1 Cup: 9th %ile; 3.1 Cups: 48th %ile; 5 cups: 71st %ile; 7 cups: 85th %ile; 9 cups: 91st %ile.

C. "Most Sensitive Study." See Discussion in Section VI. Conclusion and Opinion

Table V.A.1. Margin-of-Exposure Ratios (M.O.E.'s for $BMDL_{10}$ for Combined Tumor Cancer Potency Estimates in the NTP (2012) Rat and Mouse 2-year bioassays to the amount of AA in 1, 3.1, 5, 7, and 9 cups of coffee/day. $BMDL_{10}$'s and AA Amounts/Cup of Coffee Estimates are in Units of μg/kg –b.w. for Animals and Humans, respectively.

Sex/Species	Animal Cancer slope Factor $(mg/kg\text{-}bw\text{-}d)^{-1}$	$BMDL_{10}$ μg/kg-b.w -d	Cups of Coffee Consumed/day[A] (10.7 oz./cup) 1	3.1	5	7	9
			μg/kg-b.w. Human -day				
			0.0318	0.0979	0.1588	0.2225	0.2862
			Margin-Of-Exposure Ratios				
Male Rat	0.262	383	12017	3907	2406	1718	1335
Female Rat	0.309	323	10170	3307	2036	1453	1130
Male Mouse	0.381	262	8249	2682	1652	1179	916
Female Mouse	0.370	270	8497	2762	1702	1214	944

Estimated Percentiles based on the NCA's Survey of 428 coffee drinkers age 18+ who lived in the "West". 1 Cup: 9th %ile; 3.1 Cups: 48th %ile; 5 cups 71st %ile; 7 cups: 85th %ile; 9 cups 91st %ile.

EXHIBIT “B”

Addendum to Statement of

Steven P. Bayard **April 27, 2014**

CANCER RISK ASSESSMENT OF ACRYLAMIDE IN COFFEE <u>NOT</u> INCLUDING HARDERIAN TUMORS IN NTP MICE

TABLE I.B.3 MALE MICE (NOT INCLUDING HARDERIAN GLAND TUMORS.)									
Calculation of Summed Risks for tumors at Several Sites in **MALE B6C3F1 MICE/NOT INCLUDING HARDERIAN Tumors** exposed									
to AA in Drinking Water in the NTP Bioassay (2012; Plaintiff's Exhibit 48449). Dose Groups: 0, 1.04, 2.20, 4.11, and 8.93 mg/kg/day.									
Using BMD10 and BMDL10 output from EPA's BMDS Software 2.4 and BMDS Wizard 1.8. Calculations for									
for Combined Risks for Multiple Tumor Sites based on method in U.S. EPA Tox. Review of Acrylamide,									
Table D8 and Table D!)in Appendix D. (U.S. EPA; 2010; Plaintiff's Exhibit 48631).									
			Mouse Oral						
			Cancer Slope						
TUMOR SITE	**BMD10**	**BMDL10**	central est.		MOUSE Oral Cancer		t-statistic	s.d.	Variance
					slope factor				
	mg/kg-day	**mg/kg-day**			(upper-bound)				
			(mg/kg-day)-1		(per mg/kg-day)				
	0	**0**							
Harderian Gland Adenoma									
Lung Alv./Bronch Adenoma	**2.414**	**1.57**	0.041425		0.063694		1.645	0.013538	0.00018
Cumulative Variance =	0.00018								
Cumulative s.d. =	0.01354								
Sum of Central tendency Risks=	0.04143	(mg/kg-day)-1							
Upper-bound on cum risk=	**0.04143**	(mg/kg-day)-1		**CANCER SLOPE FACTOR** FOR TWO Combined SITES					
Harderian Gland Adenoma	**0**	**0**							
Lung Alv./Bronch Adenoma	**2.414**	**1.57**	0.041425		0.063694		1.645	0.013538	0.00018
Stomach (fore) SQ. cell Pappillor	**4.548**	**3.094**	0.021988		0.032321		1.645	0.006281	3.9E-05
Cumulative Variance =	0.000223								
Cumulative s.d. =	0.014924								
Sum of Central tendency Risks=	0.063413								
Upper-bound on cum risk=	**0.063413**	(mg/kg-day)-1		**CANCER SLOPE FACTOR** FOR THREE Combined SITES					

TABLE I.B.4. Calculation of Summed Risks for tumors in **FEMALE B6C3F1 MICE WITHOUT HARDERIAN** exposed to AA in Drinking Water in the NTP Bioassay (2012). Doses Groups: 0, 1.10, 2.23, 4.65, and 9.96 mg/kg-da. BMD10 and BMDL10 output from EPA's BMDS 2.4 and BMDS Wizard 1.8. Calculations for Combined Risks for Multiple Tumor Sites based on method in U.S. EPA Tox. Rev. of Acryl. Tables D-8 and D-10 in Appendix D. (U.S. EPA; 2010; Plaintiff's Exhibit 48631).

TUMOR SITE	BMD10	BMDL10	MOUSE cent slope factor	MOUSE U.B. slope factor	t-statis	s.d.	Variance
	mg/kg-da	mg/kg-da	(mg/kg-day)-1	(mg/kg-day)-1			
Lung Alv./Bronch Ade	**1.968**	**1.471**	0.050813	0.067981	1.645	0.010436	0.0001089
Mammary Adenocarc	**2.219**	**1.679**	0.045065	0.059559	1.645	0.008811	7.763E-05
Cumulative Variance	0.000187						
Cumulative s.d. =	0.013658						
Sum of Central tende	0.095878						
Upper-bound on cum	**0.118346**	(mg/kg-day)-1		**CANCER SLOPE FACTOR** FOR **TWO** Comb. SITES			
Lung Alv./Bronch Ade	**1.968**	**1.471**	0.050813	0.067981	1.645	0.010436	0.0001089
Mammary Adenocarc	**2.219**	**1.679**	0.045065	0.059559	1.645	0.008811	7.763E-05
Ovary benign Granul	**11.4**	**6.48**	0.008772	0.015432	1.645	0.004049	1.639E-05
Cumulative Variance	0.000203						
Cumulative s.d. =	0.014246						
Sum of Central tende	0.104650						
Upper-bound on cum	**0.128085**	(mg/kg-day)-1		**CANCER SLOPE FACTOR** FOR **3** Comb. SITES			
Lung Alv./Bronch Ade	**1.968**	**1.471**	0.050813	0.067981	1.645	0.010436	0.0001089
Mammary Adenocarc	**2.219**	**1.679**	0.045065	0.059559	1.645	0.008811	7.763E-05
Ovary benign Granul	**11.4**	**6.48**	0.008772	0.015432	1.645	0.004049	1.639E-05
Skin Fibroma/Sarcom	**3.05**	**2.41**	0.032787	0.041494	1.645	0.005293	2.802E-05
Cumulative Variance	0.000231						
Cumulative s.d. =	0.015197						
Sum of Central tende	0.137437						
Upper-bound on cum	**0.162437**	(mg/kg-day)-1		**CANCER SLOPE FACTOR** FOR **Four** Comb. SITES			
Lung Alv./Bronch Ade	**1.968**	**1.471**	0.050813	0.067981	1.645	0.010436	0.0001089
Mammary Adenocarc	**2.219**	**1.679**	0.045065	0.059559	1.645	0.008811	7.763E-05
Ovary benign Granul	**11.4**	**6.48**	0.008772	0.015432	1.645	0.004049	1.639E-05
Skin Fibroma/Sarcom	**3.05**	**2.41**	0.032787	0.041494	1.645	0.005293	2.802E-05
Stomach (fore) Squar	**7.623**	**5.045**	0.013118	0.019822	1.645	0.004075	1.661E-05
Cumulative Variance	0.000248						
Cumulative s.d. =	0.015734						
Sum of Central tende	0.150555						
Upper-bound on cum	**0.176438**	(mg/kg-day)-1		**CANCER SLOPE FACTOR** FOR **FIVE** Comb. SITES			

Table 1.D.1. WITHOUT MOUSE HARDERIAN TUMORS

Animal and Human Oral Cancer Slope (Potency) Estimates for Acrylamide based on Tumor Results in the NTP (2012) Rat and Mouse 2-Year Study Bioassay. Estimates for those Tumor sites NTP judged as "Clear Evidence of Carcingenic Activity" or "Related." Animal Cancer slope calculated using U.S. EPA's BMDS Version 2.4, plus EPA's method for calculating Combined Risks. Human Cancer slope factors use adaption of Cal EPA's animal-to-human scaling factors for rats, and U.S. EPA's default procedures for the mouse.

Sex/Species	ORGAN	BMDL	Animal Canc Slope factor	Scaling Factor	Human Cancer Slope Factor
M/mouse	Harderian Adenoma	NOT CALC.	NOT CALC.		
M/mouse	Lung Adeno	1.57	0.064	6.9	**0.439**
M/mouse	Stomach	3.094	0.032	6.9	**0.223**
M/mouse	**Combined Sites**		0.063	6.9	**0.437**
F/mouse	Harderian Adeno	NOT CALC.	NOT CALC.		
F/mouse	Lung Adeno	1.471	0.068	7.3	**0.495**
F/mouse	Stomach	5.045	0.020	7.3	**0.144**
F/mouse	Mammary Adcarc/acnatho	1.679	0.060	7.3	**0.433**
F/mouse	Ovary granulosa	6.48	0.015	7.3	**0.112**
F/mouse	Skin fibroma/sarcoma	2.41	0.041	7.3	**0.302**
F/mouse	**Combined Sites**		0.176	7.3	**1.283**
M/Rat	Thyroid Glnd foll. Ad/Ca.	0.889	0.112	2.8	**0.315**
M/Rat	Heart Schwannoma	1.304	0.077	2.8	**0.215**
M/Rat	Epidy testis/mesothel.	1.21	0.083	2.8	**0.231**
M/Rat	Pancreas Islets Adeno.	1.59	0.063	2.8	**0.176**
M/Rat	**Combined Sites**		0.262	2.8	**0.733**
F/Rat	Thyroid Gland foll. Ad/Ca.	2.328	0.043	3.0	**0.129**
F/Rat	Mammary Fibroadenoma	0.441	0.227	3.0	**0.680**
F/Rat	Oral Mucosa/tongue	2.019	0.050	3.0	**0.149**
F/Rat	Skin fibroma/sarcoma	3.084	0.032	3.0	**0.097**
F/Rat	Clitoral gland carcinoma				
F/Rat	**Combined Sites**		0.309189	3.0	**0.928**

Table I.D.1. Animal and Human Oral Cancer Slope (Potency) Estimates for Acrylamide based on Tumor Results in the NTP (2012) Rat and Mouse 2-Year Study Bioassay. Estimates for those Tumor sites NTP judged as "Clear Evidence of Carcingenic Activity" or "Related." Animal Cancer slope calculated using U.S. EPA's BMDS Version 2.4, plus EPA's method for calculating Combined Risks. Human Cancer slope factors use adaption of Cal EPA's animal-to-human scaling factors for rats, and U.S. EPA's default procedures for the mouse.

Sex/Species	ORGAN	BMDL	Animal Canc Slope factor	Scaling Factor	Human Cancer Slope Factor
M/mouse	Harderian Adenoma	0.32	0.313	6.9	**2.154**
M/mouse	Lung Adeno	1.57	0.064	6.9	**0.439**
M/mouse	Stomach	3.094	0.032	6.9	**0.223**
M/mouse	**Combined Sites**		0.381	6.9	**2.628**
F/mouse	Harderian Adeno	0.474	0.211	7.3	**1.535**
F/mouse	Lung Adeno	1.471	0.068	7.3	**0.495**
F/mouse	Stomach	5.045	0.020	7.3	**0.144**
F/mouse	Mammary Adcarc/acnatho	1.679	0.060	7.3	**0.433**
F/mouse	Ovary granulosa	6.48	0.015	7.3	**0.112**
F/mouse	Skin fibroma/sarcoma	2.41	0.041	7.3	**0.302**
F/mouse	**Combined Sites**		0.370	7.3	**2.692**
M/Rat	Thyroid Gland foll. Ad/Ca.	0.889	0.112	2.8	**0.315**
M/Rat	Heart Schwannoma	1.304	0.077	2.8	**0.215**
M/Rat	Epidy testis/mesothel.	1.21	0.083	2.8	**0.231**
M/Rat	Pancreas Islets Adeno.	1.59	0.063	2.8	**0.176**
M/Rat	**Combined Sites**		0.262	2.8	**0.733**
F/Rat	Thyroid Gland foll. Ad/Ca.	2.328	0.043	3.0	**0.129**
F/Rat	Mammary Fibroadenoma	0.441	0.227	3.0	**0.680**
F/Rat	Oral Mucosa/tongue	2.019	0.050	3.0	**0.149**
F/Rat	Skin fibroma/sarcoma	3.084	0.032	3.0	**0.097**
F/Rat	Clitoral gland carcinoma				
F/Rat	**Combined Sites**		0.309189	3.0	**0.928**

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***Table I.D.2**. Estimates of Human Cancer Slope Risk Factors From Daily Oral Exposure to Acrylamide in Coffee, based on Individual Tumor Sites* and Summed Risks from Multiple Sites Combined for Three Rat Bioassays and One Mouse Bioassay, both Sexes separately. Estimates for NTP Rat study based on CAL EPA animal-to-human weight and dose scaling factors. **INCLUDING MOUSE HARDERIAN GLAND TUMORS**

	Johnson. (1986) **RATS**[A]		Friedman (1995) **Rats**[A]		NTP(2012) **RATS**		NTP (2012) **MOUSE**		
Human Cancer Potency Factors – per (mg/kg b.w.-day).									
Tumor Site Rats	**Females**	**Males**	**Females**	**Males**	**Females**	**Males**	**Tum. Sites Mice**	**Females**	**Males**
Mammary	1.0	--	0.4	--	0.68	--	Mammary	0.43	--
Cent. Nervous. Sys.	0.14	--	0.71	0.13	--	--	Harderian	1.53	2.15
Thyroid follicular	0.24	0.32	0.33	0.44	0.13	0.31	Fore-stomach	0.14	0.22
Testis	--	0.58	--	0.40	--	0.23	Lung alv/br. A/C	0.49	0.44
Heart Schwannoma Pancreas Islet Tumors	--	--	--	--	--	0.21 0.18	--	--	--
Oral Cavity	0.31	--	--	--	0.15	--	--	--	--
Uterus	0.15	--	--	--	--	--	Ovary	0.11	--
Clitoral Gland	0.26	--	--	--	--	--	Clitoral Gland	No calc.	--
skin	--	--	--		0.09		Skin	0.30	
Mult. Sites Combined.	1.5	0.75	0.69	0.77	0.93	0.73	Multi Sties Comb	2.69	2.63
Geo. Mean Risk Factor	0.70 (CAL/OEHHA, 2005)				0.82			2.66	

A. Human Cancer Potency Factors based on the Johnson (1986) and Friedman (1995) studies are from Cal OEHHA (2005, Exhibit #23930. Cal EPA and U.S. EPA methods for Estimating risks from Combined tumor sites judged quantitatively similar..

***Table I.D.2**. Estimates of Human Cancer Slope Risk Factors From Daily Oral Exposure to Acrylamide in Coffee, based on Individual Tumor Sites* and Summed Risks from Multiple Sites Combined for Three Rat Bioassays and One Mouse Bioassay, both Sexes separately. Estimates for NTP Rat study based on CAL EPA animal-to-human weight and dose scaling factors. MOUSE WITHOUT HARDERIAN TUMORS UNCLUDED

	Johnson. (1986) **RATS**[A]		Friedman (1995) **Rats**[A]		NTP (2012) **RATS**		NTP (2012) **MOUSE**		
Human Cancer Potency Factors – per (mg/kg b.w.-day).									
Tumor Site Rats	**Females**	**Males**	**Females**	**Males**	**Females**	**Males**	**Tum. Sites Mice**	**Females**	**Males**
Mammary	1.0	--	0.4	--	0.68	--	Mammary	0.43	--
Cent. Nervous. Sys.	0.14	--	0.71	0.13	--	--	Harderian	N.C.	N.C.
Thyroid follicular	0.24	0.32	0.33	0.44	0.13	0.31	Fore-stomach	0.14	0.22
Testis	--	0.58	--	0.40	--	0.23	Lung alv/br. A/C	0.49	0.44
Heart Schwannoma Pancreas Islet Tumors	--	--	--	--	--	0.21 0.18	--	--	--
Oral Cavity	0.31	--	--	--	0.15	--	--	--	--
Uterus	0.15	--	--	--	--	--	Ovary	0.11	--
Clitoral Gland	0.26	--	--	--	--	--	Clitoral Gland	Not cal.	--
skin	--	--	--		0.09		Skin	0.30	
Mult. Sites Combined.	1.5	0.75	0.69	0.77	0.93	0.73	Multi Sites Comb	1.28	0.44
Geo. Mean Risk Factor	0.70 (CAL/OEHHA, 2005)				0.82			0.75	

A. Human Cancer Potency Factors based on the Johnson (1986) and Friedman (1995) studies are from Cal OEHHA (2005, Exhibit #23930. Cal EPA and U.S. EPA methods for Estimating risks from Combined tumor sites judged quantitatively similar.

ASSUMES NO HARDERIAN GLAND TUMORS IN MICE

Table IV.A.1 Estimates of **Lifetime Increased Risk of Cancer from consuming Acrylamide in Coffee**, based on Extrapolation of Tumor Results in the NTP Rat and Mice Bioassays, and consumption of from 1-9 Cups of Coffee/day from age 16+. Comparison of my (S.B.) Estimates with Those of the U.S. EPA (2010; Plaintiff's Exhibit #48631) and CAL. EPA (2005; Plaintiff's Exhibit #23930). All Human Cancer Slope Factors first adjusted downward (by 54/70 years) to account for little coffee drinking during < 16 years. Estimates of the **NSRL** (amount AA from Coffee/day for a 1 in 100,000 risk of cancer.) [A].

Human Cancer Potency Est. from:	ADJ. Human Cancer Factor	Coffee Consumption (Cups/day)[B] 1	3.1	5	7	9	**NSR**L (1.0E-5 risk) Based on Study Cancer Potency Estimates
	$(mg/kg\text{-}day)^{-1}$	Acrylamide Content (μg/day) 2.22	6.85	11.11	15.56	20.01	
Cal.EPA	0.540	1.72E-5	5.28E-5	8.58E-5	1.2E-4	1.54E-4	1.30 μg/day
U.S. EPA	0.386	1.23E-5	3.77E-5	6.13E-5	8.58E-5	1.10E-4	1.81 μg/day
NTP RAT (S. B.)	0.633	2.01E-5	6.19E-5	1.00E-4	1.41E-4	1.81E-4	1.11 μg/day
ASSUMES NO MOUSE HARDERIAN GLAND TUMORS							
NTP MOUSE (S.B.)[D,E]	0.58	1.84E-5	5.66E-5	9.99E-5	1.29E-4	1.65E-4	1.21 μg/day
FEM. Mouse (NO Harderian) [C,D]	**0.987**	**3.14E-5**	**9.66E-5**	**1.57E-4**	**2.20E-4**	**2.82E-4**	**0.71 μg/day**
MALE Mouse(**NO** HARDERIAN)[D]	0.339	1.08E-5	3.32E-5	5.39E-5	7.55E-5	9.70E-5	2.06 μg/day

A. All Estimates of the NSRL are less than the amount of AA in 1 cup of coffee. Higher increased cancer risks are seen from more coffee consumption per day.

B. Estimated Percentiles based on the NCA's Survey of 428 coffee drinkers age 18+ who lived in the "West". 1 Cup: 9th %ile; 3.1 Cups: 48th %ile; 5 cups: 71st %ile; 7 cups: 85th %ile; 9 cups: 91st %ile.

C. "Most Sensitive Study." See Discussion in Section VI. Conclusion and Opinion .

D. Assumes NO HARDERIAN TUMORS in MICE

E. Geometric mean of Combined Tumor Risks based on NTP Female and Male mouse, **WITHOUT Harderian Tumors**

Table IV.A.1 Estimates of **Lifetime Increased Risk of Cancer from consuming Acrylamide in Coffee**, based on Extrapolation of Tumor Results in the NTP Rat and Mice Bioassays, and consumption of from 1-9 Cups of Coffee/day from age 16+. Comparison of my (S.B.) Estimates with Those of the U.S. EPA (2010; Plaintiff's Exhibit #48631) and CAL. EPA (2005; Plaintiff's Exhibit #23930). All Human Cancer Slope Factors first adjusted downward (by 54/70 years) to account for little coffee drinking during < 16 years. Estimates of the **NSRL** (amount AA from Coffee/day for a 1 in 100,000 risk of cancer.) [A].

Human Cancer Potency Est. from:	ADJ. Human Cancer Factor	Coffee Consumption (Cups/day)[B] 1	3.1	5	7	9	**NSR**L (1.0E-5 risk) Based on Study Cancer Potency Estimates
	$(mg/kg\text{-}day)^{-1}$	Acrylamide Content (μg/day) 2.22	6.85	11.11	15.56	20.01	
Cal.EPA	0.540	1.72E-5	5.28E-5	8.58E-5	1.2E-4	1.54E-4	1.30 μg/day
U.S. EPA	0.386	1.23E-5	3.77E-5	6.13E-5	8.58E-5	1.10E-4	1.81 μg/day
NTP RAT (S. B.)	0.633	2.01E-5	6.19E-5	1.00E-4	1.41E-4	1.81E-4	1.11 μg/day
NTP MOUSE (S.B.)[C]	**2.05**	**6.52E-5**	**2.01E-4**	**3.26E-4**	**4.56E-4**	**8.07E-4**	**0.34 μg/day**

A. All Estimates of the NSRL are less than the amount of AA in 1 cup of coffee. Higher increased cancer risks are seen from more coffee consumption per day.

B. Estimated Percentiles based on the NCA's Survey of 428 coffee drinkers age 18+ who lived in the "West". 1 Cup: 9th %ile; 3.1 Cups: 48th %ile; 5 cups: 71st %ile; 7 cups: 85th %ile; 9 cups: 91st %ile.

C. "Most Sensitive Study." See Discussion in Section VI. Conclusion and Opinion .

EXHIBIT “C”

Statement of Steven Bayard, Ph.D. 04/28/14

On the "Cancer Risk Assessment of Acrylamide in Coffee" that I have submitted in this case, I have discovered some typos and errors. None of these errors affects my calculations or conclusions. The list follows:

Page. Line:	Change
1. 12th line from the bottom	Change "mutagenic" to "genotoxic"
4. 12th line	Add: close quotes to "cancer-multistage models.
5. line 2	Change to: Table 1.1 A-U.
5. line 7	Change to: probability, P>0.10. However, for the cancer multistage family, the fit need only attain P>0.05,
5. end of 1st paragraph	Change to: statistics,
5. end of 2nd paragraph	Add:)
6. line 9.	Change to: 1.B.1 – 1.B.4,
6 . 9th line from bottom	Add: close quotes after species
8. Section III. 3rd line	Change to: Excel
8. Section IV. 1st line	Change to: 7.35 μg/kg
9. 1st line	Change to: 7.35 μg/kg
9. 5th line	Change to: 1, 3.1, 5, 7, and
10. 7th line	Add: parentheses around last (mg/kg-b.w. humans.)
22. Table J. 6th line	Change: 0.0556 to 0.0637 in the four columns.
27. Table O.	Delete the Table: It duplicates Table N on page 26.
28. Table P.	Delete the Table: It was an earlier version of Table Q.
34. Table I.2 Title	Delete: , plus EPA's method for Calculating Combined Risks
40 Table 1.D.2. 6th row	Change: 0.99 to 0.49

Steven Bayard

Supplemental Table 1. Comparison of U. S. EPA's BMDS 2.4 Estimates of $BMDL_{10}$ for Male and Female B6c3F1 Mouse Harderian Gland Tumors Based on Log-Logistic, Log-Probit, and Linear Multistage Models (Multi1), Including Estimate with the Linear Multistage for the fit with the Highest Dose Dropped. Units are (mg/kg-b.w.) for the Mouse. Data from the NTP (2012) Study for Acrylamide in Drinking Water.

Sex	Model			
	Log-Logistic	Log-Probit	Multi 1	Multi1- High Dose Dropped
Female	0.282	0.230	0.474[A]	0.366[B]
Male	0.173	0.159	0.320[C]	0.252

A. Table I.L in my Risk Assessment. Chosen Model result.

B. Table I.M in my Risk Assessment. Chosen Model result.

C. Table I.I in my Risk Assessment. Chosen Model result.

EXHIBIT “D”

Addendum to Statement of
Steven P. Bayard

Recalculations for Revised Exhibit #3

Corrected 5/25/2014

CANCER RISK ASSESSMENT OF ACRYLAMIDE IN COFFEE
NOT INCLUDING HARDERIAN TUMORS IN NTP MICE

(Corrected 05/25/2014) **TABLE I.B.3 MALE MICE (NOT INCLUDING HARDERIAN GLAND TUMORS.)**

Calculation of Summed Risks for tumors at Several Sites in **MALE B6C3F1 MICE/NOT INCLUDING HARDERIAN Tumors** exposed to AA in Drinking Water in the NTP Bioassay (2012; Plaintiff's Exhibit 48449). Dose Groups: 0, 1.04, 2.20, 4.11, and 8.93 mg/kg/day. Using BMD10 and BMDL10 output from EPA's BMDS Software 2.4 and BMDS Wizard 1.8. Calculations for for Combined Risks for Multiple Tumor Sites based on method in U.S. EPA Tox. Review of Acrylamide, Table D8 and Table D10) in Appendix D. (U.S. EPA; 2010; Plaintiff's Exhibit 48631).

TUMOR SITE	**BMD10** mg/kg-day	**BMDL10** mg/kg-day	**Mouse Oral Cancer Slope central est.** $(mg/kg\text{-}day)^{-1}$	**MOUSE Oral Cancer slope factor (upper-bound)** $(mg/kg\text{-}day)^{-1}$	t-statistic	standard deviation	Variance
Harderian Gland Adenoma	**NOT CALCULATED**						
Lung Alv./Bronch Adenoma	**2.414**	**1.57**	0.041425	0.063694	1.645	0.013538	0.00018323
Cumulative Variance =	0.00018						
Cumulative s.d. =	0.01354						
Sum of Central tendency Risks=	0.04143	$(mg/kg\text{-}day)^{-1}$					
Upper-bound on cum risk=	**0.06369**	$(mg/kg\text{-}day)^{-1}$		**CANCER SLOPE FACTOR** FOR **ONE** SITE			
Harderian Gland Adenoma	**NOT CALCULATED**						
Lung Alv./Bronch Adenoma	**2.414**	**1.57**	0.041425	0.063694	1.645	0.013538	0.0001833
Stomach (fore) SQ. cell Pappilloma	**4.548**	**3.094**	0.021988	0.032321	1.645	0.006281	3.94562E-05
Cumulative Variance =	0.000223						
Cumulative s.d. =	0.014924						
Sum of Central tendency Risks=	0.063413						
Upper-bound on cum risk=	**0.087962**	(mg/kg-day)-1		**CANCER SLOPE FACTOR** FOR **TWO** Combined SITES			

TABLE I.B.4. Calculation of Summed Risks for tumors in **FEMALE B6C3F1 MICE WITHOUT HARDERIAN** exposed to AA in Drinking Water in the NTP Bioassay (2012). Doses Groups: 0, 1.10, 2.23, 4.65, and 9.96 mg/kg-da. BMD10 and BMDL10 output from EPA's BMDS 2.4 and BMDS Wizard 1.8. Calculations for Combined Risks for Multiple Tumor Sites based on method in U.S. EPA Tox. Rev. of Acryl. Tables D-8 and D-10 in Appendix D. (U.S. EPA; 2010; Plaintiff's Exhibit 48631).

TUMOR SITE	BMD10	BMDL10	MOUSE cent slope factor	MOUSE U.B. slope factor	t-statis	s.d.	Variance
	mg/kg-da	mg/kg-da	(mg/kg-day)-1	(mg/kg-day)-1			
Lung Alv./Bronch Ade	**1.968**	**1.471**	0.050813	0.067981	1.645	0.010436	0.0001089
Mammary Adenocarc	**2.219**	**1.679**	0.045065	0.059559	1.645	0.008811	7.763E-05
Cumulative Variance	0.000187						
Cumulative s.d. =	0.013658						
Sum of Central tende	0.095878						
Upper-bound on cum	**0.118346**	(mg/kg-day)-1		**CANCER SLOPE FACTOR** FOR **TWO** Comb. SITES			
Lung Alv./Bronch Ade	**1.968**	**1.471**	0.050813	0.067981	1.645	0.010436	0.0001089
Mammary Adenocarc	**2.219**	**1.679**	0.045065	0.059559	1.645	0.008811	7.763E-05
Ovary benign Granul	**11.4**	**6.48**	0.008772	0.015432	1.645	0.004049	1.639E-05
Cumulative Variance	0.000203						
Cumulative s.d. =	0.014246						
Sum of Central tende	0.104650						
Upper-bound on cum	**0.128085**	(mg/kg-day)-1		**CANCER SLOPE FACTOR** FOR **3** Comb. SITES			
Lung Alv./Bronch Ade	**1.968**	**1.471**	0.050813	0.067981	1.645	0.010436	0.0001089
Mammary Adenocarc	**2.219**	**1.679**	0.045065	0.059559	1.645	0.008811	7.763E-05
Ovary benign Granul	**11.4**	**6.48**	0.008772	0.015432	1.645	0.004049	1.639E-05
Skin Fibroma/Sarcom	**3.05**	**2.41**	0.032787	0.041494	1.645	0.005293	2.802E-05
Cumulative Variance	0.000231						
Cumulative s.d. =	0.015197						
Sum of Central tende	0.137437						
Upper-bound on cum	**0.162437**	(mg/kg-day)-1		**CANCER SLOPE FACTOR** FOR **Four** Comb. SITES			
Lung Alv./Bronch Ade	**1.968**	**1.471**	0.050813	0.067981	1.645	0.010436	0.0001089
Mammary Adenocarc	**2.219**	**1.679**	0.045065	0.059559	1.645	0.008811	7.763E-05
Ovary benign Granul	**11.4**	**6.48**	0.008772	0.015432	1.645	0.004049	1.639E-05
Skin Fibroma/Sarcom	**3.05**	**2.41**	0.032787	0.041494	1.645	0.005293	2.802E-05
Stomach (fore) Squar	**7.623**	**5.045**	0.013118	0.019822	1.645	0.004075	1.661E-05
Cumulative Variance	0.000248						
Cumulative s.d. =	0.015734						
Sum of Central tende	0.150555						
Upper-bound on cum	**0.176438**	(mg/kg-day)-1		**CANCER SLOPE FACTOR** FOR **FIVE** Comb. SITES			

Corrected (5/25/22014) Table 1.D.1. WITHOUT MOUSE HARDERIAN TUMORS

Animal and Human Oral Cancer Slope (Potency) Estimates for Acrylamide based on Tumor Results in the NTP (2012) Rat and Mouse 2-Year Study Bioassays. Estimates for those Tumor sites NTP judged as "Clear Evidence of Carcinogenic Activity" or "Related." Animal Cancer slope calculated using U.S. EPA's BMDS Version 2.4, plus EPA's method for calculating Combined Risks. Human Cancer slope factors use adaption of Cal EPA's animal-to-human scaling factors for rats, and U.S. EPA's default scaling factors for the mouse.

Sex/Species	ORGAN	BMDL10	Animal Cancer Slope factor	Scaling Factor	Human Cancer Slope Factor
M/mouse	Harderian Adenoma	NOT CALC.	NOT CALC.		**0**
M/mouse	Lung Adenoma	1.57	0.064	6.9	**0.439**
M/mouse	Stomach	3.094	0.032	6.9	**0.223**
M/mouse	**Combined Sites**		0.088	6.9	**0.607**
F/mouse	Harderian Adenoma	NOT CALC.	NOT CALC.		**0**
F/mouse	Lung Adenoma	1.471	0.068	7.3	**0.495**
F/mouse	Stomach	5.045	0.020	7.3	**0.144**
F/mouse	Mammary Adcarc/acnatho	1.679	0.060	7.3	**0.433**
F/mouse	Ovary granulosa	6.48	0.015	7.3	**0.112**
F/mouse	Skin fibroma/sarcoma	2.41	0.041	7.3	**0.302**
F/mouse	**Combined Sites**		0.176	7.3	**1.283**
M/Rat	Thyroid Glnd foll. Ad/Ca.	0.889	0.112	2.8	**0.315**
M/Rat	Heart Schwannoma	1.304	0.077	2.8	**0.215**
M/Rat	Epidy testis/mesothelioma	1.21	0.083	2.8	**0.231**
M/Rat	Pancreas Islets Adenoma	1.59	0.063	2.8	**0.176**
M/Rat	**Combined Sites**		0.262	2.8	**0.733**
F/Rat	Thyroid Gland foll. Ad/Ca.	2.328	0.043	3.0	**0.129**
F/Rat	Mammary Fibroadenoma	0.441	0.227	3.0	**0.680**
F/Rat	Oral Mucosa/tongue	2.019	0.050	3.0	**0.149**
F/Rat	Skin fibroma/sarcoma	3.084	0.032	3.0	**0.097**
F/Rat	Clitoral gland carcinoma	NOT CALC.			**0**
F/Rat	**Combined Sites**		0.309189	3.0	**0.928**

Table I.D.1. Animal and Human Oral Cancer Slope (Potency) Estimates for Acrylamide based on Tumor Results in the NTP (2012) Rat and Mouse 2-Year Study Bioassay. Estimates for those Tumor sites NTP judged as "Clear Evidence of Carcingenic Activity" or "Related." Animal Cancer slope calculated using U.S. EPA's BMDS Version 2.4, plus EPA's method for calculating Combined Risks. Human Cancer slope factors use adaption of Cal EPA's animal-to-human scaling factors for rats, and U.S. EPA's default procedures for the mouse.

			Animal Canc	Scaling		Human Cancer
Sex/Species	**ORGAN**	**BMDL**	**Slope factor**	**Factor**		**Slope Factor**
M/mouse	Harderian Adenoma	0.32	0.313	6.9		**2.154**
M/mouse	Lung Adeno	1.57	0.064	6.9		**0.439**
M/mouse	Stomach	3.094	0.032	6.9		**0.223**
M/mouse	**Combined Sites**		0.381	6.9		**2.628**
F/mouse	Harderian Adeno	0.474	0.211	7.3		**1.535**
F/mouse	Lung Adeno	1.471	0.068	7.3		**0.495**
F/mouse	Stomach	5.045	0.020	7.3		**0.144**
F/mouse	Mammary Adcarc/acnatho	1.679	0.060	7.3		**0.433**
F/mouse	Ovary granulosa	6.48	0.015	7.3		**0.112**
F/mouse	Skin fibroma/sarcoma	2.41	0.041	7.3		**0.302**
F/mouse	**Combined Sites**		0.370	7.3		**2.692**
M/Rat	Thyroid Gland foll. Ad/Ca.	0.889	0.112	2.8		**0.315**
M/Rat	Heart Schwannoma	1.304	0.077	2.8		**0.215**
M/Rat	Epidy testis/mesothel.	1.21	0.083	2.8		**0.231**
M/Rat	Pancreas Islets Adeno.	1.59	0.063	2.8		**0.176**
M/Rat	**Combined Sites**		0.262	2.8		**0.733**
F/Rat	Thyroid Gland foll. Ad/Ca.	2.328	0.043	3.0		**0.129**
F/Rat	Mammary Fibroadenoma	0.441	0.227	3.0		**0.680**
F/Rat	Oral Mucosa/tongue	2.019	0.050	3.0		**0.149**
F/Rat	Skin fibroma/sarcoma	3.084	0.032	3.0		**0.097**
F/Rat	Clitoral gland carcinoma					
F/Rat	**Combined Sites**		0.309189	3.0		**0.928**

Table I.D.2. Estimates of Human Cancer Slope Risk Factors From Daily Oral Exposure to Acrylamide in Coffee, based on Individual Tumor Sites and Summed Risks from Multiple Sites Combined for Three Rat Bioassays and One Mouse Bioassay, both Sexes separately. Estimates for NTP Rat study based on CAL EPA animal-to-human weight and dose scaling factors.

	Johnson. (1986) **RATS**[A]		Friedman (1995) **Rats**[A]		NTP(2012) **RATS**		NTP (2012) **MOUSE**		
Human Cancer Potency (Slope) Factors - per (mg/kg b.w.-day).									
Tumor Site Rats	**Females**	**Males**	**Females**	**Males**	**Females**	**Males**	**Tum. Sites Mice**	**Females**	**Males**
Mammary	1.0	--	0.4	--	0.68	--	Mammary	0.43	--
Cent. Nervous. Sys.	0.14	--	0.71	0.13	--	--	Harderian	1.53	2.15
Thyroid follicular	0.24	0.32	0.33	0.44	0.13	0.31	Fore-stomach	0.14	0.22
Testis	--	0.58	--	0.40	--	0.23	Lung alv/br. A/C	0.0.49	0.44
Heart Schwannoma Pancreas Islet Tumors	--	--	--	--	--	0.21 0.18	--	--	--
Oral Cavity	0.31	--	--	--	0.15	--	--	--	--
Uterus	0.15	--	--	--	--	--	Ovary	0.11	--
Clitoral Gland	0.26	--	--	--	--	--	Clitoral Gland	No calc.	--
skin	--	--	--		0.09		Skin	0.30	
Mult. Sites Combined.	1.5	0.75	0.69	0.77	0.93	0.73	Multi Sties Comb	2.69	2.63
Geo. Mean Slope Factor	0.70 (CAL EPA/OEHHA, 2005)				0.82			2.66	

A. Human Cancer Potency or Slope Factors based on the Johnson (1986) and Friedman (1995) studies are from Cal OEHHA (2005, Exhibit #23930. Cal EPA and U.S. EPA methods for Estimating risks from Combined tumor sites judged quantitatively similar..

Table I.D.2. Estimates of Human Cancer Slope Risk Factors From Daily Oral Exposure to Acrylamide in Coffee, based on Individual Tumor Sites and Summed Risks from Multiple Sites Combined for Three Rat Bioassays and One Mouse Bioassay, both Sexes separately. Estimates for NTP Rat study based on CAL EPA animal-to-human weight and dose scaling factors. **ASSUMES MOUSE WITHOUT HARDERIAN TUMORS INCLUDED**

	Johnson. (1986) **RATS**[A]		Friedman (1995) **Rats**[A]		NTP (2012) **RATS**		NTP (2012) **MOUSE**		
Human Cancer Potency Factors - per (mg/kg b.w.-day).									
Tumor Site Rats	**Females**	**Males**	**Females**	**Males**	**Females**	**Males**	**Tum. Sites Mice**	**Females**	**Males**
Mammary	1.0	--	0.4	--	0.68	--	Mammary	0.43	--
Cent. Nervous. Sys.	0.14	--	0.71	0.13	--	--	Harderian	N.C.	N.C.
Thyroid follicular	0.24	0.32	0.33	0.44	0.13	0.31	Fore-stomach	0.14	0.22
Testis	--	0.58	--	0.40	--	0.23	Lung alv/br. A/C	0.49	0.44
Heart Schwannoma Pancreas Islet Tumors	--	--	--	--	--	0.21 0.18	--	--	--
Oral Cavity	0.31	--	--	--	0.15	--	--	--	--
Uterus	0.15	--	--	--	--	--	Ovary	0.11	--
Clitoral Gland	0.26	--	--	--	--	--	Clitoral Gland	Not cal.	--
skin	--	--	--		0.09		Skin	0.30	
Multiple Sites Combined	1.5	0.75	0.69	0.77	0.93	0.73	Multi Sites Comb	1.28	0.61
Geo. Mean Slope Factor	0.70 (CAL EPA/OEHHA, 2005)				0.82			0.88	

A. Human Cancer Potency Factors based on the Johnson (1986) and Friedman (1995) studies are from Cal OEHHA (2005, Exhibit #23930. Cal EPA and U.S. EPA methods for Estimating risks from Combined tumor sites judged quantitatively similar.

ASSUMES NO HARDERIAN GLAND TUMORS IN MICE

Table IV.A.1 Estimates of **Lifetime Increased Risk of Cancer from consuming Acrylamide in Coffee**, based on Extrapolation of Tumor Results in the NTP Rat and Mice Bioassays, and consumption of from 1-9 Cups of Coffee/day from age 16+. Comparison of my (S.B.) Estimates with Those of the U.S. EPA (2010; Plaintiff's Exhibit #48631) and CAL. EPA (2005; Plaintiff's Exhibit #23930). All Human Cancer Slope Factors first adjusted downward (by 54/70 years) to account for little coffee drinking during < 16 years of age. Estimates of the **NSRL** (amount AA from Coffee/day for 54 years for a 1 in 100,000 risk of cancer.) [A]

Human Cancer Slope Factor. from:	ADJ. Human Cancer Slope Factor	Coffee Consumption (Cups/day)[B] 1	3.1	5	7	9	**NSR**L (1.0E-5 risk) Based on Adj.Human Cancer Slope Factors
	$(mg/kg\text{-}day)^{-1}$	Acrylamide Content (μg/day) 2.22	6.85	11.11	15.56	20.01	
Cal.EPA	0.540	1.72E-5	5.28E-5	8.58E-5	1.2E-4	1.54E-4	1.30 μg/day
U.S. EPA	0.386	1.23E-5	3.77E-5	6.13E-5	8.58E-5	1.10E-4	1.81 μg/day
NTP RAT (S. B.)	0.633	2.01E-5	6.19E-5	1.00E-4	1.41E-4	1.81E-4	1.11 μg/day
NTP MOUSE Comb.[D,E]: (S.B.)	0.679	2.16E-5	6.64E-5	1.17E-4	1.51E-4	1.94E-4	1.03 μg/day
FEM. Mouse (NO Harderian) [C,D]	**0.987**	**3.14E-5**	**9.66E-5**	**1.57E-4**	**2.20E-4**	**2.82E-4**	**0.71 μg/day**
MALE Mouse(**NO** HARDERIAN)[D]	0.468	1.49E-5	4.58E-5	7.44E-5	1.05E-4	1.34E-4	1.49 μg/day

A. All Estimates of the NSRL are less than the amount of AA in 1 cup of coffee. Higher increased cancer risks are seen from more coffee consumption per day.

B. Estimated Percentiles based on the NCA's Survey of 428 coffee drinkers age 18+ who lived in the "West". 1 Cup: 9th %ile; 3.1 Cups: 48th %ile; 5 cups: 71st %ile; 7 cups: 85th %ile; 9 cups: 91st %ile.

C. "Most Sensitive Study." See Discussion in Section VI. Conclusions and Opinion .

D. Assumes NO HARDERIAN TUMORS in MICE

E. Geometric Mean of Human Cancer Slope Factors based on Male and Female Mice.

Table IV.A.1 Estimates of **Lifetime Increased Risk of Cancer from consuming Acrylamide in Coffee**, based on Extrapolation of Tumor Results in the NTP Rat and Mice Bioassays, and consumption of from 1-9 Cups of Coffee/day from age 16+. Comparison of my (S.B.) Estimates with Those of the U.S. EPA (2010; Plaintiff's Exhibit #48631) and CAL. EPA (2005; Plaintiff's Exhibit #23930). All Human Cancer Slope Factors first adjusted downward (by 54/70 years) to account for little coffee drinking during < 16 years. Estimates of the **NSRL** (amount AA from Coffee/day for a 1 in 100,000 risk of cancer.) [A].

Human Cancer Potency Est. from:	ADJ. Human Cancer Factor	Coffee Consumption (Cups/day)[B] 1	3.1	5	7	9	**NSR**L (1.0E-5 risk) Based on Study Cancer Potency Estimates
	$(mg/kg\text{-}day)^{-1}$	Acrylamide Content (μg/day) 2.22	6.85	11.11	15.56	20.01	
Cal.EPA	0.540	1.72E-5	5.28E-5	8.58E-5	1.2E-4	1.54E-4	1.30 μg/day
U.S. EPA	0.386	1.23E-5	3.77E-5	6.13E-5	8.58E-5	1.10E-4	1.81 μg/day
NTP RAT (S. B.)	0.633	2.01E-5	6.19E-5	1.00E-4	1.41E-4	1.81E-4	1.11 μg/day
NTP MOUSE (S.B.)[C]	**2.05**	**6.52E-5**	**2.01E-4**	**3.26E-4**	**4.56E-4**	**8.07E-4**	**0.34 μg/day**

A. All Estimates of the NSRL are less than the amount of AA in 1 cup of coffee. Higher increased cancer risks are seen from more coffee consumption per day.

B. Estimated Percentiles based on the NCA's Survey of 428 coffee drinkers age 18+ who lived in the "West". 1 Cup: 9th %ile; 3.1 Cups: 48th %ile; 5 cups: 71st %ile; 7 cups: 85th %ile; 9 cups: 91st %ile.

C. "Most Sensitive Study." See Discussion in Section VI. Conclusion and Opinion .

EXHIBIT “E”

Statement of Steven Bayard Regarding: Calculation of the No Significant Risk Level (NSRL) for Acrylamide; Calculations of Cancer Potency Slopes Using the Animal-to-Human Species Conversion Factor; and Number of Previous Depositions.

I. Clarification of NSRL for acrylamide in coffee.

Note: All statements below on the risk from drinking coffee refer to the risk due to the acrylamide content in coffee.

In my Exhibit #1, "Statement of Steven Bayard, Ph.D.", which contained my cancer risk assessment for acrylamide in coffee, I calculate what I have called a "NSRL (amount of AA from coffee/day for a 1 in 100,000 (increased) risk of cancer.)" Table IV.A.1, page 41. This "NSRL" is based on coffee consumption for 54 years, starting at age 16 and continuing through the remainder of a 70 year lifespan. I calculate this "NSRL" for several acrylamide risk assessments and note, in footnote A of Table IV.A.1, that "All estimates of the NSRL are less than the amount of AA in 1 cup of coffee."

However, the California Code of Regulations, Section 25703, (reference) defines NSRL as:

> (T)he level of exposure to the chemical which is calculated to result in no more than one excess case of cancer in an exposed population of 100,000, assuming exposure over a 70-year lifetime. (reference).

In order to avoid any confusion over whether an NSRL for acrylamide in coffee should refer to actual years of coffee consumption, or to averaging over 70 years, I have revised Table IV.A.1 by retitling that last column, and by adding a new column in which I express the NSRL as the amount of AA, which, if drunk every day for 70 years would increase the risk of cancer by 1/100,000. This revised Table IV.A.1 is included in this statement.

II. Use of $(W_h/W_a)^{1/4}$ Animal-to-Human Scaling factor for Interspecies Conversion. Revision of Human cancer Potency Estimates based on the NTP Rat Studies.

In my Exhibit #1, in estimating human cancer potency values from the NTP rat and mouse bioassay studies, I used two different scaling factors for interspecies conversion from animal cancer potency to human cancer potency. For the NTP mouse studies I used $(W_{hum}/W_{mouse})^{1/4}$ (i.e. human body weight default of 70 kg/mouse body weight kg)$^{1/4}$, and these scaling factors were 6.9 for the male mouse and 7.3 for the female mouse (my Exhibit #1: page 7, and Table I.D.1, page 39. For the NTP rat studies, I used the $(W_{hum}/W_{rat})^{1/3}$ default for the pharmacodynamic part of the scaling factor, to calculate conversion factors of 2.82 for the male rat and 3.05 for the female rat (Exhibit #1: page 7, and Table I.D.1 page 39.) After reading the current California Code of Regulations 27 CCR Section 25703 on the topic "Quantitative Risk Assessment", which specifies use of the $(W_h/W_a)^{1/4}$, (25703 (a) (6), http://weblinks.westlaw.com/result/default.aspx.aspx?action=Search&cfid=1&cnt=DOC&db=C... (6/5/2013), I have concluded that, in view of the current specifications, using this ¼ power factor for the NTP rat is appropriate in this case. I have, therefore, revised the scaling factors for the rat-to-human extrapolation to reflect this change. The revised factors and the affected risk calculations are presented in my revised Table I.D.1, below. These revised factors also affect calculations in Tables I.D.2 and IV.A.1. I have included the new calculations in the revised Tables 1.D.2 and IV.A.1, below.

III. Previous Depositions.

In my April 28, 2014 deposition in this case, I testified that I had previously made 3 depositions/court hearings. At that time I could not remember any additional depositions. While I still cannot remember any additional instances, I may have given a deposition as an U.S. EPA employee in the 1993 Phillip Morris Inc. lawsuit against the U.S. EPA on Environmental Tobacco Smoke. It is possible; I just don't remember if I did.

Table Revised 06/07/2014 for $(W_{hum}/W_{rat})^{1/4}$ Scaling Factor

Table I.D.1. Animal and Human Oral Cancer Slope (Potency) Estimates for Acrylamide based on Tumor Results in the NTP (2012) Rat and Mouse 2-Year Study Bioassay. Estimates for those Tumor sites NTP judged as "Clear Evidence of Carcinogenic Activity" or "Related." Animal Cancer slopes calculated using U.S. EPA's BMDS Version 2.4, plus EPA's method for calculating Combined Risks.
Human Cancer slope factors use adaption of Cal EPA's animal-to-human scaling factors for rats, and U.S. EPA's default procedures for the mouse.

Sex/Species	ORGAN	BMDL	Animal Canc Slope factor	Scaling Factor	Human Cancer Slope Factor
M/mouse	Harderian Adenoma	0.32	0.313	6.9	**2.154**
M/mouse	Lung Adeno	1.57	0.064	6.9	**0.439**
M/mouse	Stomach	3.094	0.032	6.9	**0.223**
M/mouse	**Combined Sites**		0.381	6.9	**2.628**
F/mouse	Harderian Adeno	0.474	0.211	7.3	**1.535**
F/mouse	Lung Adeno	1.471	0.068	7.3	**0.495**
F/mouse	Stomach	5.045	0.020	7.3	**0.144**
F/mouse	Mammary Adcarc/acnatho	1.679	0.060	7.3	**0.433**
F/mouse	Ovary granulosa	6.48	0.015	7.3	**0.112**
F/mouse	Skin fibroma/sarcoma	2.41	0.041	7.3	**0.302**
F/mouse	**Combined Sites**		0.370	7.3	**2.692**
M/Rat	Thyroid Gland foll. Ad/Ca.	0.889	0.112	2.3	**0.259**
M/Rat	Heart Schwannoma	1.304	0.077	2.3	**0.176**
M/Rat	Epidy testis/mesothel.	1.21	0.083	2.3	**0.190**
M/Rat	Pancreas Islets Adeno.	1.59	0.063	2.3	**0.145**
M/Rat	**Combined Sites**		0.262	2.3	**0.602**
F/Rat	Thyroid Gland foll. Ad/Ca.	2.328	0.043	2.4	**0.104**
F/Rat	Mammary Fibroadenoma	0.441	0.227	2.4	**0.546**
F/Rat	Oral Mucosa/tongue	2.019	0.050	2.4	**0.119**
F/Rat	Skin fibroma/sarcoma	3.084	0.032	2.4	**0.078**
F/Rat	Clitoral gland carcinoma				
F/Rat	**Combined Sites**		0.309189	2.4	**0.745**

Table I.D.2. **REVISED**. Estimates of Human Cancer Slope Risk Factors From Daily Oral Exposure to Acrylamide in Coffee, based on Individual Tumor Sites and Summed Risks from Multiple Sites Combined for Three Rat Bioassays and One Mouse Bioassay, both Sexes separately. Estimates for NTP Rat study based on CAL EPA animal-to-human weight and dose scaling factors. **TABLE REVISED 06/07/2014**

	Johnson. (1986) **RATS**[A]		Friedman (1995) **Rats**[A]		NTP (2012) **RATS**		NTP (2012) **MOUSE**		
Human Cancer Potency Factors - per (mg/kg b.w.-day).									
Tumor Site Rats	**Females**	**Males**	**Females**	**Males**	**Females**	**Males**	**Tumor Sites Mice**	**Females**	**Males**
Mammary	1.0	--	0.4	--	0.55	--	Mammary	0.43	--
Cent. Nervous. Sys.	0.14	--	0.71	0.13	--	--	Harderian	1.53	2.15
Thyroid follicular	0.24	0.32	0.33	0.44	0.10	0.26	Fore-stomach	0.14	0.22
Testis	--	0.58	--	0.40	--	0.19	Lung alv/br. A/C	0.99	0.44
Heart Schwannoma Pancreas Islet Tumors	--	--	--	--	--	0.18 0.14	--	--	--
Oral Cavity	0.31	--	--	--	0.12	--	--	--	--
Uterus	0.15	--	--	--	--	--	Ovary	0.11	--
Clitoral Gland	0.26	--	--	--	--	--	Clitoral Gland	Not calc.	--
Skin	--	--	--		0.08		Skin	0.30	
Mult. Sites Combined.	1.5	0.75	0.69	0.77	0.75	0.60	Multi. Sties Comb.	2.69	2.63
Geo. Mean Risk Factor	0.70 (CAL/OEHHA, 2005)				0.67			2.66	

A. Human Cancer Potency Factors based on the Johnson (1986) and Friedman (1995) studies are from Cal OEHHA (2005, Exhibit #23930. Cal EPA and U.S. EPA methods for Estimating risks from Combined tumor sites judged quantitatively similar.

REVISED (6/7/2014) for both Clarification of the NSRL and use of $(W_{hum}/W_{rat})^{1/4}$ scaling factor.

Table IV.A.1 Estimates of **Lifetime Increased Risk of Cancer from consuming Acrylamide (AA) in Coffee**, based on Extrapolation of Tumor Results in the NTP Rat and Mice Bioassays, and consumption of from 1-9 Cups of Coffee/day from age 16+. Comparison of my (S.B.) Estimates with Those of the U.S. EPA (2010; Plaintiff's Exhibit #48631) and CAL. EPA (2005; Plaintiff's Exhibit #23930). All Human Cancer Slope Factors first adjusted downward (by 54/70 years) to account for little coffee drinking during < 16 years. Acrylamide amounts/day both for 54 years of coffee consumption, and for a full 70-year lifetime **(NSRL)** that will result in a 1 in 100,000 increased risk of cancer.

Human Cancer Potency Est. from:	ADJ. Human Cancer Factor	Coffee Consumption (Cups/day)[B] 1	3.1	5	7	9	Acrylamide amounts/day for a lifetime 1/100,000 increased risk [A]. Based on Study Cancer Potency Estimates.	
	$(mg/kg\text{-}day)^{-1}$	Acrylamide Content (μg/day) 2.22	6.85	11.11	15.56	20.01	For AA from 54 Years of coffee consumption	For 70 Years "**NSRL**"
Cal. EPA (2005)	0.540	1.72E-5	5.28E-5	8.58E-5	1.2E-4	1.54E-4	1.30 μg/day	1.00 μg/day [D]
U.S. EPA (2010)	0.386	1.23E-5	3.77E-5	6.13E-5	8.58E-5	1.10E-4	1.81 μg/day	1.40 μg/day
NTP RAT (S. B.)	0.517	1.64E-5	5.09E-5	8.20E-5	1.15E-4	1.48E-4	1.11 μg/day	0.86 μg/day
NTP MOUSE (S.B.)[C]	**2.05**	**6.52E-5**	**2.01E-4**	**3.26E-4**	**4.56E-4**	**5.86E-4**	**0.34 μg/day**	**0.26 μg/day**

A. All Estimates of the both the NSRL for AA and for AA from actual 54 years of coffee consumption are less than the amount of AA in 1 cup of coffee. Higher increased cancer risks are seen from more coffee consumption per day.

B. Estimated Percentiles based on the NCA's Survey of 428 coffee drinkers age 18+ who lived in the "West". 1 Cup: 9th %ile; 3.1 Cups: 48th %ile; 5 cups: 71st %ile; 7 cups: 85th %ile; 9 cups: 91st %ile.

C. "Most Sensitive Study." See Discussion in Section VI. Conclusions and Opinion

D. NSRL derived by Cal. EPA (2005).

EXHIBIT “F”

Corrections to April 28, 2014 Deposition: Exhibits #1 and #3.

Steven P. Bayard

I. Clarification of NSRL for Acrylamide in Coffee. Exhibit #1.

Note: All statements below on the risk from drinking coffee refer to the risk due to the acrylamide content in coffee.

In my Exhibit #1, "Statement of Steven Bayard, Ph.D.", which contained my cancer risk assessment for acrylamide in coffee, I calculate what I have called a "NSRL (amount of AA from coffee/day for a 1 in 100,000 (increased) risk of cancer.)" Table IV.A.1, page 41. I based this "NSRL" on coffee consumption for 54 years, starting at age 16 and continuing through the remainder of a 70 year lifespan. I calculated this "NSRL" for several acrylamide risk assessments and note, in footnote A of Table IV.A.1, that "All estimates of the NSRL are less than the amount of AA in 1 cup of coffee."

However, the California Administrative Code, (Title 27, Section 25703, Division 4, Chapter 1, (Article 7 (b)) defines NSRL as the level of exposure to the chemical

> "which is calculated to result in one excess case of cancer in an exposed population of 100,000, assuming lifetime exposure at the level in question …"

According to this above definition of "lifetime exposure at the level in question," this exposure level is interpreted as 'lifetime exposure expressed as a daily level averaged over a 70-year lifetime."

In order to avoid any confusion over whether an NSRL for acrylamide in coffee should refer to actual years of coffee consumption, or to daily averaging over 70 years, I have revised Table IV.A.1 by retitling that last column, and by adding a new column in which I express the NSRL as the amount of AA, which, if drunk every day for 70 years would increase the risk of cancer by 1/100,000. This

revised (6/9/2014) Table IV.A.1 is included in this statement. Also, the following lines in Exhibit #1should be changed:

Pg. 9, 2nd full paragraph, last line: Change "1 per 1,000" to "6 per 10,000"

Page 9, 3rd full paragraph, last line: Change to read "NSRL level of AA is exceeded in just 2/3 cup of coffee per day, averaged over the 70 years (2.22 μg/day x (54/70) = 1.71 μg/day.)

II. Use of $(W_h/W_a)^{1/4}$ Animal-to-Human Scaling factor for Interspecies Conversion. Revision of Human Cancer Potency Estimates and Increased Cancer Risks based on the NTP Rat Studies. Exhibit #1.

In my Exhibit #1, in estimating human cancer potency values from the NTP rat and mouse bioassay studies, I used two different scaling factors for interspecies conversion from animal cancer potency to human cancer potency. For the NTP mouse studies I used $(W_{hum}/W_{mouse})^{1/4}$ (i.e. human body weight default of 70 kg/mouse body weight kg)$^{1/4}$, and these scaling factors were 6.9 for the male mouse and 7.3 for the female mouse (my Exhibit #1: page 7, and Table I.D.1, page 39. For the NTP rat studies, I used the $(W_{hum}/W_{rat})^{1/3}$ default for the pharmacodynamic part of the scaling factor, to calculate conversion factors of 2.8 for the male rat and 3.0 for the female rat (Exhibit #1: page 7, and Table I.D.1 page 39.) After reading the current California Code of Regulations 27 CCR Section 25703 on the topic "Quantitative Risk Assessment", which specifies use of the $(W_h/W_a)^{1/4}$, (25703 (a) (6), http://weblinks.westlaw.com/result/default.aspx.aspx?action=Search&cfid=1&cnt=DOC&db=C... (6/5/2013), I have concluded that, in view of the current specifications, using this ¼ power factor for the NTP rat is appropriate in this case. I have, therefore, revised the scaling factors for the rat-to-human extrapolation to reflect this change. The revised factors, and the affected risk calculations are presented in my revised (06/07/2014) Table I.D.1, below. These revised factors also affect calculations in Tables I.D.2 (Revised 06/07/2014) and IV.A.1 (Revised 06/09/2014). I have included the new calculations in the revised Tables 1.D.2 and

IV.A.1, below. Corresponding wording changes, in Exhibit #1, page 7, lines 12-13 are: "For my analysis of the NTP (2012) data, I use $(W_{hum}/W_{rat})^{1/4}$ for the PD factor. I also substitute the NTP male rat = 418.1 gm, and female rat = 262.0 gm weights and calculate interspecies scaling factors of 2.27 for the male rat and 2.41 for the …"

III. Recalculation of Combined Tumor Cancer Slope Factors and Human Risk Estimates for Male Mice Risk Assessment Without Harderian Gland Tumors. Exhibit #3.

Table 1.B.3 in original Exhibit #3 contained two calculation errors for the Upper-bound on cumulative risks for the NTP male mouse. The first should be changed from 0.04143 to 0.06369; the second from 0.063413 to 0.087962. These revised calculations affect the male mouse cancer slope factors (without Harderian tumors) and the human cancer slope factors that are based on the male mouse. This is shown in Table 1.D.1 Without Mouse Harderian Tumors (corrected (06/09/2014). Other tables included here for Exhibit #3 corrections reflect the corrections made for Exhibit #1, specifically, the $(W_{human}/W_{rat})^{1/4}$ scaling factor and the NSRL.

Revised Exhibit #1 Tables

Table Revised 06/07/2014 for $(W_{hum}/W_{rat})^{1/4}$ Scaling Factor

Table I.D.1. Animal and Human Oral Cancer Slope (Potency) Estimates for Acrylamide based on Tumor Results in the NTP (2012) Rat and Mouse 2-Year Study Bioassay. Estimates for those Tumor sites NTP judged as "Clear Evidence of Carcinogenic Activity" or "Related." Animal Cancer slopes calculated using U.S. EPA's BMDS Version 2.4, plus EPA's method for calculating Combined Risks.
Human Cancer slope factors use adaption of Cal EPA's animal-to-human scaling factors for rats, and U.S. EPA's default procedures for the mouse.

Sex/Species	ORGAN	BMDL	Animal Canc Slope factor	Scaling Factor	Human Cancer Slope Factor
M/mouse	Harderian Adenoma	0.32	0.313	6.9	**2.154**
M/mouse	Lung Adeno	1.57	0.064	6.9	**0.439**
M/mouse	Stomach	3.094	0.032	6.9	**0.223**
M/mouse	**Combined Sites**		0.381	6.9	**2.628**
F/mouse	Harderian Adeno	0.474	0.211	7.3	**1.535**
F/mouse	Lung Adeno	1.471	0.068	7.3	**0.495**
F/mouse	Stomach	5.045	0.020	7.3	**0.144**
F/mouse	Mammary Adcarc/acnatho	1.679	0.060	7.3	**0.433**
F/mouse	Ovary granulosa	6.48	0.015	7.3	**0.112**
F/mouse	Skin fibroma/sarcoma	2.41	0.041	7.3	**0.302**
F/mouse	**Combined Sites**		0.370	7.3	**2.692**
M/Rat	Thyroid Gland foll. Ad/Ca.	0.889	0.112	2.3	**0.259**
M/Rat	Heart Schwannoma	1.304	0.077	2.3	**0.176**
M/Rat	Epidy testis/mesothel.	1.21	0.083	2.3	**0.190**
M/Rat	Pancreas Islets Adeno.	1.59	0.063	2.3	**0.145**
M/Rat	**Combined Sites**		0.262	2.3	**0.602**
F/Rat	Thyroid Gland foll. Ad/Ca.	2.328	0.043	2.4	**0.104**
F/Rat	Mammary Fibroadenoma	0.441	0.227	2.4	**0.546**
F/Rat	Oral Mucosa/tongue	2.019	0.050	2.4	**0.119**
F/Rat	Skin fibroma/sarcoma	3.084	0.032	2.4	**0.078**
F/Rat	Clitoral gland carcinoma				
F/Rat	**Combined Sites**		0.309189	2.4	**0.745**

Table I.D.2. **REVISED**. Estimates of Human Cancer Slope Risk Factors From Daily Oral Exposure to Acrylamide in Coffee, based on Individual Tumor Sites and Summed Risks from Multiple Sites Combined for Three Rat Bioassays and One Mouse Bioassay, both Sexes separately. Estimates for NTP Rat study based on CAL EPA animal-to-human weight and dose scaling factors. **TABLE REVISED 06/07/2014**

	Johnson. (1986) **RATS**[A]		Friedman (1995) **Rats**[A]		NTP (2012) **RATS**		NTP (2012) **MOUSE**		
Human Cancer Potency Factors - per (mg/kg b.w.-day).									
Tumor Site Rats	**Females**	**Males**	**Females**	**Males**	**Females**	**Males**	**Tumor Sites Mice**	**Females**	**Males**
Mammary	1.0	--	0.4	--	0.55	--	Mammary	0.43	--
Cent. Nervous. Sys.	0.14	--	0.71	0.13	--	--	Harderian	1.53	2.15
Thyroid follicular	0.24	0.32	0.33	0.44	0.10	0.26	Fore-stomach	0.14	0.22
Testis	--	0.58	--	0.40	--	0.19	Lung alv/br. A/C	0.49	0.44
Heart Schwannoma Ppancreas Islet Tumors	--	--	--	--	--	0.18 0.14	--	--	--
Oral Cavity	0.31	--	--	--	0.12	--	--	--	--
Uterus	0.15	--	--	--	--	--	Ovary	0.11	--
Clitoral Gland	0.26	--	--	--	--	--	Clitoral Gland	Not calc.	--
skin	--	--	--		0.08		Skin	0.30	
Mult. Sites Combined.	1.5	0.75	0.69	0.77	0.75	0.60	Multi. Sties Comb.	2.69	2.63
Geo. Mean Risk Factor	0.70 (CAL/OEHHA, 2005)				0.67			2.66	

A. Human Cancer Potency Factors based on the Johnson (1986) and Friedman (1995) studies are from Cal OEHHA (2005, Exhibit #23930. Cal EPA and U.S. EPA methods for Estimating risks from Combined tumor sites judged quantitatively similar..

REVISED (6/09/2014) for both Clarification of the NSRL and use of $(W_{hum}/W_{rat})^{1/4}$ scaling factor.

Table IV.A.1 Estimates of **Lifetime Increased Risk of Cancer from consuming Acrylamide (AA) in Coffee**, based on Extrapolation of Tumor Results in the NTP Rat and Mice Bioassays, and consumption of from 1-9 Cups of Coffee/day from age 16+. Comparison of my (S.B.) Estimates with Those of the U.S. EPA (2010; Plaintiff's Exhibit #48631) and CAL. EPA (2005; Plaintiff's Exhibit #23930). All Human Cancer Slope Factors first adjusted downward (by 54/70 years) to account for little coffee drinking during < 16 years. Acrylamide amounts/day both for 54 years of coffee consumption, and for a full 70-year lifetime **(NSRL)** that will result in a 1 in 100,000 increased risk of cancer.

Human Cancer Potency Est. from:	ADJ. Human Cancer Factor	Coffee Consumption (Cups/day)[B] 1	3.1	5	7	9	Acrylamide amounts/day for a lifetime 1/100,000 increased risk [A]. Based on Study Cancer Potency Estimates.	
	$(mg/kg\text{-}day)^{-1}$	Acrylamide Content (μg/day) 2.22	6.85	11.11	15.56	20.01	For AA from 54 Years of coffee consumption	For 70 Years "**NSRL**"
Cal. EPA (2005)	0.540	1.72E-5	5.28E-5	8.58E-5	1.2E-4	1.54E-4	1.30 μg/day	1.00 μg/day [D]
U.S. EPA (2010)	0.386	1.23E-5	3.77E-5	6.13E-5	8.58E-5	1.10E-4	1.81 μg/day	1.40 μg/day
NTP RAT (S. B.)	0.517	1.64E-5	5.09E-5	8.20E-5	1.15E-4	1.48E-4	1.11 μg/day	0.86 μg/day
NTP MOUSE (S.B.)[C]	**2.05**	**6.52E-5**	**2.01E-4**	**3.26E-4**	**4.56E-4**	**5.86E-4**	**0.34 μg/day**	**0.26 μg/day**

A. All Estimates of both the NSRL for AA and for AA from actual 54 years of coffee consumption are less than the amount of AA in 1 cup of coffee. Higher increased cancer risks are seen from more coffee consumption per day.

B. Estimated Percentiles based on the NCA's Survey of 428 coffee drinkers age 18+ who lived in the "West". 1 Cup: 9th %ile; 3.1 Cups: 48th %ile; 5 cups: 71st %ile; 7 cups: 85th %ile; 9 cups: 91st %ile.

C. "Most Sensitive Study." See Discussion in Section VI. Conclusions and Opinion

D. NSRL derived by Cal. EPA (2005).

REVISED EXHIBIT #3 TABLES

(Corrected 05/25/2014) **TABLE I.B.3 MALE MICE (NOT INCLUDING HARDERIAN GLAND TUMORS.)**

Calculation of Summed Risks for tumors at Several Sites in **MALE B6C3F1 MICE/NOT INCLUDING HARDERIAN Tumors** exposed to AA in Drinking Water in the NTP Bioassay (2012; Plaintiff's Exhibit 48449). Dose Groups: 0, 1.04, 2.20, 4.11, and 8.93 mg/kg/day. Using BMD10 and BMDL10 output from EPA's BMDS Software 2.4 and BMDS Wizard 1.8. Calculations for for Combined Risks for Multiple Tumor Sites based on method in U.S. EPA Tox. Review of Acrylamide, Table D8 and Table D10) in Appendix D. (U.S. EPA; 2010; Plaintiff's Exhibit 48631).

TUMOR SITE	**BMD10** mg/kg-day	**BMDL10** mg/kg-day	**Mouse Oral Cancer Slope central est.** $(mg/kg\text{-}day)^{-1}$	**MOUSE Oral Cancer slope factor (upper-bound)** $(mg/kg\text{-}day)^{-1}$	t-statistic	standard deviation	Variance
Harderian Gland Adenoma	**NOT CALCULATED**						
Lung Alv./Bronch Adenoma	**2.414**	**1.57**	0.041425	0.063694	1.645	0.013538	0.00018323
Cumulative Variance =	0.00018						
Cumulative s.d. =	0.01354						
Sum of Central tendency Risks=	0.04143	$(mg/kg\text{-}day)^{-1}$					
Upper-bound on cum risk=	**0.06369**	$(mg/kg\text{-}day)^{-1}$		**CANCER SLOPE FACTOR** FOR **ONE** SITE			
Harderian Gland Adenoma	**NOT CALCULATED**						
Lung Alv./Bronch Adenoma	**2.414**	**1.57**	0.041425	0.063694	1.645	0.013538	0.0001833
Stomach (fore) SQ. cell Pappilloma	**4.548**	**3.094**	0.021988	0.032321	1.645	0.006281	3.94562E-05
Cumulative Variance =	0.000223						
Cumulative s.d. =	0.014924						
Sum of Central tendency Risks=	0.063413						
Upper-bound on cum risk=	**0.087962**	(mg/kg-day)-1		**CANCER SLOPE FACTOR** FOR **TWO** Combined SITES			

TABLE I.B.4. Calculation of Summed Risks for tumors in **FEMALE B6C3F1 MICE WITHOUT HARDERIAN** exposed to AA in Drinking Water in the NTP Bioassay (2012). Doses Groups: 0, 1.10, 2.23, 4.65, and 9.96 mg/kg-da. BMD10 and BMDL10 output from EPA's BMDS 2.4 and BMDS Wizard 1.8. Calculations for Combined Risks for Multiple Tumor Sites based on method in U.S. EPA Tox. Rev. of Acryl. Tables D-8 and D-10 in Appendix D. (U.S. EPA; 2010; Plaintiff's Exhibit 48631).

TUMOR SITE	BMD10	BMDL10	MOUSE cent slope factor	MOUSE U.B. slope factor	t-statis	s.d.	Variance
	mg/kg-da	mg/kg-da	(mg/kg-day)-1	(mg/kg-day)-1			
Lung Alv./Bronch Ade	**1.968**	**1.471**	0.050813	0.067981	1.645	0.010436	0.0001089
Mammary Adenocarc	**2.219**	**1.679**	0.045065	0.059559	1.645	0.008811	7.763E-05
Cumulative Variance	0.000187						
Cumulative s.d. =	0.013658						
Sum of Central tende	0.095878						
Upper-bound on cum	**0.118346**	(mg/kg-day)-1		**CANCER SLOPE FACTOR** FOR **TWO** Comb. SITES			
Lung Alv./Bronch Ade	**1.968**	**1.471**	0.050813	0.067981	1.645	0.010436	0.0001089
Mammary Adenocarc	**2.219**	**1.679**	0.045065	0.059559	1.645	0.008811	7.763E-05
Ovary benign Granul	**11.4**	**6.48**	0.008772	0.015432	1.645	0.004049	1.639E-05
Cumulative Variance	0.000203						
Cumulative s.d. =	0.014246						
Sum of Central tende	0.104650						
Upper-bound on cum	**0.128085**	(mg/kg-day)-1		**CANCER SLOPE FACTOR** FOR **3** Comb. SITES			
Lung Alv./Bronch Ade	**1.968**	**1.471**	0.050813	0.067981	1.645	0.010436	0.0001089
Mammary Adenocarc	**2.219**	**1.679**	0.045065	0.059559	1.645	0.008811	7.763E-05
Ovary benign Granul	**11.4**	**6.48**	0.008772	0.015432	1.645	0.004049	1.639E-05
Skin Fibroma/Sarcom	**3.05**	**2.41**	0.032787	0.041494	1.645	0.005293	2.802E-05
Cumulative Variance	0.000231						
Cumulative s.d. =	0.015197						
Sum of Central tende	0.137437						
Upper-bound on cum	**0.162437**	(mg/kg-day)-1		**CANCER SLOPE FACTOR** FOR **Four** Comb. SITES			
Lung Alv./Bronch Ade	**1.968**	**1.471**	0.050813	0.067981	1.645	0.010436	0.0001089
Mammary Adenocarc	**2.219**	**1.679**	0.045065	0.059559	1.645	0.008811	7.763E-05
Ovary benign Granul	**11.4**	**6.48**	0.008772	0.015432	1.645	0.004049	1.639E-05
Skin Fibroma/Sarcom	**3.05**	**2.41**	0.032787	0.041494	1.645	0.005293	2.802E-05
Stomach (fore) Squar	**7.623**	**5.045**	0.013118	0.019822	1.645	0.004075	1.661E-05
Cumulative Variance	0.000248						
Cumulative s.d. =	0.015734						
Sum of Central tende	0.150555						
Upper-bound on cum	**0.176438**	(mg/kg-day)-1		**CANCER SLOPE FACTOR** FOR **FIVE** Comb. SITES			

Corrected (6/09/2014) Table 1.D.1. WITHOUT MOUSE HARDERIAN TUMORS

Animal and Human Oral Cancer Slope (Potency) Estimates for Acrylamide based on Tumor Results in the NTP (2012) Rat and Mouse 2-Year Study Bioassays. Estimates for those Tumor sites NTP judged as "Clear Evidence of Carcinogenic Activity" or "Related." Animal Cancer slope calculated using U.S. EPA's BMDS Version 2.4, plus EPA's method for calculating Combined Risks. Human Cancer slope factors use adaptation of Cal EPA's animal-to-human scaling factors for rats, and U.S. EPA's default scaling Factors, $(W_h/W_m)^{1/4}$, for the mouse.

Corrected to reflect change to $(W_{human}/W_{rat})^{1/4}$

Sex/Species	ORGAN	BMDL10	Animal Cancer Slope factor	Scaling Factor	Human Cancer Slope Factor
M/mouse	Harderian Adenoma	NOT CALC.	NOT CALC.		**0**
M/mouse	Lung Adenoma	1.57	0.064	6.9	**0.439**
M/mouse	Stomach	3.094	0.032	6.9	**0.223**
M/mouse	**Combined Sites**		0.088	6.9	**0.607**
F/mouse	Harderian Adenoma	NOT CALC.	NOT CALC.		**0**
F/mouse	Lung Adenoma	1.471	0.068	7.3	**0.495**
F/mouse	Stomach	5.045	0.020	7.3	**0.144**
F/mouse	Mammary Adcarc/acnatho	1.679	0.060	7.3	**0.433**
F/mouse	Ovary granulosa	6.48	0.015	7.3	**0.112**
F/mouse	Skin fibroma/sarcoma	2.41	0.041	7.3	**0.302**
F/mouse	**Combined Sites**		0.176	7.3	**1.283**
M/Rat	Thyroid Glnd foll. Ad/Ca.	0.889	0.112	2.3	**0.259**
M/Rat	Heart Schwannoma	1.304	0.077	2.3	**0.176**
M/Rat	Epidy testis/mesothelioma	1.21	0.083	2.3	**0.190**
M/Rat	Pancreas Islets Adenoma	1.59	0.063	2.3	**0.145**
M/Rat	**Combined Sites**		0.262	2.3	**0.602**
F/Rat	Thyroid Gland foll. Ad/Ca.	2.328	0.043	2.4	**0.104**
F/Rat	Mammary Fibroadenoma	0.441	0.227	2.4	**0.546**
F/Rat	Oral Mucosa/tongue	2.019	0.050	2.4	**0.119**
F/Rat	Skin fibroma/sarcoma	3.084	0.032	2.4	**0.078**
F/Rat	Clitoral gland carcinoma	NOT CALC.			
F/Rat	**Combined Sites**		0.309189	2.4	**0.745**

Table Revised 06/07/2014 To Reflect Change To $(W_{hum}/W_{rat})^{1/4}$ Scaling Factor for NTP Rat

Table I.D.1. Animal and Human Oral Cancer Slope (Potency) Estimates for Acrylamide based on Tumor Results in the NTP (2012) Rat and Mouse 2-Year Study Bioassay. Estimates for those Tumor sites NTP judged as "Clear Evidence of Carcinogenic Activity" or "Related." Animal Cancer slopes calculated using U.S. EPA's BMDS Version 2.4, plus EPA's method for calculating Combined Risks.
Human Cancer slope factors use adaption of Cal EPA's animal-to-human scaling factors for rats, and U.S. EPA's default procedures for the mouse.

Sex/Species	ORGAN	BMDL	Animal Canc Slope factor	Scaling Factor	Human Cancer Slope Factor
M/mouse	Harderian Adenoma	0.32	0.313	6.9	**2.154**
M/mouse	Lung Adenoma	1.57	0.064	6.9	**0.439**
M/mouse	Stomach	3.094	0.032	6.9	**0.223**
M/mouse	**Combined Sites**		0.381	6.9	**2.628**
F/mouse	Harderian Adenoma	0.474	0.211	7.3	**1.535**
F/mouse	Lung Adenoma	1.471	0.068	7.3	**0.495**
F/mouse	Stomach	5.045	0.020	7.3	**0.144**
F/mouse	Mammary Adcarc/acnatho	1.679	0.060	7.3	**0.433**
F/mouse	Ovary granulosa	6.48	0.015	7.3	**0.112**
F/mouse	Skin fibroma/sarcoma	2.41	0.041	7.3	**0.302**
F/mouse	**Combined Sites**		0.370	7.3	**2.692**
M/Rat	Thyroid Gland foll. Ad/Carcinoma.	0.889	0.112	2.3	**0.259**
M/Rat	Heart Schwannoma	1.304	0.077	2.3	**0.176**
M/Rat	Epidy testis/mesothelioma	1.21	0.083	2.3	**0.190**
M/Rat	Pancreas Islets Adenoma	1.59	0.063	2.3	**0.145**
M/Rat	**Combined Sites**		0.262	2.3	**0.602**
F/Rat	Thyroid Gland foll. Ad/Carcinoma.	2.328	0.043	2.4	**0.104**
F/Rat	Mammary Fibroadenoma	0.441	0.227	2.4	**0.546**
F/Rat	Oral Mucosa/tongue	2.019	0.050	2.4	**0.119**
F/Rat	Skin fibroma/sarcoma	3.084	0.032	2.4	**0.078**
F/Rat	Clitoral gland carcinoma				
F/Rat	**Combined Sites**		0.309189	2.4	**0.745**

Table I.D.2. Estimates of Human Cancer Slope Risk Factors From Daily Oral Exposure to Acrylamide in Coffee, based on Individual Tumor Sites and Summed Risks from Multiple Sites Combined for Three Rat Bioassays and One Mouse Bioassay, both Sexes separately. Estimates for NTP Rat study based on adaptation of CAL EPA animal-to-human weight and dose scaling factors. **(REVISED 06/09/2014 to reflect change to $(W_h/W_{rat})^{1/4}$ scaling factor for the NTP rat.)**

	Johnson. (1986) **RATS**[A]		Friedman (1995) **RATS**[A]		NTP(2012) **RATS**		NTP (2012) **MOUSE**		
Human Cancer Potency (Slope) Factors - per (mg/kg b.w.-day).									
Tumor Site Rats	**Females**	**Males**	**Females**	**Males**	**Females**	**Males**	**Tum. Sites Mice**	**Females**	**Males**
Mammary	1.0	--	0.4	--	0.55	--	Mammary	0.43	--
Cent. Nervous. Sys.	0.14	--	0.71	0.13	--	--	Harderian	1.53	2.15
Thyroid follicular	0.24	0.32	0.33	0.44	0.10	0.26	Fore-stomach	0.14	0.22
Testis	--	0.58	--	0.40	--	0.19	Lung alv/br. A/C	0.49	0.44
Heart Schwannoma Pancreas Islet Tumors	--	--	--	--	--	0.18 0.14	--	--	--
Oral Cavity	0.31	--	--	--	0.12	--	--	--	--
Uterus	0.15	--	--	--	--	--	Ovary	0.11	--
Clitoral Gland	0.26	--	--	--	--	--	Clitoral Gland	No calc.	--
Skin	--	--	--		0.08		Skin	0.30	
Mult. Sites Combined.	1.5	0.75	0.69	0.77	0.75	0.60	Sties Combined	2.69	2.63
Geo. Mean Slope Factor	0.70 (CAL EPA/OEHHA, 2005)				0.67			2.66	

A. Human Cancer Potency or Slope Factors based on the Johnson (1986) and Friedman (1995) studies are from Cal OEHHA (2005, Exhibit #23930. Cal EPA and U.S. EPA methods for Estimating risks from Combined tumor sites judged quantitatively similar.

Table I.D.2. **Revised 06/09/2014.** Estimates of Human Cancer Slope Risk Factors From Daily Oral Exposure to Acrylamide in Coffee, based on Individual Tumor Sites and Summed Risks from Multiple Sites Combined for Three Rat Bioassays and One Mouse Bioassay, both Sexes. Estimates for NTP Rat study based on CAL EPA animal-to-human weight and dose scaling factors. **ASSUMES NO HARDERIAN TUMORS IN MICE**

	Johnson. (1986) **RATS**[A]		Friedman (1995) **Rats**[A]		NTP (2012) **RATS**		NTP (2012) **MOUSE**		
Human Cancer Potency Factors - per (mg/kg b.w.-day).									
Tumor Site Rats	**Females**	**Males**	**Females**	**Males**	**Females**	**Males**	**Tum. Sites Mice**	**Females**	**Males**
Mammary	1.0	--	0.4	--	0.55	--	Mammary	0.43	--
Cent. Nervous. Sys.	0.14	--	0.71	0.13	--	--	**Harderian**	**N.C.**	**N.C.**
Thyroid follicular	0.24	0.32	0.33	0.44	0.10	0.26	Fore-stomach	0.14	0.22
Testis	--	0.58	--	0.40	--	0.19	Lung alv/br. A/C	0.49	0.44
Heart Schwannoma Pancreas Islet Tumors	--	--	--	--	--	0.18 0.14	--	--	--
Oral Cavity	0.31	--	--	--	0.12	--	--	--	--
Uterus	0.15	--	--	--	--	--	Ovary	0.11	--
Clitoral Gland	0.26	--	--	--	--	--	Clitoral Gland	Not cal.	--
skin	--	--	--		0.08		Skin	0.30	
Multiple Sites Combined	1.5	0.75	0.69	0.77	0.75	0.60	Sites Combined	1.28	0.61
Geo. Mean Slope Factor	0.70 (CAL EPA/OEHHA, 2005)				0.67			0.88	

A. Human Cancer Potency Factors based on the Johnson (1986) and Friedman (1995) studies are from Cal OEHHA (2005, Exhibit #23930. Cal EPA and U.S. EPA methods for Estimating risks from combined tumor sites judged quantitatively similar.

ASSUMES NO HARDERIAN GLAND TUMORS IN MICE

REVISED (6/9/2014) for both Clarification of the NSRL and use of $(W_{hum}/W_{rat})^{1/4}$ scaling factor.

Table IV.A.1 Estimates of **Lifetime Increased Risk of Cancer from consuming Acrylamide (AA) in Coffee**, based on Extrapolation of Tumor Results in the NTP Rat and Mice Bioassays, and consumption of from 1-9 Cups of Coffee/day from age 16+. Comparison of my (S.B.) Estimates with Those of the U.S. EPA (2010; Plaintiff's Exhibit #48631) and CAL. EPA (2005; Plaintiff's Exhibit #23930). All Human Cancer Slope Factors first adjusted downward (by 54/70 years) to account for little coffee drinking during < 16 years. Acrylamide amounts/day both for 54 years of coffee consumption, and for a full 70-year lifetime **(NSRL)** that will result in a 1 in 100,000 increased risk of cancer.

Human Cancer Potency Est. from:	ADJ. Hum Cancer Factor	Coffee Consumption (Cups/day)[B] 1	3.1	5	7	9	Acrylamide amounts/day for a lifetime 1/100,000 increased risk[A]. Based on Study Cancer Potency Estimates.	
	(mg/kg-day	Acrylamide Content (μg/day) 2.22	6.85	11.11	15.56	20.01	For AA from 54 Yrs coffee consumpt.	For 70 Years "**NSRL**"
Cal. EPA (2005)	0.540	1.72E-5	5.28E-5	8.58E-5	1.2E-4	1.54E-4	1.30 μg/day	1.00 μg/day [E]
U.S. EPA (2010)	0.386	1.23E-5	3.77E-5	6.13E-5	8.58E-5	1.10E-4	1.81 μg/day	1.40 μg/day
NTP RAT (S.B.)	0.517	1.64E-5	5.09E-5	8.20E-5	1.15E-4	1.48E-4	1.35 μg/day	1.04 μg/day
NTP MOUSE M + F COMB.[D]: (S.B.)	0.679	2.16E-5	6.64E-5	1.17E-4	1.51E-4	1.94E-4	1.03 μg/day	0.79 μg/day
NTP FEM. Mouse (NO Harderian)[C,D]	**0.987**	**3.14E-5**	**9.66E-5**	**1.57E-4**	**2.20E-4**	**2.82E-4**	**0.71** μg/day	**0.55** μg/day
NTP MALE Mouse(NO HARDERIAN)[D]	0.468	1.49E-5	4.58E-5	7.44E-5	1.05E-4	1.34E-4	1.49 μg/day	1.15 μg/day

A. All Estimates of both the 70 year NSRL for AA and for AA from actual 54 years of coffee consumption are less than the amount of AA in 1 cup of coffee. Higher increased cancer risks are seen from more coffee consumption per day.

B. Estimated Percentiles based on the NCA's Survey of 428 coffee drinkers age 18+ who lived in the "West". 1 Cup: 9th %ile; 3.1 Cups: 48th %ile; 5 cups: 71st %ile; 7 cups: 85th %ile; 9 cups: 91st %ile.

C. "Most Sensitive Study." See Discussion in Section VI. Conclusions and Opinion.

D. Assumes NO Harderian Tumors in Mice.

E. NSRL derived by Cal. EPA (2005).

REVISED (6/9/2014) for both Clarification of the NSRL and use of $(W_{hum}/W_{rat})^{1/4}$ scaling factor.

Table IV.A.1. Estimates of **Lifetime Increased Risk of Cancer from consuming Acrylamide (AA) in Coffee**, based on Extrapolation of Tumor Results in the NTP Rat and Mice Bioassays, and consumption of from 1-9 Cups of Coffee/day from age 16+. Comparison of my (S.B.) Estimates with Those of the U.S. EPA (2010; Plaintiff's Exhibit #48631) and CAL. EPA (2005; Plaintiff's Exhibit #23930). All Human Cancer Slope Factors first adjusted downward (by 54/70 years) to account for little coffee drinking during < 16 years. Acrylamide amounts/day both for 54 years of coffee consumption, and for a full 70-year lifetime **(NSRL)** that will result in a 1 in 100,000 increased risk of cancer.

Human Cancer Potency Est. from:	ADJ. Human Cancer Factor	Coffee Consumption (Cups/day)[B] 1	3.1	5	7	9	Acrylamide amounts/day for a lifetime 1/100,000 increased risk [A]. Based on Study Cancer Potency Estimates.	
	$(mg/kg\text{-}day)^{-1}$	Acrylamide Content (μg/day) 2.22	6.85	11.11	15.56	20.01	For AA from 54 Years of coffee consumption	For 70 Years "**NSRL**"
Cal. EPA (2005)	0.540	1.72E-5	5.28E-5	8.58E-5	1.2E-4	1.54E-4	1.30 μg/day	1.00 μg/day [D]
U.S. EPA (2010)	0.386	1.23E-5	3.77E-5	6.13E-5	8.58E-5	1.10E-4	1.81 μg/day	1.40 μg/day
NTP RAT (S. B.)	0.517	1.64E-5	5.09E-5	8.20E-5	1.15E-4	1.48E-4	1.35 μg/day	1.04.86 μg/day
NTP MOUSE (S.B.)[C]	**2.05**	**6.52E-5**	**2.01E-4**	**3.26E-4**	**4.56E-4**	**5.86E-4**	**0.34 μg/day**	**0.26 μg/day**

A. All Estimates of the both the NSRL for AA and for AA from actual 54 years of coffee consumption are less than the amount of AA in 1 cup of coffee. Higher increased cancer risks are seen from more coffee consumption per day.

B. Estimated Percentiles based on the NCA's Survey of 428 coffee drinkers age 18+ who lived in the "West". 1 Cup: 9th %ile; 3.1 Cups: 48th %ile; 5 cups: 71st %ile; 7 cups: 85th %ile; 9 cups: 91st %ile.

C. "Most Sensitive Study." See Discussion in Section VI. Conclusions and Opinion

D. NSRL derived by Cal. EPA (2005).

END

EXHIBIT “G”

SUPERIOR COURT OF THE STATE OF CALIFORNIA

FOR THE COUNTY OF LOS ANGELES

DEPARTMENT NO. 323 HON. ELIHU M. BERLE, JUDGE

COUNCIL FOR EDUCATION AND RESEARCH ON TOXICS,

PLAINTIFF,

VS. NO. BC435759

STARBUCKS CORPORATION, ET AL.,

DEFENDANTS.

AND CONSOLIDATED ACTION.

REPORTER'S TRANSCRIPT OF TRIAL PROCEEDINGS

MONDAY, OCTOBER 27, 2014

MORNING SESSION

APPEARANCES:

FOR THE PLAINTIFF: METZGER LAW GROUP
BY: RAPHAEL METZGER
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MICHAEL CABRAL
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CCROLA JOB NO. 114678

DANA L. SHELLEY, RPR, CSR #10177
OFFICIAL REPORTER PRO TEM

INDEX FOR OCTOBER 27, 2014
MORNING SESSION

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1 CASE NUMBER: BC435759
2 CASE NAME: CERT VS. STARBUCKS
3 DEPARTMENT: 323 HON. ELIHU M. BERLE
4 REPORTER: DANA SHELLEY, RPR, CSR #10177
5 LOS ANGELES, CALIFORNIA MONDAY, OCTOBER 27, 2014
6 TIME: 9:26 A.M.
7 APPEARANCES: (AS HERETOFORE NOTED.)
8
9 THE COURT: GOOD MORNING, COUNSEL. CALLING THE
10 CASE OF CERT VS. STARBUCKS.
11 YOUR APPEARANCES.
12 MR. SCHURZ: JAMES SCHURZ AND MICHELE CORASH ON
13 BEHALF OF THE DEFENDANT COFFEE COMPANIES.
14 THE COURT: GOOD MORNING.
15 MR. METZGER: GOOD MORNING, YOUR HONOR. RAPHAEL
16 METZGER AND KEN HOLDREN ON BEHALF OF THE PLAINTIFF
17 COUNCIL FOR EDUCATION AND RESEARCH ON TOXICS.
18 THE COURT: GOOD MORNING.
19 THE WITNESS: GOOD MORNING.
20 THE COURT: IS EVERYBODY READY?
21 MR. METZGER, DO YOU WANT TO CALL YOUR NEXT
22 WITNESS.
23 MR. METZGER: YES, YOUR HONOR. MAY I JUST HAVE A
24 MOMENT TO GET SITUATED HERE?
25 THE COURT: YES.
26 (PAUSE IN PROCEEDINGS.)
27
28

1 THE COURT: MR. METZGER, ARE YOU READY TO PROCEED?
2 MR. METZGER: YES, YOUR HONOR. THE PLAINTIFF
3 CALLS DR. STEVEN BAYARD.
4 THE CLERK: SIR, WILL YOU PLEASE STAND AND RAISE
5 YOUR RIGHT HAND.
6
7 STEVEN BAYARD,
8 CALLED AS A WITNESS BY THE PLAINTIFF, WAS SWORN AND
9 TESTIFIED AS FOLLOWS:
10 THE CLERK: THANK YOU. PLEASE BE SEATED.
11 THE WITNESS: GOOD MORNING.
12 THE COURT: GOOD MORNING, DR. BAYARD.
13 THE CLERK: AND WILL YOU STATE AND SPELL YOUR NAME
14 FOR THE RECORD.
15 THE WITNESS: YES. STEVEN BAYARD: S-T-E-V-E-N
16 B-A-Y-A-R-D.
17 THE CLERK: THANK YOU.
18 THE COURT: OKAY. MR. METZGER, YOU MAY PROCEED
19 WITH THE TESTIMONY OF DR. BAYARD.
20 MR. METZGER: THANK YOU, YOUR HONOR.
21 MR. SCHURZ: YOUR HONOR, IF WE MAY ASK FOR A POINT
22 OF DIRECTION.
23 WE RECEIVED THE MATERIALS -- THE PROPOSED
24 DEMONSTRATIVE FOR DR. BAYARD YESTERDAY. INCLUDED IN
25 THAT IS A TABLE THAT ENCOMPASSES DR. BAYARD'S ULTIMATE
26 CONCLUSIONS WITH RESPECT TO HIS QUANTITATIVE RISK
27 ASSESSMENT, HIS NUMBERS, HIS ULTIMATE VALUES THAT HE
28 RECOMMENDS.

1 WE OBSERVED THAT IN THAT TABLE, EVERY SINGLE
2 ONE OF THE FOUR VALUES THAT IS PRESENTED IS DIFFERENT
3 THAN THE VALUES THAT WERE INCLUDED AS PART OF HIS
4 WRITTEN REPORT THAT WAS THE BASIS FOR HIS DEPOSITION.
5 SO WE HAVE A NEW SET OF NUMBERS THAT WE'VE
6 SEEN FOR THE FIRST TIME THAT -- IT'S NOT CLEAR FOR WHAT
7 PURPOSE THEY'VE BEEN RECALCULATED. ALL THE NUMBERS WE
8 OBSERVED HAVE GONE DOWN, BUT WE SEE THESE AS NEW,
9 UNDISCLOSED OPINIONS, WHICH WERE NOT PROVIDED TO US
10 BEFORE YESTERDAY AT 9:00 A.M.
11 THE COURT: OKAY. MR. METZGER?
12 MR. METZGER: CANDIDLY, I'M NOT SURE WHAT THE
13 DIFFERENCES ARE. DR. BAYARD DID PREPARE A SET OF
14 ERRATA, CORRECTIONS TO HIS OPINIONS WHICH WERE PRODUCED
15 AT HIS DEPOSITION. I THINK THAT THESE TOOK -- EXPLAINED
16 THIS.
17 BUT I THINK THE WAY TO PROCEED IS TO GO
18 AHEAD. AND WE CAN ASK DR. BAYARD, IF THERE ARE ANY
19 CORRECTIONS, WHAT'S THE BASIS FOR THEM.
20 THE COURT: ALL RIGHT. WELL, WE'LL SEE.
21 THE COURT IS GOING TO OVERRULE THE
22 OBJECTIONS OF DEFENDANT AT THE PRESENT TIME, BUT WITHOUT
23 PREJUDICE TO REASSERTING THE OBJECTION AFTER CROSS-
24 EXAMINATION AND A MOTION TO STRIKE AT THE END OF THE
25 TESTIMONY.
26 COUNSEL, YOU MAY PROCEED.
27 MR. METZGER: THANK YOU, YOUR HONOR.
28

1 DIRECT EXAMINATION
2 BY MR. METZGER:
3 Q GOOD MORNING, DR. BAYARD. HOW ARE YOU?
4 A GOOD MORNING, MR. METZGER.
5 Q IS THIS YOUR FIRST TIME TESTIFYING IN A
6 COURT OF LAW?
7 A IT IS.
8 Q I'M GOING TO SHOW YOU WHAT'S BEEN MARKED AS
9 PLAINTIFF'S EXHIBIT 79. AND I WILL ASK YOU IF THIS IS
10 YOUR CURRICULUM VITAE.
11 A YES, IT IS.
12 (EXHIBIT 79 MARKED FOR IDENTIFICATION.)
13 Q BY MR. METZGER: DOES IT SET FORTH YOUR
14 EDUCATION, AREAS OF EXPERTISE, YOUR EXPERIENCE, AND
15 PUBLICATIONS?
16 A YES, SIR.
17 MR. METZGER: OKAY. YOUR HONOR, I WOULD OFFER
18 EXHIBIT 79 IN EVIDENCE.
19 THE COURT: ALL RIGHT. ANY OBJECTION?
20 MR. SCHURZ: NO OBJECTION, YOUR HONOR.
21 THE COURT: EXHIBIT 79 IS IN EVIDENCE.
22 (EXHIBIT 79 RECEIVED IN EVIDENCE.)
23 Q BY MR. METZGER: NEXT, I'D LIKE TO PROVIDE
24 YOU A DOCUMENT TITLED "STATEMENT OF STEVEN BAYARD,"
25 WHICH IS A 42-PAGE DOCUMENT MARKED AS EXHIBIT 120, AND
26 ASK YOU, FIRST OF ALL: IS THIS A DOCUMENT THAT YOU
27 PREPARED?
28 A YES, SIR, IT IS.

1 (EXHIBIT 120 MARKED FOR IDENTIFICATION.)
2 Q BY MR. METZGER: AND IS THIS A REPORT OF
3 YOURS IN WHICH YOU PROVIDE YOUR QUANTITATIVE CANCER RISK
4 ASSESSMENT FOR THE RISK OF CANCER FROM EXPOSURE TO
5 ACRYLAMIDE AND CONSUMPTION OF COFFEE?
6 A YES, SIR, IT IS.
7 MR. METZGER: YOUR HONOR, I WOULD OFFER THAT IN
8 EVIDENCE, EXHIBIT 120.
9 THE COURT: ANY OBJECTION?
10 MR. SCHURZ: YES, WE OBJECT; HEARSAY.
11 THE COURT: OBJECTION SUSTAINED.
12 (DISCUSSION HELD OFF THE RECORD.)
13 THE COURT: YOU MAY PROCEED.
14 Q BY MR. METZGER: DR. BAYARD, I'M NOW GOING
15 TO SHOW YOU A DOCUMENT THAT'S BEEN MARKED EXHIBIT 121,
16 ALSO ENTITLED "STATEMENT OF STEVEN BAYARD, PH.D."
17 IS EXHIBIT 121 A LIST OF ERRATA OR
18 CORRECTIONS THAT YOU PREPARED WITH REFERENCE TO YOUR
19 REPORT, WHICH IS EXHIBIT 120?
20 A YES, SIR.
21 (EXHIBIT 121 MARKED FOR IDENTIFICATION.)
22 Q BY MR. METZGER: NEXT I'M GOING TO PROVIDE
23 YOU A COPY OF WHAT'S BEEN MARKED AS EXHIBIT 122, A
24 DOCUMENT ENTITLED "ADDENDUM TO STATEMENT OF STEVEN P.
25 BAYARD," DATED APRIL 27, 2014; FURTHER TITLED "CANCER
26 RISK ASSESSMENT OF ACRYLAMIDE IN COFFEE, NOT INCLUDING
27 HARDERIAN TUMORS IN NTP MICE."
28 IS EXHIBIT 122 AN ADDENDUM TO YOUR REPORT,

1 YOUR QUANTITATIVE CANCER RISK ASSESSMENT, THAT YOU
2 PREPARED?
3 A YES, SIR.
4 (EXHIBIT 122 MARKED FOR IDENTIFICATION.)
5 Q BY MR. METZGER: OKAY. NOW I'M GOING TO
6 PROVIDE YOU WHAT'S BEEN MARKED EXHIBIT 123, A DOCUMENT
7 ENTITLED "STATEMENT OF STEVEN BAYARD; CALCULATION FOR
8 THE INCREASED RISK OF TWO CHILD ACUTE LEUKEMIAS DUE TO
9 MATERNAL COFFEE-DRINKING DURING PREGNANCY."
10 IS EXHIBIT 123 YOUR REPORT OF A QUANTITATIVE
11 CANCER RISK ASSESSMENT THAT YOU PREPARED TO ASSESS THE
12 RISK OF CHILDHOOD LEUKEMIA FROM MATERNAL CONSUMPTION OF
13 COFFEE DURING PREGNANCY?
14 A IT'S ACTUALLY TO ASSESS THE INCREASED RISK,
15 YES, SIR.
16 (EXHIBIT 123 MARKED FOR IDENTIFICATION.)
17 Q BY MR. METZGER: THANK YOU. OKAY.
18 AND LASTLY, I'M GOING TO SHOW YOU WHAT'S
19 BEEN MARKED AS PLAINTIFF'S EXHIBIT 128. AND IS EXHIBIT
20 128 A POWERPOINT PRESENTATION THAT YOU PREPARED, BASED
21 UPON YOUR WRITTEN REPORTS, TO FACILITATE THE
22 PRESENTATION AND UNDERSTANDING OF YOUR RISK ASSESSMENTS?
23 A YES, SIR.
24 (EXHIBIT 128 MARKED FOR IDENTIFICATION.)
25 Q BY MR. METZGER: ALL RIGHT. THANK YOU.
26 DR. BAYARD, WOULD YOU TELL THE COURT YOUR
27 DEGREES, WHEN YOU RECEIVED THEM, AND WHAT YOUR DEGREES
28 WERE IN.

1 A YES, SIR. I HAVE A BS IN MATHEMATICS FROM
2 TUFTS UNIVERSITY, IN MASSACHUSETTS, IN 1965; MINORS IN
3 BIOLOGY AND CHEMISTRY.
4 Q I'M SORRY, WHAT?
5 A MINORS IN BIOLOGY AND CHEMISTRY.
6 Q OKAY.
7 A MAJOR IN MATHEMATICS.
8 Q AND YOUR DOCTORAL DEGREE?
9 A MY DOCTORAL DEGREE IS IN BIOSTATISTICS, FROM
10 JOHNS HOPKINS UNIVERSITY, IN 1971; MAJOR IN
11 BIOSTATISTICS.
12 Q OKAY. AND HAVE YOU ALSO TAKEN GRADUATE
13 COURSES, IN ADDITION TO THOSE DEGREES, IN TOXICOLOGY?
14 A I HAVE.
15 Q AND WHERE DID YOU DO THAT?
16 A I DID THOSE AT M.I.T.
17 Q OKAY. WHAT IS YOUR FIELD OF EXPERTISE?
18 A BIOSTATISTICS AND RISK ASSESSMENT.
19 Q OKAY. HOW MANY YEARS OF EXPERIENCE DO YOU
20 HAVE AS A BIOSTATISTICIAN AND RISK ASSESSOR?
21 A WITH THE FEDERAL GOVERNMENT, I HAVE ABOUT 32
22 YEARS. MOST OF THAT, JUST SHY OF 30, WAS IN RISK
23 ASSESSMENT.
24 Q OKAY. AND FOR WHAT FEDERAL AGENCIES DID YOU
25 DO RISK ASSESSMENT WORK DURING YOUR GOVERNMENT SERVICE?
26 A I BEGAN WITH THE CONSUMER PRODUCTS SAFETY
27 COMMISSION. I LEFT THERE IN 1978 TO JOIN THE
28 ENVIRONMENTAL PROTECTION AGENCY. I WORKED THERE FOR 19

1 YEARS. AND FOLLOWING MY WORK AT EPA, I WENT TO THE
2 OCCUPATIONAL -- U.S. OCCUPATIONAL SAFETY AND HEALTH
3 AGENCY.
4 Q AND HOW LONG WERE YOU THERE?
5 A OSHA, TEN YEARS.
6 Q ALL RIGHT. AND HAVE YOU RETIRED FROM
7 GOVERNMENT SERVICE?
8 A I HAVE.
9 (DISCUSSION HELD OFF THE RECORD BETWEEN
10 THE COURT AND CLERK.)
11 Q BY MR. METZGER: WHAT IS RISK ASSESSMENT,
12 DR. BAYARD?
13 A IT'S TRYING TO ASSESS THE RISK OF TOXIC
14 CHEMICALS, AT LEAST IN MY FIELD.
15 Q WHEN YOU WERE EMPLOYED BY THE CONSUMER
16 PRODUCTS SAFETY COMMISSION, DID YOU HAVE ANY INVOLVEMENT
17 IN DEVELOPING THE QUANTITATIVE RISK ASSESSMENT PROCESS
18 FOR CANCER RISK?
19 A YES, I DID. I -- IT WAS A NEW AGENCY, AND
20 THEY HAD JUST GOT IT. SO THEY DIDN'T HAVE A PROCESS.
21 Q WHAT DID YOU DO?
22 A I DEVELOPED RISK ASSESSMENT, THE WHOLE RISK
23 ASSESSMENT PROCESS -- QUANTITATIVE PART OF THE RISK
24 ASSESSMENT PROCESS; AND I DID RISK ASSESSMENTS.
25 Q APPROXIMATELY HOW MANY QUANTITATIVE CANCER
26 RISK ASSESSMENTS DID YOU PERFORM WHEN YOU WERE AT THE
27 EPA?
28 A I'D SAY AT LEAST 20.

1 Q AND DID YOU HAVE INVOLVEMENT IN THE
2 DEVELOPMENT OF THE CANCER RISK ASSESSMENT FOR
3 ENVIRONMENTAL TOBACCO SMOKE?
4 A YES, I DID.
5 Q WHAT WAS YOUR INVOLVEMENT?
6 A I WAS THE PROJECT MANAGER. I MANAGED THE
7 WHOLE PROCESS.
8 Q OKAY.
9 A I WAS ALSO TECHNICAL EDITOR ON SOME OF THESE
10 SECTIONS AND CHAPTERS.
11 Q OKAY.
12 A AND I DID THE QUANTITATIVE RISK ASSESSMENT
13 FOR CHILDHOOD RESPIRATORY EFFECTS FROM ENVIRONMENTAL
14 TOBACCO SMOKE. SO I WAS ALSO AN AUTHOR.
15 Q OKAY. HAS THE FEDERAL OCCUPATIONAL SAFETY
16 AND HEALTH ADMINISTRATION HAD AN OFFICE OF RISK
17 ASSESSMENT?
18 A YES, THEY DO.
19 Q AND WERE YOU THE DIRECTOR OF THAT OFFICE?
20 A YES, I WAS.
21 Q OKAY. HAS THE NATIONAL CANCER INSTITUTE
22 ISSUED A MONOGRAPH ON HEALTH EFFECTS OF PASSIVE SMOKING?
23 A YES, THEY HAVE.
24 Q AND WERE YOU THE SCIENTIFIC EDITOR FOR THAT
25 MONOGRAPH?
26 A I WAS.
27 Q WOULD YOU TELL THE COURT BRIEFLY ABOUT YOUR
28 EXPERIENCE TEACHING RISK ASSESSMENT.

1 A MY EXPERIENCE TEACHING --
2 Q YES.
3 A -- CANCER RISK ASSESSMENT, OR TEACHING?
4 Q TEACHING.
5 A OH, MY TEACHING EXPERIENCE.
6 Q YES.
7 A I HAD -- AFTER I LEFT GRADUATE SCHOOL, I WAS
8 HIRED AS AN ASSISTANT PROFESSOR AT YALE UNIVERSITY. AND
9 I TAUGHT GRADUATE COURSES IN STATISTICS, BIOSTATISTICS,
10 EPIDEMIOLOGY, AND TOMOGRAPHY.
11 LATER ON IN MY CAREER, I TAUGHT GRADUATE
12 COURSES IN STATISTICS AND EPIDEMIOLOGY AT THE UNIFORM
13 SERVICES UNIVERSITY OF CAL SCIENCES, WHICH IS BASICALLY
14 THE MILITARY MEDICAL SCHOOL.
15 Q OKAY. AND HAVE YOU TAUGHT A COURSE IN
16 CARCINOGEN RISK ASSESSMENT FOR THE PAN AMERICAN HEALTH
17 ORGANIZATION?
18 A I TAUGHT THAT, BUT THAT WAS A LONG TIME AGO.
19 Q OKAY. WOULD YOU TELL THE COURT BRIEFLY
20 ABOUT YOUR EXPERIENCE IN CONSULTATION REGARDING RISK
21 ASSESSMENT.
22 A WELL, I'VE DONE RISK -- AS A CONSULTANT,
23 AFTER I RETIRED, I DID BERYLLIUM RISK ASSESSMENT FOR
24 OSHA.
25 Q OKAY.
26 A I DID ANOTHER RISK ASSESSMENT FOR ACRYLAMIDE
27 IN WATER -- NOT DRINKING WATER, BUT WATER.
28 Q OKAY.

1 A AND I DID A RISK ASSESSMENT FOR
2 ENVIRONMENTAL TOBACCO SMOKE AND EXPOSURE AT A CASINO.
3 Q OKAY. AND WOULD YOU TELL THE COURT ABOUT
4 SOME OF THE AWARDS THAT YOU HAVE RECEIVED FOR YOUR WORK
5 IN RISK ASSESSMENT AND BIOSTATISTICS.
6 A YES. AT THE CONSUMER PRODUCTS SAFETY
7 COMMISSION, I RECEIVED THE CHAIRMAN'S SPECIAL AWARD FOR
8 DEVELOPMENT OF THE RISK ASSESSMENT PROCESS.
9 I ALSO HELPED TO WRITE THE 1986
10 GUIDELINES -- CANCER RISK ASSESSMENT GUIDELINES FOR EPA,
11 AND I RECEIVED AN AWARD FOR THAT. THAT WAS KIND OF A
12 TEAM AWARD.
13 FOR MY WORK ON ENVIRONMENTAL TOBACCO SMOKE,
14 I RECEIVED THE -- IT WAS THE EPA GOLD MEDAL AWARD,
15 WHICH -- THE EPA HAS 22,000 PEOPLE, AND THERE WERE
16 ONLY -- WELL, EVEN AT THE TIME, THERE WERE ONLY TWO GOLD
17 MEDALS GIVEN THAT YEAR, AND I RECEIVED ONE OF THEM.
18 AND I RECEIVED ANOTHER AWARD FOR THAT WORK,
19 ALSO, AND AN EPA STATISTICIANS AWARD; A FEW OTHERS.
20 Q HAVE YOU SERVED AS AN EXPERT WITNESS ON
21 BEHALF OF THE FEDERAL GOVERNMENT?
22 A YES, I HAVE.
23 Q IN WHAT CONTEXT?
24 A SEVERAL.
25 Q PLEASE TELL THE COURT.
26 A THE FIRST ONE WAS FOR MY WORK ON DIOXIN. I
27 DID A RISK ASSESSMENT ON DIOXIN. DIOXIN IS A
28 CONTAMINANT OF AGENT ORANGE. SO I WAS CALLED I AS A --

1 I WAS DEPOSED AS A WITNESS IN THE VETERANS' AGENT ORANGE
2 SUIT AGAINST THE GOVERNMENT.
3 I WAS -- I DID A LOT OF TESTIFYING FOR THE
4 EPA ON ENVIRONMENTAL TOBACCO SMOKE. I TESTIFIED BEFORE
5 CONGRESS, BEFORE THE SENATE IN MARYLAND, AND I THINK THE
6 HOUSE IN VIRGINIA AND PENNSYLVANIA; SEVERAL LOCAL AREAS.
7 Q OKAY. AND WERE YOU AN EXPERT WITNESS FOR --
8 STRIKE THAT. EXCUSE ME.
9 WERE YOU A WITNESS FOR THE UNITED STATES
10 DEPARTMENT OF JUSTICE IN THE TOBACCO LITIGATION?
11 A OH, YES. I WAS IN THERE TOO.
12 Q OKAY. ALL RIGHT. I'D LIKE TO ASK YOU ABOUT
13 SOME OF THE RISK ASSESSMENTS THAT YOU'VE DONE; AND
14 SPECIFICALLY, FOCUSING ON CARCINOGENS IN SOMETHING.
15 A YES, SIR.
16 Q OKAY. SO FOR EXAMPLE, DID YOU DO A RISK
17 ASSESSMENT OF ASBESTOS?
18 A I DID.
19 Q AND COULD YOU TELL US WHAT THE CONTEXT OF
20 THAT WAS.
21 A I DID SEVERAL, BUT MY FIRST RISK ASSESSMENT
22 OF ASBESTOS WAS ASBESTOS IN WALL JOINT COMPOUND.
23 Q WALL JOINT COMPOUND?
24 A WALL JOINT COMPOUND, YES. THAT'S MATERIAL
25 THAT YOU SPRAY BETWEEN THE SHEET ROCK TO EVEN IT UP.
26 Q OKAY. AND WHEN YOU DID THAT, WAS THAT A
27 QUANTITATIVE CANCER RISK ASSESSMENT?
28 A YES, IT WAS.

1 Q AND WHEN YOU DID THAT RISK ASSESSMENT, DID
2 YOU QUANTIFY THE CANCER RISK OF THE WALL JOINT COMPOUND
3 OR THE ASBESTOS?
4 A OH, THE ASBESTOS IN THE WALL JOINT COMPOUND.
5 Q AND WHY DID YOU QUANTIFY THE CANCER RISK FOR
6 THE ASBESTOS RATHER THAN THE MEDIUM IN WHICH THE
7 ASBESTOS EXISTED, THE WALL -- THE JOINT COMPOUND?
8 A WELL, ASBESTOS IS A CARCINOGEN. THE WALL
9 JOINT COMPOUND ISN'T THE CARCINOGEN; THE ASBESTOS IS.
10 Q AND IN QUANTIFYING CANCER RISK, HAS IT
11 ALWAYS BEEN YOUR PRACTICE TO QUANTIFY THE CANCER RISK OF
12 THE CARCINOGEN?
13 A YES, YES.
14 Q ALL RIGHT. HAVE YOU DONE A QUANTITATIVE
15 CANCER RISK ASSESSMENT FOR BENZENE?
16 A YES, I HAVE.
17 Q IN WHAT CONTEXT?
18 A I DID BENZENE IN PAINT STRIPPERS.
19 Q OKAY. AND WHEN YOU DID THAT QUANTITATIVE
20 CANCER RISK ASSESSMENT, DID YOU QUANTIFY THE CANCER RISK
21 FROM BENZENE OR FROM THE PAINT STRIPPER?
22 A I QUANTIFIED IT FROM THE BENZENE IN THE
23 PAINT STRIPPER.
24 Q AND WHY DID YOU DO IT THAT WAY, RATHER THAN
25 QUANTIFYING THE RISK FOR A PAINT STRIPPER?
26 A BECAUSE BENZENE IS THE CARCINOGEN; IT'S NOT
27 THE PAINT STRIPPER ITSELF.
28 Q OKAY.

1 THE COURT: LET ME ASK YOU THIS: IS THE RISK
2 AFFECTED BY DILUTION?
3 THE WITNESS: THE RISK IS AFFECTED BY -- IT'S
4 AFFECTED BY THE EXPOSURE THAT THE PERSON GETS.
5 THE COURT: WELL, DOES SOMEBODY GET EXPOSED TO THE
6 PARTICULAR ITEM INSIDE IT, OR IS IT EXPOSED TO THE
7 PRODUCT ITSELF?
8 THE WITNESS: WELL, IT'S EXPOSED TO THE BENZENE IN
9 THE PRODUCT. AND SO ONE -- WHEN I DO THE RISK
10 ASSESSMENTS, I HAVE TO FACTOR IN THE AMOUNT OF BENZENE
11 IN THE PRODUCT AND HOW MUCH OF THAT GETS INTO THE
12 PERSON.
13 THE COURT: ALL RIGHT. SO YOU WOULD ANALYZE THE
14 MIXTURE ITSELF?
15 THE WITNESS: NO, I -- THE RISK IS BASED ON THE
16 AMOUNT OF BENZENE FROM THE PRODUCT THAT GETS INTO THE
17 PERSON.
18 THE COURT: GETS INTO THE PERSON THROUGH THE
19 PRODUCT?
20 THE WITNESS: YES, SIR. THROUGH THE USE OF THE
21 PRODUCT.
22 THE COURT: OKAY.
23 THE WITNESS: SO THE PERSON IS PUTTING PAINT
24 STRIPPERS ON. AND THE BENZENE VAPORIZES, AND THE PERSON
25 ABSORBS THAT BENZENE EITHER THROUGH BREATHING IT OR
26 THROUGH HIS HANDS, IF HE HAS IT.
27 THE COURT: FOR EXAMPLE, LET'S SAY THERE'S -- I'M
28 MAKING THIS UP. LET'S SAY THERE'S ARSENIC IN WATER.

1 THE WITNESS: YES, SIR.
2 THE COURT: DO YOU LOOK AT WHETHER THERE'S A
3 CANCER RISK FOR -- OR OTHER HARMFUL EFFECTS FROM
4 ARSENIC, OR DO YOU LOOK AT IT THAT THE ARSENIC IS 1 PART
5 PER 10 MILLION GALLONS OF WATER?
6 THE WITNESS: WELL, THE SMALLER THE AMOUNT OF
7 ARSENIC IN THE WATER, THE LESS OF IT THAT'S GOING TO GET
8 INTO THE PERSON.
9 BUT WHEN -- KIND OF WHAT A RISK ASSESSOR
10 DOES IS MEASURE HOW MANY OF THAT ARSENIC IS GOING TO BE
11 ABSORBED BY THE PERSON AND BE AN EFFECTIVE DOSE BY THE
12 PERSON.
13 SO YOU DON'T ANALYZE HOW MUCH WATER GETS IN,
14 ONLY TO THE EXTENT THAT YOU NEED TO KNOW HOW MUCH
15 ARSENIC GETS INTO THE PERSON: HOW MUCH ARE THEY
16 DRINKING AND HOW MUCH THEY'RE CONSUMING THAT THEREAFTER
17 IS AFFECTING THEM.
18 THE COURT: ALL RIGHT. THANK YOU.
19 MR. METZGER.
20 THE WITNESS: THANK YOU.
21 Q BY MR. METZGER: WELL, ON THIS POINT, DR.
22 BAYARD, HIS HONOR CHOSE THE EXAMPLE OF ARSENIC IN WATER.
23 IF ARSENIC IS PRESENT IN WATER AT A PARTICULAR -- SAY,
24 BOTTLED WATER AT A CONCENTRATION OF 1 PART PER MILLION
25 IN THAT WATER, AND ANOTHER BOTTLED WATER HAS ARSENIC IN
26 IT AT A CONCENTRATION OF 1 PART PER BILLION, DOES THE
27 RISK ASSESSMENT TAKE THAT DIFFERENCE INTO ACCOUNT IN
28 ASSESSING THE RISK FOR ONE BOTTLE -- OF ARSENIC FROM ONE

1 BOTTLE OF WATER VERSUS THE OTHER?
2 A THAT WILL AFFECT HOW MUCH -- THE AMOUNT OF
3 ARSENIC IN THE WATER WILL AFFECT HOW MUCH THE PERSON
4 ABSORBS.
5 Q OKAY. SO THE DILUTION OF THE CARCINOGEN IN
6 THE MIXTURE ULTIMATELY IS ACCOUNTED FOR IN THE
7 QUANTITATIVE CANCER RISK ASSESSMENT?
8 A THAT'S PRETTY MUCH WHAT MATTERS, THAT AND
9 HOW MUCH WATER THE PERSON WILL DRINK THAT CONTAINS THE
10 ARSENIC.
11 Q OKAY. ALL RIGHT. HAVE YOU DONE A
12 QUANTITATIVE CANCER RISK ASSESSMENT FOR FORMALDEHYDE?
13 A I DID ONE.
14 Q IN WHAT CONTEXT?
15 A FORMALDEHYDE IN FOAM BOARD THAT WAS ACTUALLY
16 USED IN TRAILERS.
17 Q THAT SOUNDS LIKE KATRINA WE'RE TALKING
18 ABOUT, OR SOMETHING.
19 A WHAT?
20 Q ARE WE TALKING ABOUT KATRINA?
21 A NO, THIS IS EARLIER THAN THAT.
22 Q OH, EARLIER. OKAY.
23 IN ANY EVENT, WHEN YOU DID THAT QUANTITATIVE
24 CANCER RISK ASSESSMENT, DID YOU QUANTIFY THE CANCER RISK
25 FROM FORMALDEHYDE OR FROM THE FOAM INSULATION BOARD?
26 A NO, FROM THE FORMALDEHYDE THAT EVAPORATED
27 FROM THE FOAM INSULATION BOARD BECAUSE OF THE CLOSED
28 TRAILERS.

1 Q OKAY. AND WHY DID YOU QUANTIFY THE CANCER
2 RISK FROM FORMALDEHYDE RATHER THAN THE RISK OF CANCER
3 FROM THE FOAM INSULATION BOARD?
4 A WELL, FORMALDEHYDE IS THE CARCINOGEN, AND
5 THAT'S WHAT GOT INTO THE PERSON'S BODY.
6 Q OKAY. HAVE YOU DONE A CANCER RISK
7 ASSESSMENT FOR A CHEMICAL CALLED -- A CARCINOGEN, I
8 UNDERSTAND IT, CALLED TRIS?
9 A YES. IT'S TRIS 23 DIBROMOPROPYL PHOSPHATE.
10 Q I'M GLAD YOU COULD SAY THAT.
11 OKAY. AND WHAT WAS THE CONTEXT OF THAT
12 QUANTITATIVE CANCER RISK ASSESSMENT THAT YOU DID?
13 A TRIS IS A -- I DON'T KNOW IF THEY USE IT
14 ANYMORE; I DOUBT IT -- A FLAME RETARDANT OFTEN USED IN
15 CLOTHING, AND ESPECIALLY CHILDREN'S PAJAMAS AND
16 CHILDREN'S CLOTHING.
17 Q WAS THAT THE CONTEXT THAT YOU ASSESSED THE
18 RISK?
19 A THAT'S CORRECT.
20 Q ALL RIGHT. AND WHEN YOU QUANTIFIED THE
21 CANCER RISK OF TRIS --
22 A YES.
23 Q -- WELL, WHEN YOU DID THAT RISK ASSESSMENT,
24 DID YOU QUANTIFY THE CANCER RISK TO THE CHILDREN OF TRIS
25 OR THE COTTON OR POLYESTER IN THE PAJAMAS?
26 A NO, IT WAS THE TRIS THAT LEACHED OUT OF THE
27 PAJAMAS ONTO THE CHILDREN'S SKIN AND WAS ABSORBED BY THE
28 CHILD.

1 Q OKAY. AND WHY DID YOU NOT QUANTIFY THE
2 CANCER RISK FROM THE COTTON?
3 A WELL, COTTON IS NOT A CARCINOGEN; TRIS IS.
4 Q OKAY. HAD HAVE YOU DONE A CANCER RISK
5 ASSESSMENT FOR -- REGARDING AGENT ORANGE?
6 A I DID.
7 Q AND IS AGENT ORANGE A MIXTURE OF TWO
8 CHEMICALS?
9 A YES, IT IS.
10 Q DO YOU KNOW WHAT THOSE ARE?
11 A YEAH, I DO. IT'S 2,4-D, WHICH IS ROUNDUP;
12 AND IT'S 2,4,5 TRI -- OH, GOD. TRICHLORO --
13 TRICHLORODIOXYACETIC ACID (SIC), I THINK.
14 Q OKAY. CAN WE CALL IT 2,4-D?
15 THE COURT:
16 A NO, IT'S TRICHLOROPHENOXYACETIC ACID.
17 Q ALL RIGHT. IN ANY EVENT, WHEN DID YOUR
18 QUANTITATIVE CANCER RISK ASSESSMENT OF AGENT ORANGE, DID
19 YOU DO IT OF THE 2,4-D OR THE -- WHAT IS IT, THE TCP OR
20 WHATEVER YOU JUST CALLED IT?
21 A IT'S CALLED TRI-TCP.
22 NO, I DIDN'T DO IT ON 2,4-D BECAUSE 2,4-D
23 ISN'T A CARCINOGEN.
24 Q DID YOU DO IT ON TCP?
25 A NO, I DIDN'T DO IT ON 2,4,5-TCP BECAUSE
26 THAT'S NOT A CARCINOGEN, EITHER.
27 Q WELL, WHAT DID YOU DO THE CANCER RISK
28 ASSESSMENT ON?

1 A I DID IT ON THE DIOXIN CONTAMINANT OF THAT
2 HERBICIDE.
3 Q I SEE. AND WAS DIOXIN THE CARCINOGEN?
4 A DIOXIN IS THE MOST POWERFUL CARCINOGEN KNOWN
5 TO HUMANS, BUT IT'S -- AND RATS.
6 Q OKAY. AND WHEN YOU DID THAT CANCER RISK
7 ASSESSMENT OF AGENT ORANGE AND YOU DID IT FOR THE
8 CARCINOGEN TCDD --
9 A UH-HUH.
10 Q -- OR DIOXIN --
11 A YES, THAT'S DIOXIN.
12 Q -- WHAT WAS THE CONCENTRATION OF DIOXIN IN
13 THE AGENT ORANGE?
14 A IT WAS REALLY SMALL. I THINK IT WAS IN
15 PARTS PER TRILLION.
16 Q PARTS PER TRILLION?
17 A HANG ON JUST A MINUTE. NANOGRAMS IS -- YES,
18 IT WAS IN PARTS PER TRILLION, I BELIEVE.
19 Q OKAY. WELL, LET ME ASK YOU THIS: WHY COULD
20 YOU NOT -- WHY DID YOU NOT DO THE CANCER RISK ASSESSMENT
21 FOR THE AGENT ORANGE MIXTURE AND ASSUME THAT THERE WAS
22 NO CARCINOGENIC HAZARD FROM THE DIOXIN BECAUSE IT WAS
23 JUST IN THERE IN THE CONCENTRATION OF SOME PARTS PER
24 TRILLION?
25 MR. SCHURZ: OBJECTION; INCOMPLETE HYPOTHETICAL,
26 UNINTELLIGIBLE, AND LEADING.
27 THE COURT: OVERRULED.
28 THE WITNESS: OVERRULED?

1 DIOXIN IS A CARCINOGEN. THE OTHER PRODUCTS
2 WERE NOT CARCINOGENS.
3 Q BY MR. METZGER: I UNDERSTAND. BUT SINCE
4 DIOXIN WAS IN THERE AT A CONCENTRATION OF JUST PARTS PER
5 TRILLION, COULDN'T YOU JUST ASSUME THAT IT DIDN'T CAUSE
6 CANCER AND STOP YOUR RISK ASSESSMENT THERE?
7 A YOU DON'T KNOW THAT UNLESS YOU DO THE
8 CALCULATIONS.
9 Q OKAY. ALL RIGHT.
10 DID YOU ALSO DO A CANCER RISK ASSESSMENT OF
11 DIOXIN IN TOXIC WASTE?
12 A YES, I DID.
13 Q WHEN YOU DID THAT CANCER RISK ASSESSMENT,
14 DID YOU QUANTIFY THE RISK OF CANCER FROM THE TOXIC WASTE
15 OR FROM THE DIOXIN?
16 A I QUANTIFIED IT FROM THE DIOXIN IN THE TOXIC
17 WASTE.
18 Q WHY DID YOU QUANTIFY IT FROM THE DIOXIN
19 RATHER THAN FROM THE TOXIC WASTE?
20 A BECAUSE I DIDN'T HAVE ANY KNOWLEDGE THAT THE
21 TOXIC WASTE WAS CARCINOGENIC, BUT I KNEW THAT THE DIOXIN
22 WAS AN ACTIVE CARCINOGEN IN THERE.
23 Q OKAY. HIS HONOR BROUGHT UP THE EXAMPLE OF
24 ARSENIC IN DRINKING WATER. HAVE CANCER RISK ASSESSMENTS
25 BEEN DONE OF ARSENIC IN DRINKING WATER?
26 A YES. I THINK THE NATIONAL ACADEMY OF
27 SCIENCES DID ONE IN 1987, '88.
28 Q OKAY. AND WHEN THE NATIONAL ACADEMY OF

1 SCIENCES DID THAT CANCER RISK ASSESSMENT OF ARSENIC IN
2 DRINKING WATER, DID THEY QUANTIFY THE CANCER RISK FROM
3 THE DRINKING WATER OR FROM THE ARSENIC IN THE DRINKING
4 WATER?
5 A NO, THAT WAS THE ARSENIC IN THE DRINKING
6 WATER.
7 Q OKAY. HAVE CANCER RISK ASSESSMENTS BEEN
8 DONE OF DISINFECTION BYPRODUCTS IN WATER?
9 A YES.
10 Q OKAY. AND WHEN THOSE HAVE BEEN DONE, WAS
11 THE CANCER RISK ASSESSED FOR THE WATER OR FOR THE
12 DISINFECTION BYPRODUCTS?
13 A I BELIEVE THE RISK ASSESSMENTS WERE DONE FOR
14 WHAT WOULD BE TERMED TRICHOLORO HALENES -- HALENE (SIC):
15 CHLORINE, BROMINE, FLUORINE. AND THE RISK ASSESSMENTS
16 WERE ALWAYS DONE FOR THE CHEMICAL CONTAMINANTS OF THE
17 WATER.
18 Q WERE THOSE CARCINOGENS?
19 A YES.
20 Q OKAY. ALL RIGHT. HAVE RISK ASSESSMENTS
21 BEEN DONE FOR RADON IN DRINKING WATER?
22 A OH, YES.
23 Q AND WHEN THOSE RISK ASSESSMENTS WERE DONE,
24 DID THE RISK ASSESSORS QUANTIFY THE CANCER HAZARD OF
25 WATER OR OF THE RADIOACTIVE RADON?
26 A THE RADON IN THE WATER.
27 Q AND WAS THAT BECAUSE RADON WAS A CARCINOGEN?
28 A THAT'S RIGHT.

1 Q ALL RIGHT. YOU MENTIONED, I THINK, EARLIER
2 THAT YOU HAD EARLIER DONE A RISK ASSESSMENT, A CANCER
3 RISK ASSESSMENT, OF ACRYLAMIDE IN WATER?
4 A YES, I DID.
5 Q WOULD YOU TELL US WHAT THE CONTEXT OF THAT
6 WAS.
7 A YES. IT WAS ACTUALLY FOR COAL MINERS
8 EXPOSED TO THE ACRYLAMIDE, WHICH IS USED AS A FLOCCULANT
9 FOR COAL. SO IT HELPS THE SLAGS SINK TO THE BOTTOM IN
10 THE COAL-MINING PROCESS.
11 AND ACRYLAMIDE DOES THIS, BUT THAT'S
12 POLYACRYLAMIDE. AND THE MONOMER ACRYLAMIDE IS WHAT IS A
13 CARCINOGEN. AND SO THE MONOMER IS A CONTAMINATE OF THE
14 POLYMER, WHICH IS USED AS THE FLOCCULANT.
15 SO I DID A RISK ASSESSMENT FOR THE MINERS
16 WHO WERE IN THE WATER AND WERE EXPOSED TO THE WATER, NOT
17 THROUGH DRINKING IT BUT ACTUALLY THROUGH GOING -- WADING
18 IN THE WATER TO REMOVE SOME OF THE FLOCCULANT MATERIAL.
19 AND SO I HAD TO DO A RISK ASSESSMENT TO
20 ESTIMATE -- CALCULATE THE RISK OF THE WATER THAT GOT
21 INTO THE MINERS THROUGH ABSORPTION THROUGH THE SKIN.
22 AND IT ISN'T THE WATER THAT REALLY GOT THROUGH --
23 ABSORBED THROUGH THE SKIN; IT WAS THE ACRYLAMIDE THAT
24 GOT ABSORBED THROUGH THE SKIN.
25 Q AND WHEN YOU DID THAT CANCER RISK --
26 QUANTITATIVE CANCER RISK ASSESSMENT OF ACRYLAMIDE IN
27 WATER, DID YOU ASSESS THE RISK OF WATER OR THE
28 POLYACRYLAMIDE FLOCCULANT OR THE ACRYLAMIDE?

1 A IT WAS THE ACRYLAMIDE THAT HAD THE EFFECTIVE
2 CANCER RISK.
3 Q AND WHY DID THE ACRYLAMIDE HAVE TO BE
4 ASSESSED FOR CANCER RISK RATHER THAN THE WATER OR THE
5 POLYACRYLAMIDE?
6 A WELL, NUMBER ONE, ACRYLAMIDE IS THE
7 CARCINOGEN. AND NUMBER TWO, ACRYLAMIDE IS WHAT GOT
8 ABSORBED THROUGH THE SKIN.
9 Q OKAY. IN EVERY CANCER RISK ASSESSMENT THAT
10 YOU HAVE DONE, HAVE YOU DONE THE CANCER RISK ASSESSMENT
11 OF THE -- THE QUANTITATIVE CANCER RISK ASSESSMENT OF THE
12 CARCINOGEN?
13 A ALWAYS OF -- IT'S DUE TO THE -- THE RISK IS
14 DUE TO THE CARCINOGEN EXPOSURE.
15 Q OKAY. NOW, WE HAVE HEARD DURING THIS TRIAL
16 THAT THERE ARE TWO TYPES OF RISK ASSESSMENTS: ONE
17 CALLED A QUANTITATIVE CANCER RISK ASSESSMENT, THE OTHER
18 CALLED A QUALITATIVE RISK ASSESSMENT -- OR QUALITATIVE
19 CANCER RISK ASSESSMENT.
20 ARE YOU FAMILIAR WITH THE TWO DIFFERENT
21 TYPES?
22 A YES.
23 Q ALL RIGHT. AND IN WHAT CONTEXT ARE
24 QUALITATIVE CANCER RISK ASSESSMENTS DONE?
25 A A QUALITATIVE RISK ASSESSMENT, IN MY
26 CONTEXT, IS A HAZARD ASSESSMENT: CAN THE MATERIAL CAUSE
27 CANCER? IS IT DEEMED A CARCINOGEN?
28 Q OKAY. AND HAVE GOVERNMENTAL AGENCIES DONE

1 QUALITATIVE CANCER RISK ASSESSMENTS TO DETERMINE WHETHER
2 CHEMICALS ARE CARCINOGENS?
3 A THAT'S EXACTLY WHY THEY DO THEM.
4 Q OKAY. AND ONCE A CHEMICAL IS DETERMINED TO
5 BE A CARCINOGEN, IF ONE WANTS TO ASSESS THE DEGREE OF
6 RISK TO PEOPLE FROM EXPOSURE TO THAT CARCINOGEN, WHAT
7 TYPE OF CANCER RISK ASSESSMENT DO YOU DO?
8 A YOU HAVE TO DO A QUANTITATIVE RISK
9 ASSESSMENT.
10 Q OKAY. I'D LIKE TO SHOW YOU WHAT'S
11 PREVIOUSLY BEEN MARKED AS -- THIS IS DEFENDANT'S EXHIBIT
12 10255, "THE SUPPLEMENTARY GUIDANCE FOR CONDUCTING HEALTH
13 RISK ASSESSMENT OF CHEMICAL MIXTURES."
14 ARE YOU FAMILIAR WITH THAT?
15 A IS THAT THE 2000?
16 Q YES.
17 A I'M SOMEWHAT FAMILIAR WITH IT.
18 Q OKAY. DOES THIS GUIDANCE FOR THE CONDUCT OF
19 HEALTH RISK ASSESSMENTS OF CHEMICAL MIXTURES PROVIDE ANY
20 INSIGHT AS TO WHETHER, IN DOING A CANCER RISK ASSESSMENT
21 OF A MIXTURE -- WHETHER ONE SHOULD ASSESS THE RISK OF
22 CANCER OF THE CARCINOGEN OR THE MEDIUM IN WHICH THE
23 CARCINOGEN EXISTS, THE SO-CALLED MIXTURE?
24 A CAN YOU REPEAT THAT, PLEASE.
25 Q I'M SORRY. IT WAS PROBABLY OBTUSE.
26 DOES THIS EPA GUIDANCE DOCUMENT PROVIDE
27 INSIGHT AS TO WHAT A RISK ASSESSOR, SUCH AS YOURSELF,
28 SHOULD ASSESS WHEN ASSESSING THE RISK OF CANCER FROM A

1 MIXTURE?
2 A IT GIVES MULTIPLE GUIDANCE, BUT IT SAYS YOU
3 CAN EITHER ASSESS THE MATERIAL FROM THE MIXTURE OR FROM
4 COMPONENTS IN THE MIXTURE.
5 Q OKAY.
6 A AND SO IF THE MIXTURE IS A CARCINOGEN
7 ITSELF, IF IT'S KNOWN TO BE A CARCINOGEN -- FOR
8 INSTANCE, LIKE TOBACCO SMOKE IS -- THEN YOU CAN ASSESS
9 THE RISK FROM THE MIXTURE.
10 IF THE MIXTURE IS NOT KNOWN TO BE A
11 CARCINOGEN, THEN ONE CAN ASSESS THE RISK FROM THE
12 CARCINOGENIC COMPONENTS IN THE MIXTURE.
13 Q AND WHERE IN THIS GUIDANCE IS THAT STATED?
14 A (REVIEWS DOCUMENT.)
15 I'M SORRY. I WANT TO GET IT FROM THE
16 EXECUTIVE SUMMARY.
17 (REVIEWS DOCUMENT.)
18 WOULD IT BE BETTER TO COME BACK TO THAT
19 LATER?
20 Q LET ME JUST ASK YOU TO TURN TO PAGE 6 AND
21 ASK YOU IF THIS IS THE SECTION THAT YOU'RE REFERRING TO.
22 MR. SCHURZ: WELL, I'M GOING TO OBJECT AS LEADING.
23 THE WITNESS COULDN'T FIND IT, AND NOW MR. METZGER
24 POINTING HIM TO IT DOES NOT AID IN THE PRESENTATION OF
25 THIS EVIDENCE; LEADING.
26 THE COURT: OVERRULED. MR. METZGER IS DIRECTING
27 THE WITNESS TO PAGE 6.
28 THE WITNESS: ON PAGE 6. THANKS. THE PROCEDURES

1 FOR SELECTING A RISK ASSESSMENT METHOD. "FINALLY" --
2 QUOTE:
3 "FINALLY, THE THIRD APPROACH IS TO
4 EVALUATE THE MIXTURES THROUGH AN ANALYSIS OF
5 ITS COMPONENTS. FOR EXAMPLE" --
6 THE COURT: I DON'T THINK IT'S NECESSARY TO READ
7 IT.
8 WHAT'S THE QUESTION?
9 Q BY MR. METZGER: OKAY. SO ARE THERE -- DOES
10 THIS PROVIDE THREE APPROACHES, ONE OF WHICH IS TO
11 EVALUATE THE MIXTURE THROUGH AN ANALYSIS OF ITS
12 COMPONENTS?
13 A YES, SIR.
14 Q ALL RIGHT. THANK YOU VERY MUCH.
15 ALL RIGHT. LET ME ASK YOU THIS, DR. BAYARD:
16 CAN YOU THINK OF ANY QUALITATIVE CANCER RISK ASSESSMENT
17 THAT HAS EVER BEEN DONE OTHER THAN TO DETERMINE WHETHER
18 SOMETHING CAUSES CANCER?
19 A NO. THAT'S WHAT A QUALITATIVE RISK
20 ASSESSMENT IS.
21 Q AND HAVE YOU EVER DONE A QUANTITATIVE CANCER
22 RISK ASSESSMENT OTHER THAN TO DETERMINE THE RISK OF
23 CANCER FROM A CARCINOGEN IN A MIXTURE?
24 A I DON'T KNOW IF I --
25 Q LET ME TRY AGAIN. HAVE YOU EVER DONE A
26 QUANTITATIVE CANCER RISK ASSESSMENT FOR SOMETHING THAT
27 IS -- LET ME TRY ONE MORE TIME.
28 PRIOR TO ANYTHING THAT YOU DID IN THIS

1 CASE --
2 A UH-HUH.
3 Q -- HAVE YOU EVER DONE A QUANTITATIVE CANCER
4 RISK ASSESSMENT FOR SOMETHING THAT IS NOT KNOWN TO CAUSE
5 CANCER?
6 A NO, I HAVEN'T.
7 Q ALL RIGHT. I'D LIKE YOU TO ASSUME THAT A
8 CHEMICAL MIXTURE IS NOT KNOWN TO CAUSE CANCER. WOULD IT
9 BE SCIENTIFICALLY VALID TO ASSUME THAT A CARCINOGEN IN
10 THAT MIXTURE DOES NOT INCREASE THE RISK OF CANCER?
11 A NOT UNLESS YOU'VE DONE THE QUANTITATIVE RISK
12 ASSESSMENT.
13 Q OKAY. AND WHY NOT?
14 A BECAUSE YOU HAVE TO DO THE CALCULATIONS TO
15 FIND OUT WHAT THE RISK IS.
16 Q NOW, WHEN YOU DID THE CANCER RISK ASSESSMENT
17 OF ENVIRONMENTAL TOBACCO SMOKE, WHAT TYPE OF CANCER RISK
18 ASSESSMENT DID YOU DO?
19 A WE DID BOTH A QUALITATIVE AND A QUANTITATIVE
20 RISK ASSESSMENT.
21 Q OKAY. AND WHEN YOU DID THE QUALITATIVE RISK
22 ASSESSMENT, WHAT WAS THAT RISK ASSESSMENT OF, OR ITS
23 PURPOSE?
24 A TO DETERMINE -- WELL, IT WAS ACTUALLY TWO
25 PURPOSES; BUT ONE WAS TO DETERMINE WHETHER OR NOT
26 ENVIRONMENTAL TOBACCO SMOKE SHOULD BE CONSIDERED A
27 CARCINOGEN AND IN WHAT CATEGORY OF CARCINOGEN.
28 Q OKAY. AND WHEN YOU DID THE -- YOU ALSO DID

1 A QUANTITATIVE CANCER RISK ASSESSMENT OF ENVIRONMENTAL
2 TOBACCO SMOKE?
3 A YES.
4 Q WHY DID YOU DO THAT TYPE OF RISK ASSESSMENT
5 FOR ENVIRONMENTAL TOBACCO SMOKE, AS WELL?
6 A "THAT," MEANING QUANTITATIVE OR --
7 Q YES.
8 A TO DETERMINE THE RISK TO NONSMOKERS EXPOSED
9 TO ENVIRONMENTAL TOBACCO SMOKE, THE INCREASED RISK --
10 CANCER RISK -- LUNG CANCER RISK; I'M SORRY. WE DID IT
11 ONLY FOR LUNG CANCER.
12 Q OKAY. NOW, YOU'VE TOLD US ABOUT NUMEROUS
13 RISK ASSESSMENTS, CANCER RISK ASSESSMENTS, THAT YOU'VE
14 DONE. HAVE MOST OF THE CANCER RISK ASSESSMENTS THAT
15 YOU'VE DONE BEEN BASED UPON ANIMAL DATA?
16 A I'D SAY MOST OF THEM HAVE.
17 Q OKAY. AND WHY HAVE MOST OF THEM BEEN BASED
18 ON ANIMAL DATA RATHER THAN HUMAN DATA?
19 A BECAUSE WHEN -- THERE AREN'T THAT MANY KNOWN
20 HUMAN CARCINOGENS. I THINK WHEN I LEFT EPA, THERE WERE
21 AROUND 24 OR 25. AND THERE ARE HUNDREDS OF ANIMALS
22 KNOWN -- KNOWN ANIMAL CARCINOGENS.
23 Q OKAY. IS THERE ANY OTHER REASON WHY THESE
24 CANCER RISK ASSESSMENTS WERE BASED ON ANIMAL DATA RATHER
25 THAN HUMAN DATA?
26 A BECAUSE THERE WEREN'T ENOUGH HUMAN DATA --
27 HUMAN EXPOSURE DATA WITH THE CHEMICAL IN QUESTION TO GET
28 A DOSE RESPONSE, OFTEN.

1 Q OKAY. NOW, WHEN YOU DID YOUR QUANTITATIVE
2 CANCER RISK ASSESSMENT FOR ENVIRONMENTAL TOBACCO SMOKE,
3 DID YOU BASE THAT ON ANIMAL DATA OR HUMAN DATA?
4 A HUMAN DATA.
5 Q AND WHY, WHEN YOU DID THAT QUANTITATIVE
6 CANCER RISK ASSESSMENT, DID YOU DO IT USING HUMAN DATA?
7 A BECAUSE TOBACCO SMOKE IS A CARCINOGEN. AND
8 THE FIRST -- ONE OF THE FIRST CHAPTERS IN THE
9 ENVIRONMENTAL TOBACCO SMOKE RISK ASSESSMENT IS
10 DETERMINING THAT MAINSTREAM, OR THE INHALED TOBACCO
11 SMOKE FROM PUFFING ON THE CIGARETTE, IS A CARCINOGEN.
12 SO -- AND IT WAS WELL ESTABLISHED, BUT IT
13 HAD TO BE COVERED FOR ENVIRONMENTAL TOBACCO SMOKE.
14 AND THE REASON WE DID THAT IS BECAUSE
15 MAINSTREAM SMOKE IS A CARCINOGEN; AND ENVIRONMENTAL
16 TOBACCO SMOKE, WHICH IS ABOUT HALF MAINSTREAM SMOKE,
17 EXHALED MAINSTREAM SMOKE, IS VERY, VERY SIMILAR TO
18 MAINSTREAM SMOKE.
19 Q OKAY. WHEN YOU USED HUMAN DATA TO DO THE
20 QUANTITATIVE CANCER RISK ASSESSMENT FOR ENVIRONMENTAL
21 TOBACCO SMOKE, DID YOU -- HAD THERE BEEN EPIDEMIOLOGIC
22 STUDIES DONE SPECIFICALLY ADDRESSING THE RISK OF CANCER
23 FROM ENVIRONMENTAL TOBACCO SMOKE?
24 A YES. BOTH THE SURGEON GENERAL, IN 1986, AND
25 THE NATIONAL ACADEMY OF SCIENCES, IN 1986, EVALUATED THE
26 HUMAN DATA FOR ENVIRONMENTAL TOBACCO SMOKE IN THE SAME
27 LIGHT.
28 Q DID THAT EPIDEMIOLOGIC DATA PROVIDE ADEQUATE

1 DATA TO DERIVE A DOSE RESPONSE FOR THE ENVIRONMENTAL
2 TOBACCO SMOKE?
3 A YES, IT DID. IT DID FOR THE ENVIRONMENTAL
4 PROTECTION AGENCY, YES.
5 Q OKAY. AND BECAUSE OF THAT, WERE YOU ABLE TO
6 USE THAT EPIDEMIOLOGIC DATA?
7 A YES, WE DID.
8 Q ALL RIGHT. AND IF THERE WERE NO SUCH
9 DATA -- WELL, I'LL WITHDRAW THAT.
10 WHEN HUMAN EPIDEMIOLOGIC DATA DEMONSTRATE A
11 DOSE-RESPONSE RELATIONSHIP FOR A CARCINOGEN, WHY DO YOU
12 USE THAT DATA RATHER THAN THE ANIMAL DATA?
13 A BECAUSE, NUMBER ONE, WE'RE DEALING WITH THE
14 SAME SPECIES. WE WANT -- OUR OBJECTIVE IS TO ESTIMATE
15 THE RISK OF CANCER IN HUMANS, NOT IN ANIMALS. SO IF WE
16 HAVE ANIMALS -- I MEAN, IF WE HAVE HUMAN DATA, WE WANT
17 TO EXTRAPOLATE THE RISK TO HUMAN CONDITIONS.
18 Q OKAY. NOW, IF THE HUMAN EPIDEMIOLOGIC DATA
19 DOES NOT MEASURE OR ASSESS THE RISK OF CANCER FROM A
20 CARCINOGEN IN THE MIXTURE, DO YOU USE HUMAN DATA OR
21 ANIMAL DATA FOR THE CARCINOGEN?
22 MR. SCHURZ: INCOMPLETE HYPOTHETICAL, LACKS
23 FOUNDATION.
24 THE COURT: OVERRULED.
25 THE WITNESS: ARE YOU TALKING ABOUT HUMAN DATA
26 FOR -- FROM MIXTURES OR FOR CARCINOGENIC MIXTURES?
27 Q BY MR. METZGER: OKAY. WHAT I'M TALKING
28 ABOUT -- I SEE YOUR POINT. LET'S -- I'LL CLARIFY.

1 I'D LIKE YOU TO ASSUME THAT WE'RE TALKING
2 ABOUT A MIXTURE WHICH ITSELF HAS NOT BEEN DETERMINED TO
3 BE A CARCINOGEN.
4 A YES, SIR.
5 Q ALL RIGHT. AND THERE IS A CARCINOGEN IN THE
6 MIXTURE.
7 A YES, SIR.
8 Q ALL RIGHT. IF THE HUMAN EPIDEMIOLOGIC DATA
9 REGARDING THE MIXTURE HAS NOT ASSESSED THE RISK OF
10 CANCER FROM THE CARCINOGEN IN THE MIXTURE, DO YOU DO THE
11 RISK ASSESSMENT USING THE HUMAN DATA ON THE MIXTURE, OR
12 DO YOU DO THE RISK ASSESSMENT USING THE ANIMAL DATA ON
13 THE CARCINOGEN?
14 A WELL --
15 MR. SCHURZ: I'D INTERPOSE AN OBJECTION --
16 THE COURT: OBJECTION SUSTAINED. I THINK IT'S --
17 FIRST, THE QUESTION IS CONVOLUTED. AND SECOND OF ALL,
18 IT ASSUMES THERE ARE ONLY TWO CHOICES, AND THERE COULD
19 BE A MULTITUDE OF CHOICES.
20 Q BY MR. METZGER: WELL, LET ME ASK YOU THIS.
21 LET'S TURN TO THE CONTEXT OF ACRYLAMIDE IN COFFEE.
22 A OKAY.
23 Q AND I'D LIKE YOU TO ASSUME THAT COFFEE HAS
24 NOT BEEN LISTED AS A CARCINOGEN, BUT THAT ACRYLAMIDE HAS
25 BEEN LISTED AS A CARCINOGEN. AND I'D LIKE YOU TO ASSUME
26 THAT THE HUMAN EPIDEMIOLOGIC STUDIES REGARDING COFFEE
27 AND CANCER DID NOT ASSESS THE RISK OF CANCER FROM
28 ACRYLAMIDE IN COFFEE.

1 A UH-HUH.
2 MR. SCHURZ: WELL, I'LL INTERPOSE AN OBJECTION:
3 LACKS FOUNDATION. IT LACKS FOUNDATION AS TO THIS
4 WITNESS'S UNDERSTANDING.
5 THE COURT: IT'S A HYPOTHETICAL QUESTION.
6 MR. SCHURZ: INCOMPLETE HYPOTHETICAL, LACKS
7 FOUNDATION AS TO THE PREDICATE.
8 THE COURT: OVERRULED.
9 Q BY MR. METZGER: IN THAT CONTEXT, ASSUMING
10 THOSE ASSUMPTIONS, WOULD YOU DO THE RISK ASSESSMENT
11 BASED UPON THE MIXTURE, WHICH HAS NOT BEEN DETERMINED TO
12 BE A CARCINOGEN, OR BASED UPON THE CARCINOGEN IN THE
13 MIXTURE?
14 A IF I WANTED TO FIND OUT THE INCREASED RISK,
15 I WOULD DO THE INCREASED CANCER -- I'M SORRY -- I WOULD
16 DO THE CANCER RISK ASSESSMENT ON THE CARCINOGEN IN THE
17 MIXTURE.
18 Q OKAY. AND IN DOING THAT, WOULD YOU USE
19 ANIMAL DATA FOR THE CARCINOGEN OR HUMAN DATA FOR THE
20 MIXTURE?
21 A WELL, I WOULD USE THE ANIMAL DATA BECAUSE
22 THE HUMAN DATA HAVEN'T BEEN ESTABLISHED.
23 Q THANK YOU.
24 ALL RIGHT. NOW, DR. BAYARD, WHEN I
25 CONTACTED YOU REGARDING THIS CASE, WHAT DID I ASK YOU TO
26 DO?
27 A YOU ASKED ME TO DO A RISK ASSESSMENT ON THE
28 INCREASED CANCER RISK FOR ACRYLAMIDE IN COFFEE.

1 Q OKAY. AND DID YOU UNDERTAKE THAT TASK?
2 A I DID.
3 MR. METZGER: ALL RIGHT. CAN WE DISPLAY THE
4 POWERPOINT, SLIDE 12.
5 Q LET ME ASK YOU: ARE THERE A CERTAIN NUMBER
6 OF COMPONENTS IN DOING A QUANTITATIVE CANCER RISK
7 ASSESSMENT?
8 A YES.
9 Q AND WHAT ARE THEY?
10 MR. SCHURZ: I'LL INTERPOSE AN OBJECTION: IT'S
11 REDUNDANT, UNDER EVIDENCE CODE 352. THIS SAME SLIDE WAS
12 IN DR. MELNICK'S SET. WE'VE ALREADY SEEN THIS AND
13 TALKED ABOUT IT AT SOME LENGTH.
14 THE COURT: ALL RIGHT. IT'S FOUNDATIONAL.
15 YOU MAY PROCEED.
16 OBJECTION OVERRULED.
17 THE WITNESS: I WON'T TALK ABOUT IT LONG, BUT I
18 THINK I CAN MAKE MY POINT PRETTY QUICKLY.
19 Q BY MR. METZGER: GO AHEAD.
20 A THERE ARE FOUR POINTS OF RISK ASSESSMENT:
21 THE HAZARD IDENTIFICATION, WHICH IS THE QUALITATIVE
22 ASSESSMENT; IS IT CARCINOGENIC?
23 THE DOSE-RESPONSE ASSESSMENT, WHICH IS THE
24 POTENCY; HOW STRONG A CARCINOGEN IS IT?
25 THE EXPOSURE ASSESSMENT; HOW MUCH IS THE
26 HUMAN EXPOSED TO?
27 AND THE RISK CHARACTERIZATION, WHICH IS THE
28 QUANTITATIVE PART OF IT.

1 CAN I JUST DRAW ONE QUICK FORMULA ON THE
2 BOARD? AND I THINK I CAN MAKE THIS PRETTY QUICK.
3 Q YES. PLEASE GO AHEAD.
4 THE WITNESS: MAY I, YOUR HONOR?
5 THE COURT: ALL RIGHT.
6 THE WITNESS: THANK YOU.
7 TO ME, THIS IS PRETTY SIMPLE: IT'S RISK
8 EQUALS POTENCY TIMES DOSE.
9 "RISK" IS THE RISK CHARACTERIZATION THAT YOU
10 SEE HERE, OR THE QUANTITATIVE RISK ASSESSMENT.
11 "POTENCY" IS HOW STRONG A CHEMICAL IS WITH
12 RESPECT TO EACH INCREASING DOSE.
13 AND "DOSE" IS THE AMOUNT THAT PEOPLE GET.
14 OKAY? AND THAT'S ALL IT IS. IT'S JUST --
15 YOU LEARNED IN HIGH SCHOOL: IT WAS Y EQUALS BX; RIGHT?
16 AND THAT'S ALL I TRY TO DO.
17 THERE'S A LOT OF NUANCES AND TRICKY
18 CALCULATIONS, BUT THAT'S ALL IT DOES: RISK EQUALS
19 POTENCY TIMES DOSE.
20 SO HERE IS THE RISK CHARACTERIZATION, HERE'S
21 THE DOSE RESPONSE, AND HERE'S THE EXPOSURE RESPONSE.
22 IT'S THAT EASY.
23 Q BY MR. METZGER: OKAY. PLEASE RESUME YOUR
24 SEAT, AND LET ME JUST ASK YOU: SO POTENCY IS DOSE
25 RESPONSE?
26 A THAT'S CORRECT.
27 Q OKAY. VERY GOOD.
28 NOW, DR. BAYARD, IN DOING YOUR QUANTITATIVE

1 CANCER RISK ASSESSMENT FOR THE RISK OF CANCER FROM
2 EXPOSURE TO ACRYLAMIDE IN COFFEE FOR THIS CASE, DID YOU
3 UNDERTAKE A REVIEW OF CALIFORNIA REGULATIONS REGARDING
4 HOW CALIFORNIA REQUIRES SUCH RISK ASSESSMENTS TO BE
5 DONE?
6 A YES, I DID.
7 MR. METZGER: ALL RIGHT. COULD WE HAVE THE NEXT
8 SLIDE; THE NEXT ONE.
9 Q DID YOU REVIEW CALIFORNIA HEALTH AND SAFETY
10 CODE SECTION 25249.10(C)?
11 A YES, I DID.
12 Q BY MR. SCHURZ: YOUR HONOR, WE'LL OBJECT, AS
13 IT CALLS FOR LEGAL CONCLUSIONS. MOREOVER, UNDER 352,
14 THIS IS CUMULATIVE AND REDUNDANT. WHAT WE HAVE HERE IS
15 A SLIDE, ACTUALLY, THAT WAS PART OF DR. MELNICK'S SET;
16 IDENTICAL. YOU'LL FIND --
17 THE COURT: IT'S A SLIDE. THE ONLY QUESTION IS,
18 DID HE REVIEW IT? LET'S GO ON TO THE NEXT QUESTION AND
19 SEE WHERE IT'S GOING.
20 Q BY MR. METZGER: OKAY. AND DID YOU RELY ON
21 THIS REGULATION IN PERFORMING YOUR RISK ASSESSMENT?
22 A YES. THIS IS THE "NO SIGNIFICANT RISK"
23 CLAUSE. THERE'S GOT TO BE A PLACE IN WHICH RISK IS DE
24 MINIMIS, IT'S NOT WORTH LOOKING AT, BY SOME DEFINITION.
25 MR. METZGER: OKAY. CAN WE HAVE THE NEXT SLIDE.
26 Q DID YOU ALSO REVIEW 27 CCR SECTION 25701(A)?
27 A YES, I DID.
28 Q AND DID YOU RELY ON THIS REGULATION IN

1 PERFORMING YOUR QUANTITATIVE CANCER RISK ASSESSMENT?
2 A YES.
3 MR. METZGER: COULD WE HAVE THE NEXT SLIDE.
4 Q DID YOU ALSO REVIEW 27 CCR SECTION 25701(B)?
5 A YES.
6 Q AND DID YOU RELY ON THIS REGULATION IN
7 PERFORMING YOUR QUANTITATIVE RISK ASSESSMENT?
8 A YES.
9 MR. SCHURZ: I'M GOING TO INTERPOSE AN OBJECTION.
10 THIS AGAIN IS LEADING, AND IT CALLS FOR A LEGAL
11 CONCLUSION. AND ONCE AGAIN, WE'VE NOW SEEN THREE
12 SLIDES. ALL OF THEM WERE IN DR. MELNICK'S SET, EVERY
13 SINGLE ONE OF THEM.
14 THE COURT: HE'S JUST PUTTING SLIDES UP. THE
15 WITNESS TESTIFIED HE RELIED ON THEM. COUNSEL MAY
16 INQUIRE.
17 Q BY MR. METZGER: DID YOU REVIEW ALSO 27 CCR
18 SECTION 25703(A)?
19 A YES. THAT'S THE QUANTITATIVE RISK
20 ASSESSMENT SECTION, YES.
21 Q AND DID YOU RELY ON THIS REGULATION IN
22 PERFORMING YOUR QUANTITATIVE RISK ASSESSMENT?
23 A YES, I DID.
24 Q DID YOU ALSO RELY ON THE PART OF THAT
25 REGULATION THAT STATES THAT:
26 "THE ASSESSMENT SHALL BE BASED ON
27 EVIDENCE AND STANDARDS" --
28 THE COURT: YOU DON'T HAVE TO QUOTE THE

1 REGULATION. HE RELIED ON IT, YES. LET'S GO.
2 MR. METZGER: ALL RIGHT. THANK YOU.
3 WOULD YOU GO TO SLIDE 20, PLEASE.
4 Q DID YOU ALSO REVIEW 27 CCR SECTION
5 257039(A)(1)?
6 A YES, I DID.
7 Q ALL RIGHT. AND DID YOU DO YOUR QUANTITATIVE
8 CANCER RISK ASSESSMENT BASED UPON THIS REGULATION, IN
9 PART?
10 A YES. IT SAYS, "GET GOOD ANIMAL BIOASSAYS,
11 AND MAKE SURE THEY'RE WELL DONE."
12 Q OKAY. AND DID YOU ALSO RELY ON SUBSECTION 2
13 OF THAT REGULATION?
14 A I DIDN'T -- THIS TALKS ABOUT THE
15 EPIDEMIOLOGIC DATA, AND I DIDN'T DO THIS -- I DON'T
16 THINK I USED THIS FOR ACRYLAMIDE IN COFFEE.
17 Q AND WHY DID YOU NOT USE THE EPIDEMIOLOGIC
18 DATA FOR COFFEE FOR YOUR RISK ASSESSMENT OF ACRYLAMIDE
19 IN COFFEE?
20 A BECAUSE COFFEE ISN'T THE CARCINOGENIC AGENT.
21 THAT HAS NOT BEEN DECLARED A CARCINOGEN.
22 Q OKAY. DID YOU RELY ON SUBSECTION 3?
23 THAT'S:
24 "THE RISK ANALYSIS SHALL BE BASED ON THE
25 MOST SENSITIVE STUDY DEEMED TO BE OF
26 SUFFICIENT QUALITY."
27 MR. SCHURZ: I'LL OBJECT. HE'S JUST READING THE
28 REGULATIONS INTO THE RECORD. AGAIN, IT CALLS FOR A

1 LEGAL CONCLUSION. THIS IS REDUNDANT. ALL OF THIS IS --
2 THE COURT: THIS IS A REGULATION. IF YOU RELIED
3 ON THE REGULATIONS, THEN GIVE US YOUR OPINIONS AND TELL
4 US WHAT THEY'RE BASED ON.
5 THE WITNESS: THIS IS CITING THE MOST SENSITIVE
6 ANIMAL STUDY TO BE USED IN --
7 OH, I'M SORRY. MAY I -- DID I INTERRUPT
8 YOU?
9 THE COURT: NO. LET'S GO ON TO THE NEXT QUESTION.
10 Q BY MR. METZGER: ALL RIGHT. DID YOU RELY ON
11 THE REGULATIONS REGARDING ABSENCE OF A THRESHOLD?
12 A YES, I DID.
13 Q OKAY. DID YOU RELY ON THE REGULATIONS
14 REGARDING THE USE OF A LINEARIZED MULTISTAGE MODEL FOR
15 EXTRAPOLATION FROM HIGH TO LOW DOSES?
16 A YES, I DID.
17 Q ALL RIGHT. DR. BAYARD, I'D LIKE TO DISCUSS
18 WITH YOU THAT CONCEPT. I'M WONDERING, PERHAPS, IF THIS
19 MIGHT BE ANOTHER TIME FOR YOU TO DRAW, IF IT WOULD BE
20 HELPFUL IN ELUCIDATING IT.
21 BUT COULD YOU EXPLAIN TO THE COURT -- AND
22 USE THE BOARD, IF YOU NEED -- THE CONCEPT OF
23 EXTRAPOLATION FROM HIGH DOSES IN ANIMALS TO HUMAN
24 EXPOSURE LEVELS.
25 A YES. MAY I?
26 Q PLEASE.
27 A MAY I ERASE THIS?
28 THE CONCEPT OF LINEAR -- LINEARITY, NO

1 THRESHOLD. I'M GOING TO TRY TO ESTABLISH IT THIS WAY.
2 Q WELL, FIRST, JUST DRAW IT. AND THEN GO BACK
3 TO YOUR CHAIR WITH THE MICROPHONE, IF YOU WOULD.
4 A THANK YOU.
5 MR. SCHURZ: I'LL INTERPOSE AN OBJECTION. I DON'T
6 KNOW IF THERE'S A QUESTION PENDING. THERE WAS A
7 QUESTION PENDING, AND THEN THE WITNESS LAUNCHED INTO
8 SOMETHING ENTIRELY DIFFERENT THAN WHAT THE QUESTION WAS.
9 THE COURT: ALL RIGHT. WELL, WE'LL LET THE
10 WITNESS FINISH THE DRAWING. EVENTUALLY, WE'LL HAVE THE
11 WITNESS'S ANSWER, AND THEN WE'LL HAVE THE QUESTION.
12 A (MAKES DIAGRAM.)
13 Q ALL RIGHT. ARE YOU FINISHED DRAWING?
14 A YES.
15 Q PLEASE TAKE YOUR SEAT.
16 LET ME ASK YOU THIS: AND I SEE IN THE UPPER
17 RIGHT PORTION OF THE BOARD, YOU'VE WRITTEN "RATS" AND
18 "MICE," AND THERE ARE FOUR X'S THERE.
19 A YES, SIR.
20 Q WHAT ARE YOU REPRESENTING BY THAT?
21 A I'M REPRESENTING THE DOSES GIVEN TO THE --
22 AND GIVEN IN THE NATIONAL TOXICOLOGY PROGRAM STUDY,
23 WHICH I'M GOING TO SPEAK ABOUT, WHICH I USED FOR THE
24 QUANTITATIVE RISK ASSESSMENT.
25 THE TOP ONE WOULD BE THE MAXIMUM TOLERATED
26 DOSE, THEN SUBSEQUENTLY, LOWER; THE HIGH, MEDIUM, AND
27 LOW DOSES IN --
28 Q LET ME SEE IF I UNDERSTAND. SO THE FOUR X'S

1 ARE THE DOSE LEVELS FOR ACRYLAMIDE THAT WERE
2 ADMINISTERED TO MICE AND RATS, WITH AN INDICATION OF THE
3 CANCER RESPONSE?
4 A SCHEMATICALLY, YES.
5 Q OKAY. AND KIND OF IN THE MIDDLE OF THE
6 DRAWING, I SEE ANOTHER X WITH THE WORD "OCCUPATIONAL."
7 WHAT DOES THAT INDICATE?
8 A THAT'S A SCHEMATIC REPRESENTATION OF THE
9 LOWER EXPOSURE THAT ONE WOULD GET IN AN OCCUPATIONAL
10 ACRYLAMIDE PRODUCTION FACILITY, AS DR. INFANTE, I
11 BELIEVE, HAS TESTIFIED.
12 MR. SCHURZ: SO YOUR HONOR, WE'LL OBJECT AND MOVE
13 TO STRIKE --
14 THE COURT: SUSTAINED.
15 MR. SCHURZ: -- WITH RESPECT TO ANYTHING THAT THIS
16 WITNESS HAS TO SAY ABOUT OCCUPATIONAL STUDIES.
17 THE COURT: OBJECTION IS SUSTAINED TO THE LAST
18 STATEMENT.
19 NEXT QUESTION.
20 THE WITNESS: EXCUSE ME.
21 Q BY MR. METZGER: ALL RIGHT. AND IN THE
22 BOTTOM LEFT-HAND CORNER, I SEE A BOX THAT HAS THE WORD
23 "COFFEE" IN IT. WHAT IS IT REPRESENTING?
24 A THAT'S THE DOSE RANGE OF COFFEE -- EXPOSURE
25 TO ACRYLAMIDE IN COFFEE, FROM CONSUMPTION OF COFFEE.
26 Q I SEE. SO I SEE THAT YOU'VE DRAWN A LINE
27 THROUGH THE FOUR X'S AND GOING DOWN TO THE ORIGIN OF THE
28 GRAPH; IS THAT CORRECT?

1 A THAT'S CORRECT.
2 Q AND WHAT IS THAT REPRESENTING?
3 A THAT IS SUPPOSED TO REPRESENT THE "NO
4 THRESHOLD" RESPONSE THAT CALIFORNIA SPECIFIES IN THE
5 CALIFORNIA REGULATIONS THAT USE THE GENOTOXIC
6 CARCINOGENS.
7 Q OKAY.
8 A SO YOU JUST DRAW A STRAIGHT -- YOU BASICALLY
9 JUST DRAW A STRAIGHT LINE. IT'S A POTENCY SLOPE.
10 MR. SCHURZ: WE'LL OBJECT TO THE EXTENT THAT IT
11 CALLS FOR A LEGAL CONCLUSION, WHEN DR. BAYARD SAYS,
12 "THIS IS WHAT CALIFORNIA LAW CALLS FOR." HE IS NOT HERE
13 TO INTERPRET THE REGULATIONS.
14 THE COURT: ALL RIGHT. HE'S TESTIFYING AS TO HIS
15 UNDERSTANDING.
16 GO AHEAD.
17 Q BY MR. METZGER: ALL RIGHT. DR. BAYARD, I
18 DON'T SEE AN X IN THE BOX WHERE YOU'VE WRITTEN "COFFEE."
19 WHY IS THERE NO X THERE?
20 A WELL, THAT JUST SYMBOLIZES THE RANGE OF
21 EXPOSURE WITH THE COFFEE, BUT NO RISK ASSESSMENTS HAVE
22 BEEN DONE OF ACRYLAMIDE IN COFFEE BEFORE.
23 Q SO YOU DON'T KNOW -- OR I'M SORRY. SO THERE
24 IS -- LET ME TRY AGAIN.
25 ARE DOSES OF ACRYLAMIDE IN RELATION TO
26 CANCER -- HAS THAT BEEN ESTABLISHED IN ANY STUDIES OF
27 COFFEE?
28 A MAY I CORRECT MY ANSWER FOR THE LAST

1 QUESTION?
2 Q GO AHEAD.
3 A MY ANSWER IS THAT NO STUDIES OF -- FOR
4 INCREASED RISK OF ACRYLAMIDE IN COFFEE HAVE BEEN DONE
5 BEFORE. THAT'S WHAT I MEANT TO SAY. THAT'S WHY THERE'S
6 NO X THERE.
7 Q ALL RIGHT. AND WHAT IS THE CONCEPT THAT
8 YOU'RE TRYING TO CONVEY BY THIS GRAPHIC ILLUSTRATION?
9 A THAT WE HAVE ANIMALS AT HIGH DOSES, BECAUSE
10 THAT'S HOW YOU TEST FOR CARCINOGENICITY WHEN YOU DON'T
11 HAVE HUMAN STUDIES. AND WE HAVE THE ACRYLAMIDE -- SO
12 THOSE ARE ACRYLAMIDE STUDIES IN ANIMALS AT HIGH DOSES.
13 AND WE HAVE TO TEST WHETHER OR NOT -- WE
14 HAVE TO ANALYZE AND CALCULATE WHETHER OR NOT THE
15 ACRYLAMIDE IN COFFEE CAN CAUSE AN INCREASED RISK;
16 IN THIS CASE, WHETHER OR NOT THAT INCREASED
17 RISK IS GREATER THAN THAT PRESCRIBED BY CALIFORNIA IN
18 ITS "NO SIGNIFICANT RISK" LEVEL FOR ITS LISTED
19 CARCINOGEN, ACRYLAMIDE.
20 Q OKAY. AND WHY DO YOU DRAW A LINE FROM THE
21 DOSES IN THE ANIMALS TO THE ORIGIN?
22 A THAT'S THE CONCEPT OF THE "NO THRESHOLD,"
23 "ASSUME NO THRESHOLD FOR GENOTOXIC CARCINOGENS," WHICH
24 WERE IN THE CALIFORNIA CODE OF REGULATIONS.
25 THE COURT: LET ME ASK YOU: THIS CURVE THAT YOU
26 DREW, THIS LINE, THE LINEAR DEPICTION -- IS THIS JUST
27 BASED UPON ASSUMPTION? OR IS IT BASED ON ACTUAL
28 STUDIES, THAT YOU EXTRAPOLATE THAT THE LINE IS GOING

1 THROUGH ON THAT ANGLE RATHER THAN VEERING OFF TO THE
2 RIGHT OR LEFT AT LOWER DOSES?
3 THE WITNESS: ARE YOU TALKING ABOUT A STRAIGHT
4 LINE AS OPPOSED TO --
5 THE COURT: YES; RIGHT. YES, I AM.
6 THE WITNESS: THAT'S BEEN ESTABLISHED OVER A LONG
7 PERIOD BY ALL THE REGULATORY AGENCIES: THAT IF THE
8 CARCINOGEN IS GENOTOXIC AND AFFECTS THE DNA, THEN UNLESS
9 ONE KNOWS WITH A HIGH DEGREE OF CERTAINTY, ONE ASSUMES
10 FOR QUANTITATIVE RISK ASSESSMENT PURPOSES THAT THAT LINE
11 IS STRAIGHT.
12 THE COURT: SO IT'S AN ASSUMPTION; IT'S NOT
13 ESTABLISHED SCIENTIFIC FACT?
14 THE WITNESS: THERE'S LOTS OF ESTABLISHMENT BASED
15 UPON MUTAGENICITY STUDIES AND HOW CARCINOGENS ARE MOSTLY
16 MUTAGENS.
17 THE COURT: BUT IT'S A GENERALIZED STATEMENT, NOT
18 RELATED TO ANY SPECIFIC CARCINOGEN?
19 THE WITNESS: IT'S A GENERALIZED STATEMENT. THIS
20 IS HOW PEOPLE DO RISK ASSESSMENTS, AND IT'S CODIFIED.
21 THE COURT: I TAKE IT, THEORETICALLY, THE LINE AT
22 THE LOWER END COULD CURVE ALL THE WAY TO THE RIGHT OR
23 CURVE TO THE LEFT OR --
24 THE WITNESS: THAT'S CORRECT. THAT'S ABSOLUTELY
25 CORRECT; IT COULD.
26 THE COURT: ALL RIGHT. THANK YOU.
27 Q BY MR. METZGER: WELL, LET ME ASK YOU: HAVE
28 RISK ASSESSMENTS BEEN DONE OF GENOTOXIC CARCINOGENS

1 WHICH ESTABLISHED LINEAR DOSE RESPONSE?
2 A THERE WAS ONE IN ANIMALS A LONG TIME AGO --
3 IT WAS IN THE LATE '70S; IT WAS CALLED THE MEGAMALL
4 STUDIES -- WHICH DID ESTABLISH LINEARITIES DOWN TO VERY,
5 VERY LOW DOSES, YES.
6 Q OKAY.
7 A AND I THINK THAT WAS WITH -- AAF WAS THE
8 NAME, SOMETHING -- AFLA-SOMETHING TOXIN. AFLATOXIN;
9 AFLATOXIN.
10 Q ALL RIGHT. IN ANY EVENT, HAS THE CONCEPT OF
11 A LINEAR MODEL WITHOUT A THRESHOLD BEEN GENERALLY
12 ACCEPTED IN THE RISK ASSESSMENT COMMUNITY FOR ASSESSING
13 THE CANCER RISK TO HUMANS OF GENOTOXIC CARCINOGENS?
14 MR. SCHURZ: OBJECTION; OVERBROAD, LACKS
15 FOUNDATION.
16 THE COURT: OVERRULED.
17 THE WITNESS: YES, IT HAS.
18 Q BY MR. METZGER: AND HOW DO YOU KNOW THAT
19 THAT'S BEEN GENERALLY ACCEPTED?
20 A I'VE WORKED IN THIS AREA SINCE 1974.
21 Q WELL, IS THAT HOW IT WAS DONE AT THE EPA
22 WHEN YOU WERE AT THE EPA?
23 A YES.
24 Q IS THAT HOW IT WAS DONE AT THE CONSUMER
25 PRODUCTS SAFETY COMMISSION WHEN YOU WERE THERE?
26 A IT WAS ACTUALLY BEFORE IT WAS WELL
27 ESTABLISHED BECAUSE I HELPED ESTABLISH THESE RULES
28 MYSELF.

1 Q SO YOU HELPED ESTABLISH THIS CONCEPT AT
2 CONSUMER PRODUCT SAFETY COMMISSION?
3 A I -- YOU KNOW, IT WAS SO LONG AGO, I'M NOT
4 EVEN SURE WE WORKED ON THAT CONCEPT.
5 Q ALL RIGHT. AND HAS THIS BEEN ADOPTED IN
6 CANCER RISK ASSESSMENT GUIDELINES?
7 A YES, IT HAS.
8 Q AND HAS THIS BEEN SPECIFICALLY ADOPTED IN
9 CALIFORNIA'S PROPOSITION 65 REGULATIONS?
10 A YES.
11 MR. SCHURZ: OBJECTION; CALLS FOR A LEGAL
12 CONCLUSION.
13 THE COURT: ON THE LAST QUESTION, THE IS OBJECTION
14 SUSTAINED. THE ANSWER WILL BE STRICKEN.
15 GO AHEAD. NEXT QUESTION.
16 Q BY MR. METZGER: WELL, LET ME ASK YOU: HAVE
17 YOU FOUND SUPPORT FOR THIS LINEAR NO-THRESHOLD MODEL IN
18 THE CALIFORNIA REGULATIONS THAT YOU REVIEWED?
19 MR. SCHURZ: SAME OBJECTION; CALLS FOR --
20 THE COURT: SUSTAINED.
21 Q BY MR. METZGER: ALL RIGHT. AND DR. BAYARD,
22 WHAT IS THE SCIENTIFIC JUSTIFICATION FOR EXTRAPOLATING
23 FROM THE DOSES IN RODENTS TO HUMAN EXPOSURE LEVELS IN A
24 LINEAR NO-THRESHOLD FASHION?
25 A WELL, I THINK, IN A LINEAR NO-THRESHOLD
26 FASHION -- I THINK THE ANSWER IS PRETTY MUCH WHAT I'VE
27 BEEN GIVING BEFORE -- THAT I'VE GIVEN BEFORE.
28 AND THAT IS THAT IF -- NUMBER ONE, IF IT

1 CAUSES CANCERS IN CANCERS IN THE ANIMALS AND IT'S
2 GENOTOXIC, THEN IT'S BEEN PRETTY WELL ESTABLISHED
3 THROUGH LOTS OF DIFFERENT TYPES OF EXPERIMENTS THAT
4 THERE IS LOW-DOSE LINEARITY.
5 HOW LOW WE DON'T KNOW, BECAUSE IT CAN ALWAYS
6 BE LOWER THAN WE CAN GO.
7 Q OKAY. ALL RIGHT. THANK YOU.
8 IN PERFORMING YOUR QUANTITATIVE CANCER RISK
9 ASSESSMENT FOR ACRYLAMIDE IN COFFEE, DID YOU FOLLOW ALL
10 OF THE PROVISIONS SET FORTH IN 27 CCR SECTION 25703(A)?
11 A I THINK SO.
12 Q ALL RIGHT. AND WHEN YOU DID YOUR
13 QUANTITATIVE RISK ASSESSMENT, CANCER RISK ASSESSMENT,
14 FOR ACRYLAMIDE IN COFFEE, WHAT WAS THE CHEMICAL IN
15 QUESTION, OR THE AGENT -- WHAT WAS THE AGENT THAT YOU
16 ASSESSED THE CANCER RISK FOR?
17 A ACRYLAMIDE.
18 MR. METZGER: AND CAN YOU TURN TO SLIDE 29,
19 PLEASE.
20 Q IS THE SELECTION OF ACRYLAMIDE AS THE
21 CHEMICAL FOR YOUR QUANTITATIVE CANCER RISK ASSESSMENT
22 SUPPORTED BY THE 27 CCR SECTION 25721(A)?
23 MR. SCHURZ: OBJECTION; LEADING, CALLS FOR A LEGAL
24 CONCLUSION.
25 THE COURT: SUSTAINED.
26 Q BY MR. METZGER: ALL RIGHT. LET ME TRY
27 THIS: IS THERE ANYTHING IN THE REGULATION THAT
28 SUPPORTS -- THAT YOU REVIEWED, THAT SUPPORTS YOUR

1 CONCLUSION THAT THE CORRECT AGENT TO BE ASSESSED FOR
2 CANCER RISK IS ACRYLAMIDE RATHER THAN THE COFFEE?
3 MR. SCHURZ: OBJECTION; CALLS FOR --
4 THE COURT: SUSTAINED.
5 MR. METZGER: OKAY. ONE MORE REGULATION, AND
6 WE'LL BE DONE WITH THIS.
7 GO TO SLIDE 33, PLEASE.
8 Q DID YOU RELY ON 27 CCR SECTION 25721(D)(4)
9 IN DOING YOUR EXPOSURE ASSESSMENT AS PART OF YOUR CANCER
10 RISK ASSESSMENT?
11 A YES. THIS REFERS TO THE INTAKE FOR AVERAGE
12 DRINKERS. SO WHEN I DID MY EXPOSURE ASSESSMENT, I
13 LOOKED AT DRINKERS; NOT THE GENERAL POPULATION, BUT THE
14 PEOPLE WHO ACTUALLY DRANK COFFEE.
15 Q ALL RIGHT. THANK YOU.
16 I'D LIKE TO NOW START WITH THE HAZARD
17 ASSESSMENT THAT YOU DID -- OR WELL, LET ME ASK YOU THAT.
18 FIRST OF ALL, IN DOING YOUR QUANTITATIVE
19 CANCER RISK ASSESSMENT, DID YOU DO A HAZARD ASSESSMENT?
20 A NO. I DIDN'T HAVE TO.
21 Q WHY DID YOU NOT HAVE TO DO A HAZARD
22 ASSESSMENT?
23 A BECAUSE ACRYLAMIDE IS LISTED BY THE STATE OF
24 CALIFORNIA AS A CARCINOGEN.
25 Q OKAY.
26 A HOWEVER, OTHER PEOPLE HAVE ALSO DONE --
27 OTHER PEOPLE HAVE DONE HAZARD ASSESSMENTS.
28 Q OKAY. AND DID YOU RELY ON THE HAZARD

1 ASSESSMENTS THAT WERE DONE BY OTHER AGENCIES FOR THE
2 CARCINOGENICITY OF ACRYLAMIDE?
3 A ONLY TO THE EXTENT THAT EACH ONE OF THEM
4 DECLARED THAT IT WAS A GENOTOXIC CARCINOGEN AS ITS MODE
5 OF ACTION, WHICH IS TERM OF ART USED BY GOVERNMENT
6 AGENCIES. ITS MODE OF ACTION IS THAT OF GENOTOXICITY,
7 AND IT CAUSES CANCER THROUGH GENOTOXICITY.
8 Q OKAY. AND WHAT GOVERNMENT AGENCIES HAD
9 ALREADY ASSESSED THE HAZARD OF ACRYLAMIDE AS A
10 CARCINOGEN?
11 A WELL, CALIFORNIA, THE U.S. EPA, U.S. FOOD
12 AND DRUG ADMINISTRATION, WORLD HEALTH ORGANIZATION,
13 HEALTH CANADA, NORTH CAROLINA. I GUESS THERE'S A LOT
14 MORE. BUT EVERYONE WHO HAS DONE IT HAS DECLARED IT A
15 CARCINOGEN.
16 Q RIGHT. SO YOU FELT YOU DIDN'T HAVE TO DO
17 THAT ASSESSMENT YOURSELF?
18 A NOT ONLY THAT, BUT IT'S NOT REALLY WHERE MY
19 EXPERTISE IS.
20 Q ALL RIGHT. NOW, THE SECOND COMPONENT OF THE
21 RISK ASSESSMENT IS DOSE RESPONSE?
22 A THAT'S CORRECT. THAT'S POTENCY.
23 Q OKAY. SO WHEN YOU SAY "POTENCY" AND "DOSE
24 RESPONSE," ARE WE TALKING THE SAME THING?
25 A THAT'S WHAT I MEAN. IT'S THE SAME THING,
26 YES.
27 Q ALL RIGHT. AND DID THAT COMPONENT OF THE
28 RISK ASSESSMENT TAKE A BIT OF WORK TO DO?

1 A YES -- NO, IT WAS MORE THAN A BIT OF WORK.
2 Q ALL RIGHT. LET ME ASK YOU, FIRST: WHAT
3 DATA DID YOU USE FOR DOING THE DOSE RESPONSE OR POTENCY
4 ASSESSMENT?
5 A I USED THE ANIMAL DATA FROM THE NATIONAL
6 TOXICOLOGY TWO-YEAR CANCER BIOASSAYS OF ACRYLAMIDE IN
7 RATS AND -- IN DRINKING WATER IN RATS AND MICE.
8 Q AND WHY DID YOU USE THE NATIONAL TOXICOLOGY
9 PROGRAMS CANCER BIOASSAYS FOR ACRYLAMIDE AS THE BASIS
10 FOR YOUR DOSE-RESPONSE ASSESSMENT?
11 A WELL, THERE'S LOTS OF REASONS; BUT
12 BASICALLY, THE NATIONAL TOXICOLOGY PROGRAM IS CONSIDERED
13 THE GOLD STANDARD OF ALL ANIMAL BIOASSAYS.
14 THEY'VE BEEN -- THEY'VE BEEN DOING ANIMAL
15 BIOASSAYS, LIFETIME ANIMAL BIOASSAYS, FOR
16 CARCINOGENICITY SINCE PROBABLY 1981, WHEN THEY TOOK OVER
17 THE PROGRAM FROM THE NATIONAL CANCER INSTITUTE. THAT
18 WOULD BE MY BEST GUESS. IT'S BEEN MANY, MANY YEARS.
19 YOU'LL SEE THE REPORT THAT -- ON ACRYLAMIDE,
20 IT WAS NO. 575 IN THEIR REPORT. SO THEY'VE BEEN DOING
21 IT FOR A LONG TIME.
22 Q OKAY. AND DID YOU APPRAISE THE DATA IN THE
23 NTP ANIMAL BIOASSAY STUDY FOR ITS QUALITY?
24 A I DIDN'T, BECAUSE I TRUSTED THE NATIONAL
25 TOXICOLOGY PROGRAM. SO I -- AND I DON'T KNOW IF I HAVE
26 THE REAL EXPERTISE TO SAY HOW GOOD THE STUDY WAS. BUT I
27 RELIED ON THE JUDGMENTS OF THE TOXICOLOGISTS WHO MET TO
28 PRODUCE THE REPORT.

1 Q ALL RIGHT. AND WHAT WAS THE DATA IN THE
2 NATIONAL TOXICOLOGY PROGRAM BIOASSAYS THAT YOU ACTUALLY
3 USED?
4 A I USED INDIVIDUAL TUMOR SITES IN THE RATS
5 AND MICE OF BOTH SEXES.
6 Q ALL RIGHT. AND IN SELECTING THE DATA TO USE
7 IN YOUR ASSESSMENT, DID YOU HAVE ANY CONDITIONS ON WHICH
8 DATA WITHIN THE NTP STUDY YOU WOULD USE?
9 A YES.
10 Q WHAT WERE THOSE?
11 A I THINK THERE WERE ABOUT THREE CONDITIONS
12 THAT I CONSIDERED ABOVE ALL OTHERS. AND ONE WAS, DID
13 THE NATIONAL TOXICOLOGY PROGRAM DECLARE THESE TUMORS TO
14 BE CLEAR EVIDENCE OR RELATED EVIDENCE OF CARCINOGENICITY
15 IN THE ANIMALS?
16 TWO, WERE THE INCREASES IN THE DOSE
17 GROUPS -- IN ANY OF THE DOSE GROUPS STATISTICALLY
18 SIGNIFICANT?
19 AND THREE, WAS THERE A SIGNIFICANT DOSE
20 RESPONSE TREND FOR THE TUMOR SITES?
21 Q AND -- OKAY. DID YOU CONSIDER ANY
22 CONCLUSIONS THAT THE NTP HAD REACHED IN ITS REPORT ON
23 THE CARCINOGENICITY OF ACRYLAMIDE IN THESE BIOASSAYS?
24 A YES, I DID.
25 Q AND WHAT WERE THE CONCLUSIONS THAT YOU
26 RELIED ON?
27 A WELL, I DID IT FOR EACH TUMOR SITE,
28 ACTUALLY. THE NTP MADE CONCLUSIONS OF CLEAR

1 CARCINOGENICITY IN ANIMALS, BUT THEY -- AND THEN THEY
2 SUBDIVIDED THOSE CONCLUSIONS BY NUMBERS OF TUMOR SITES
3 THAT THEY FOUND CLEAR EVIDENCE OF CARCINOGENIC ACTIVITY
4 IN.
5 Q OKAY. SO TELL US WHAT YOU DID AT -- THE
6 FIRST STEP OF WHAT YOU DID WITH THIS DATA FROM THE NTP
7 STUDY.
8 A I CALCULATED A POTENCY FOR EACH INDIVIDUAL
9 TUMOR SITE.
10 Q AND WHAT DOES THAT MEAN, EXACTLY?
11 A A DOSE RESPONSE BASED ON EXTRAPOLATING
12 FROM -- THE SLOPE OF THE CURVE FITS WITH THE
13 DOSE-RESPONSE POINTS.
14 Q FOR THE RAT -- FOR THE ANIMAL DATA?
15 A EACH MALE AND FEMALE SEPARATELY, EACH RAT
16 AND MOUSE -- RAT STUDY AND MOUSE STUDY.
17 Q OKAY.
18 A SO THERE WERE ABOUT 17 TO 19 OF THEM THAT I
19 TRIED DOING ACTUAL FITTING OF THE POTENCY SLOPES.
20 Q OKAY. WHEN YOU SAY THERE WERE 17 TO 19, 17
21 TO 19 OF WHAT?
22 A TUMOR SITES THAT WERE STATISTICALLY
23 SIGNIFICANT AND WHICH THE NTP HAD DECLARED CLEAR OR
24 RELATED EVIDENCE OF CARCINOGENICITY. I THINK THEY
25 RELATED "CLEAR EVIDENCE" ON 15 OF THEM AND "RELATED TO
26 CARCINOGENICITY," WHICH WAS A LOWER DEGREE OF CERTAINTY,
27 ON 4 OTHERS.
28 Q ALL RIGHT. SO ONCE YOU IDENTIFIED THOSE

1 SITES, WHAT DID YOU DO?
2 A I DID A -- I CALCULATED MODELS TO CALCULATE
3 A POTENCY, A DOSE RESPONSE.
4 Q OKAY. DID YOU PREPARE TABLES IN YOUR REPORT
5 TO SHOW THAT?
6 A YES, I DID.
7 THE COURT: WE'RE GOING TO TAKE A RECESS AT THIS
8 TIME. WE'LL BE BACK TO CONTINUE THE TRIAL IN ABOUT TEN
9 MINUTES.
10 (RECESS.)
11 THE COURT: GOOD MORNING AGAIN, COUNSEL. BACK ON
12 THE RECORD IN THE CASE OF CERT VS. STARBUCKS. ALL
13 COUNSEL ARE PRESENT, AND DR. BAYARD IS ON THE STAND.
14 DR. BAYARD, YOU UNDERSTAND YOU'RE STILL
15 UNDER OATH?
16 THE WITNESS: EXCUSE ME, SIR?
17 THE COURT: DO YOU UNDERSTAND YOU'RE STILL UNDER
18 OATH?
19 THE WITNESS: THANK YOU.
20 THE COURT: PLEASE RESTATE YOUR NAME ON THE
21 RECORD.
22 THE WITNESS: THANK YOU, YOUR HONOR.
23 THE COURT: PLEASE RESTATE YOUR NAME.
24 THE WITNESS: STEVEN BAYARD.
25 THE COURT: ALL RIGHT. THANK YOU. MR. METZGER IS
26 INQUIRING.
27 COUNSEL.
28 MR. METZGER: THANK YOU, YOUR HONOR.

1 THE COURT: COUNSEL, PLEASE PROCEED.
2 Q BY MR. METZGER: DR. BAYARD, REGARDING YOUR
3 ANALYSIS OF DOSE RESPONSE FOR POTENCY, WHAT DATA OR
4 RESULTS FROM THE NTP ACRYLAMIDE BIOASSAY WERE IMPORTANT
5 TO YOU FOR THAT ANALYSIS?
6 A WELL, THE DATA WERE THE 17 TUMOR SITES THAT
7 THE NTP SAID -- 19, ACTUALLY -- SHOWED CLEAR OR RELATED
8 CARCINOGENIC ACTIVITY IN BOTH -- IN THE MALES, FEMALES,
9 RATS, AND MICE.
10 I THINK THAT WAS YOUR QUESTION.
11 Q OKAY. ALL RIGHT. NOW, SO THERE WERE DATA
12 SETS WITHIN THE NTP STUDY FOR RATS AND MICE OF BOTH
13 SEXES, SO ESSENTIALLY FOUR GROUPS OF DATA; IS THAT
14 CORRECT?
15 A THAT'S CORRECT.
16 Q ALL RIGHT. AND WHAT DID YOU DO WITH THOSE
17 DATA SETS?
18 A I MODELED THEM ALL, EACH DATASET SEPARATELY,
19 FOR DOSE RESPONSE.
20 Q AND HOW DID YOU GO ABOUT MODELING THEM?
21 A I USED THE EPA BMDS SOFTWARE. BUT MAY I --
22 Q WELL, LET ME ASK YOU: WHY DID YOU MODEL
23 THEM?
24 A WHAT I HAVE TO DO -- THE REASON I MODELED
25 THEM WAS TO DETERMINE WHICH IS THE MOST SENSITIVE STUDY
26 TO USE FOR RISK ASSESSMENT. AND IN ORDER TO FIGURE OUT
27 THE MOST SENSITIVE STUDY FOR RISK ASSESSMENT, YOU HAVE
28 TO MODEL EACH DATASET.

1 Q OKAY.
2 A SO I WANT TO FIND OUT WHETHER IT'S RATS OR
3 MICE AND WHETHER IT'S MALES OR FEMALES. BUT THEY EACH
4 HAVE DIFFERENT SITES.
5 SO NOT ONLY THAT, IT'S NOT JUST ONE SLOPE
6 THAT YOU'RE GOING TO GET, ONE TUMOR SLOPE -- YOU GET ONE
7 SLOPE FOR EACH TUMOR SITE; BUT THE ANIMALS HAD MULTIPLE
8 TUMOR SITES.
9 SO IN ORDER TO FIND OUT WHICH IS THE MOST
10 SENSITIVE SEX/SPECIES, YOU HAVE TO FIGURE OUT A WAY TO
11 COMBINE TUMOR SITES TO GET AN OVERALL POTENCY ESTIMATE
12 FOR EACH ANIMAL.
13 Q AND WHY ARE YOU DOING ALL OF THIS WORK TO
14 ASCERTAIN THE MOST SENSITIVE SITE?
15 A BECAUSE THE CALIFORNIA CODE OF REGULATIONS
16 AND THE EPA -- ACTUALLY, THE U.S. EPA, SAY, "USE THE
17 MOST SENSITIVE ANIMAL SEX/SPECIES STUDY TO" -- THEY
18 DON'T SAY "SEX/SPECIES"; BUT IT SAYS, "USE THE MOST
19 SENSITIVE ANIMAL STUDY FOR EXTRAPOLATION TO HUMANS."
20 MR. SCHURZ: YOUR HONOR, WE'LL OBJECT TO THE
21 EXTENT THAT IT CALLS FOR A LEGAL CONCLUSION AND DR.
22 BAYARD'S OPINIONS WITH RESPECT TO WHAT CALIFORNIA
23 REGULATIONS CALL FOR.
24 THE COURT: OVERRULED.
25 Q BY MR. METZGER: OKAY. SO HOW DID YOU GO
26 ABOUT DOING THE MODELING?
27 A WELL --
28 Q WELL, LET ME ASK YOU: FIRST, HOW MANY TUMOR

1 SITES DID YOU MODEL?
2 A I TRIED 19.
3 Q OKAY. AND WHAT HAPPENED WITH THAT EFFORT?
4 A I WAS SUCCESSFUL IN 17 TO GET DOSE RESPONSE
5 CURVES. SOME OF THEM I COULDN'T GET THE MODELS TO
6 CONVERGE.
7 NOW, WE'RE JUST FITTING -- WE'RE FITTING
8 MODELS TO DATA. SOME OF THESE DATA ARE NOT MONOTONIC.
9 THEY DON'T GO UP PARTICULARLY THE SAME. THERE IS
10 VARIATIONS IN RESPONSES. AND SO SOME OF THE MODELS
11 JUST -- THE COMPUTER PROGRAMS COULD NOT CONVERGE ON A
12 SOLUTION TO DETERMINE A CORRECT MODEL.
13 Q ALL RIGHT. WELL, LET ME ASK YOU, FIRST:
14 WHAT COMPUTER PROGRAM ARE YOU TALKING ABOUT?
15 A I USE THE PUBLICLY AVAILABLE U.S. EPA'S BMDS
16 SOFTWARE. "BMDS" STANDS FOR "BENCHMARK DOSE SOFTWARE."
17 Q ALL RIGHT. AND HAS THAT SOFTWARE BEEN USED
18 BY RISK ASSESSORS, SUCH AS YOU, OVER THE YEARS?
19 A YES.
20 Q AND IS IT GENERALLY ACCEPTED TO USE THAT EPA
21 SOFTWARE IN DOING THIS EXERCISE AMONG RISK ASSESSORS IN
22 THE RISK ASSESSMENT COMMUNITY?
23 MR. SCHURZ: OBJECTION; LACKS FOUNDATION AS TO
24 GENERAL ACCEPTANCE. AND VAGUE AND AMBIGUOUS AS TO "THIS
25 EXERCISE."
26 THE COURT: OVERRULED.
27 THE WITNESS: YES. IN FACT, THE NTP, IN ITS
28 PUBLICATIONS ON THEIR STUDY, USED BMDS SOFTWARE. THAT'S

1 VIELAND 2013. VIELAND WAS THE PRINCIPAL INVESTIGATOR.
2 Q BY MR. METZGER: OKAY.
3 A OTHERS HAVE USED IT TOO. HEALTH CANADA USED
4 IT. THE EUROPEAN FOOD SAFETY AGENCY USED IT. EPA USES
5 IT.
6 Q ALL RIGHT. NOW, WHEN YOU USED IT, YOU WERE
7 SUCCESSFUL IN MODELING DOSE RESPONSE FOR 17 OF THE 19
8 SITES; IS THAT CORRECT?
9 A THAT'S CORRECT.
10 Q ALL RIGHT. AND WHAT DOES IT MEAN THAT YOU
11 WERE UNSUCCESSFUL IN DOING THAT IN -- FOR 2 OF THOSE 19
12 SITES? IS THAT A FAILURE ON YOUR PART? WHAT DOES THAT
13 MEAN?
14 A IT'S USUALLY THE DOSES -- THAT THE RESPONSE
15 IS -- IN THE ANIMALS IS NOT UNIFORMLY INCREASING OR
16 MONOTONICALLY INCREASING. IT'S USUALLY -- YOU'LL SEE
17 DOSES THAT MAY HAVE MORE -- HAVE A LOWER DOSE, AND THEN
18 YOU INCREASE THE DOSE, AND THEN IT -- THE RESPONSE CAN
19 ACTUALLY DIP, ON THESE ANIMAL STUDIES.
20 Q OKAY. SO IS IT A GENERALLY ACCEPTED
21 APPROACH TO EXCLUDE FROM THE ANALYSIS SITES WHERE --
22 ANIMAL TUMORS WHERE MODELING IS UNSUCCESSFUL BASED UPON
23 THE PROGRAM?
24 A I DON'T KNOW.
25 Q OKAY.
26 A I MEAN, I DID THE BEST I COULD, AND I
27 COULDN'T GET THESE TWO TO CONVERGE. THEY WEREN'T VERY
28 HIGH TUMOR RESPONSES, ANYWAY, AND SO I LEFT THEM OUT.

1 Q ALL RIGHT. SO THAT WAS A JUDGMENT CALL ON
2 YOUR PART TO DO THAT?
3 A IT DOESN'T INCREASE THE RISK. IT ACTUALLY
4 WOULD DECREASE THE RISK, IF THERE IS A RISK, FROM THESE
5 TUMORS.
6 Q ALL RIGHT. SO WHAT WAS THE RESULT OF THE
7 MODELING THAT YOU DID FOR THE 17 SITES?
8 A THE RESULTS WERE THAT I GOT 4 POSITIVE DOSE
9 RESPONSES FOR MALES -- MALE RATS, 4 FOR FEMALE RATS, 3
10 FOR MALE MICE, AND 6 FOR FEMALE MICE. SO I GOT 17
11 POSITIVE VOTES.
12 Q OKAY.
13 A BUT THAT DIDN'T HELP ME GET THE -- IT DIDN'T
14 HELP ME FIND THE MOST SENSITIVE SPECIES BECAUSE THEN I
15 HAD TO FIGURE OUT THE TOTAL POTENCY FOR EACH SEX AND
16 SPECIES.
17 Q OKAY. AND WAS THAT THE NEXT STEP IN YOUR
18 ANALYSIS?
19 A THAT'S WHAT I HAD TO DO.
20 Q ALL RIGHT. AND HOW DID YOU GO ABOUT DOING
21 THAT?
22 A I USED A METHOD THAT WAS USED BY THE U.S.
23 EPA IN THEIR 2010 RISK ASSESSMENT OF ACRYLAMIDE. I USED
24 THE SAME METHOD THEY DID.
25 Q AND DOES THAT HAVE A NAME?
26 A COMBINED TUMORS? I MEAN, IT'S A METHOD --
27 IT'S SIMILAR TO A METHOD THAT I HAD USED YEARS AGO.
28 Q AND CAN YOU DESCRIBE WHAT THAT METHOD IS,

1 GENERALLY.
2 A HERE GOES.
3 YOU ADD UP THE BEST ESTIMATE OF SLOPE. FOR
4 EACH SEX AND SPECIES WITHIN -- LET'S SAY, FOR THE MALE
5 RAT. YOU'VE GOT FOUR DIFFERENT SLOPES. SO YOU ADD UP
6 ESSENTIAL ESTIMATES, THE BEST POTENCY ESTIMATES.
7 AND YOU HAVE TO TAKE ALLOWANCE FOR THE FACT
8 THAT THESE ESTIMATES HAVE ERRORS IN THEM, THAT -- THEIR
9 CONFIDENCE. AND YOU'LL GET FITS OF MODELS. AND THE
10 STATISTICAL DEFINITION OF HOW WELL THE MODELS FIT ARE
11 BASED ON SOME STANDARD DEVIATION OR STANDARD ERRORS.
12 SO YOU'RE BASICALLY -- IN ORDER TO DO THIS
13 ESTIMATE FOR COMBINED POTENCY FOR MULTIPLE TUMORS, YOU
14 ADD UP THE SLOPES, TAKE A COUNT OF THE STANDARD ERRORS
15 OF THE SUM SLOPE, AND CALCULATE THESE FACTORS THAT WAY
16 FOR COMBINED POTENCY.
17 Q ALL RIGHT. WHAT IS A BENCHMARK DOSE?
18 A IT'S PROBABLY EASIER -- OKAY. A BENCHMARK
19 DOSE IS -- FOR ACRYLAMIDE, THE WAY EPA DEFINES IT OR CAL
20 EPA USES IT, IS A DOSE WHICH WILL GIVE A 10 PERCENT
21 RESPONSE.
22 Q OKAY. AND DID YOU -- I THINK THE SOFTWARE
23 THAT YOU USED, WAS THAT CALLED A BENCHMARK DOSE
24 SOFTWARE?
25 A YES, SIR.
26 Q ALL RIGHT. AND WHAT ARE YOU TRYING TO DO
27 WITH A BENCHMARK DOSE?
28 A MAY I DRAW IT SCHEMATICALLY?

1 Q SURE. I THINK IT WOULD HELP.
2 A I COULD DO IT REAL EASILY.
3 Q ALL RIGHT. PLEASE DO SO.
4 A WELL, ANIMAL RESPONSE IS -- BASICALLY,
5 ANYTHING YOU'RE GOING TO SEE IS USUALLY ABOVE 10
6 PERCENT, 10 PERCENT CANCER RESPONSE.
7 Q WHAT DOES THAT MEAN? YOU'RE DRAWING A
8 DOTTED LINE, I SEE --
9 A YES.
10 Q -- AND 10 PERCENT. WHAT DOES THAT MEAN, 10
11 PERCENT CANCER RESPONSE?
12 A IT MEANS IF I'VE GOT 5 ANIMALS -- IF I'VE
13 GOT 50 ANIMALS PER DOSE, AND 5 OF THEM GET CANCER, I GET
14 10 PERCENT RESPONSE.
15 Q OKAY. PLEASE PROCEED WITH YOUR EXPLANATION.
16 A SO THE WAY THE GOVERNMENTS NOW USE
17 LINEARIZED MULTISTAGE MODELING IS, THEY SAY, "LOOK, THE
18 REALLY IMPORTANT THING IS, WE'RE SHOWING YOU THAT WE
19 BELIEVE IN LINEARITY DOWN TO LOW DOSES. OKAY? BECAUSE
20 THAT'S THE WAY THE TOXICO-KINETIC PROCESSES FOR LOTS OF
21 CARCINOGENS WORK."
22 Q OKAY.
23 A "AND MUTAGENS."
24 BUT THEY SAY, "YOU KNOW, THESE CURVES MAY
25 NOT BE THE BEST. THEY MAY TAKE SOME SORT OF CURVATURE
26 UP LIKE THIS. AND YEAH, YOU'LL SEE -- IF YOU LIMIT IT
27 TO THE HIGH-DOSE SPECTRUM, THAT CURVE WILL LOOK -- IT
28 WILL LOOK LINEAR, OF COURSE, BECAUSE YOU'RE IN A

1 NARROWER RANGE; BUT IT MAY GO UP LIKE THIS.
2 "SO WE'RE GOING TO FIT THE BENCHMARK DOSE
3 SOFTWARE" -- AND IT'S BASED ON THE AGREEMENTS OF THE
4 AGENCIES, THAT -- "YOU KNOW, WE'RE GOING TO FIT A WHOLE
5 BUNCH OF CURVES THROUGH THESE DATA. IT MAY BE LINEAR;
6 IT MAY LOOK LIKE THIS.
7 "IT MAY -- THERE'S LOTS OF CURVES THAT WOULD
8 ACTUALLY LOOK LIKE THIS, OR SOMETHING, AND MAY EVEN BE
9 SUPER-LINEAR. OKAY? SO WE'RE GOING TO FIT MANY
10 DIFFERENT CURVES, OR LOTS OF DIFFERENT CURVES" --
11 IN FACT, I FIT 11 CURVES -- 11 OR 12 CURVES,
12 FOR ALL THE DATA POINTS.
13 BUT I SAID, "OKAY. I'M GOING TO" --
14 "BUT THEN, FROM THE 10 PERCENT POINT ON
15 DOWNWARD, WE'RE GOING TO DRAW A STRAIGHT LINE." THAT'S
16 WHAT THE BMD SOUGHT TO ASSESS.
17 "SO WE'RE GOING TO ALLOW FOR SOME CURVATURE
18 UP HERE, MAINLY TO GET A BEST ESTIMATE AROUND THE 10
19 PERCENT ASSENT LINE. WE DON'T ALWAYS HAVE A POINT AT
20 THE 10 PERCENT RESPONSE LINE, BUT WE FEEL THAT THE
21 LINEAR PROCESSES SHOULD PREVAIL FROM THE 10 PERCENT LINE
22 DOWN TO THE ORIGIN, OR ZERO RESPONSE."
23 SO THAT'S -- THE CONCEPT OF THE BENCHMARK
24 DOSING IS: FIT ANYTHING YOU WANT, BASICALLY, UP HERE.
25 GET DOWN TO THE 10 PERCENT LEVEL. ALLOW FOR SOME SORT
26 OF ERROR, STANDARD ERROR IN THE RESPONSE, AND DRAW A
27 STRAIGHT LINE DOWN TO THERE.
28 Q WOULD YOU DRAW ON THE BOARD RIGHT THERE

1 "BMD," TO INDICATE WHERE THE BENCHMARK DOSE IS TO BE?
2 A OH. THIS IS BMD HERE. IT'S THE DOSE
3 CORRESPONDING TO A 10 PERCENT RESPONSE.
4 Q ALL RIGHT. VERY GOOD. NOW, IF YOU'RE DONE,
5 PLEASE RESUME YOUR SEAT.
6 HAS THE CONCEPT OF A BENCHMARK DOSE BEEN
7 ACCEPTED BY THE U.S. EPA?
8 A YES.
9 Q BY CAL EPA?
10 A YES.
11 Q ALL RIGHT. AND THE SOFTWARE THAT YOU
12 MENTIONED THAT YOU USED, WAS THAT SOFTWARE THAT WAS
13 DEVELOPED BY THE EPA FOR PURPOSES OF DOING THESE
14 CALCULATIONS?
15 A IT WAS ACTUALLY DEVELOPED BY A CONTRACTOR
16 FOR THE EPA, UNDER THE AUSPICES. AND EPA -- IT USES THE
17 EPA IMPRIMATUR.
18 Q SO DID YOU USE THE BMD -- THE BENCHMARK DOSE
19 SOFTWARE TO DEVELOP YOUR CANCER POTENCY ESTIMATES?
20 A TO DEVELOP MY INDIVIDUAL CANCER POTENCY
21 ESTIMATES --
22 Q OKAY.
23 A -- NOT FOR THE COMBINED CANCER POTENCY
24 ESTIMATES.
25 Q OKAY. WHAT DOES THAT SOFTWARE DO?
26 A IT GIVES INDIVIDUAL SLOPES.
27 Q OKAY. DOES IT PROVIDE MODELS?
28 A THAT'S WHAT A SLOPE IS.

1 Q ALL RIGHT. THANKS FOR CLARIFYING THAT.
2 NOW, WHICH MODELS DID YOU USE, OR SLOPES DID
3 YOU USE?
4 A WELL, I USED THE LINEARIZED MULTISTAGE
5 MODELS. SO I ONLY USED THAT FAMILY OF MODELS TO -- AS
6 ACCEPTABLE FOR MY EXTRAPOLATION PROCEDURE.
7 Q OKAY. AND WHY DID YOU ONLY USE THAT FAMILY
8 OF MULTISTAGE LINEARIZED MODELS?
9 A BECAUSE LINEAR -- MULTISTAGE THEORY WAS
10 DEVELOPED FOR CANCER, CARCINOGENS.
11 AND IT'S -- THAT'S WHAT MULTISTAGE THEORY
12 IS: IT'S MULTISTAGE CANCER THEORY; THAT THE CELL HAS TO
13 GO THROUGH A SERIES OF MUTATIONS AND CHANGES UNTIL IT
14 REACHES THE CARCINOGENIC STATE, WHERE GROWTH IS -- CAN
15 NO LONGER BE INHIBITED.
16 Q AND HAS THAT THEORY BEEN GENERALLY ACCEPTED
17 IN THE COMMUNITY?
18 A YES, IT HAS.
19 Q ALL RIGHT. AND IS THAT THEORY THE BASIS FOR
20 THIS MODEL, FOR THE MULTISTAGE LINEARIZED MODELS?
21 A YES.
22 Q ALL RIGHT. HOW DID YOU USE THOSE MODELS?
23 A WELL, I USED THE MODELS FOR EACH OF THE
24 INDIVIDUAL TUMORS -- AND I JUST EXPLAINED THIS, I
25 THINK -- TO COMBINE THE POTENCIES FROM EACH INDIVIDUAL
26 MODEL FOR EACH SEX AND SPECIES, TO GET AN OVERALL CANCER
27 POTENCY FOR EACH SEX/SPECIES.
28 Q WERE YOU ATTEMPTING TO DETERMINE WHICH OF

1 THESE MODELS PROVIDE THE BEST FIT TO THE DATA?
2 A NO. I WAS ATTEMPTING TO DETERMINE WHICH
3 ANIMAL SPECIES, WHICH ANIMAL SEX, WAS THE MOST SENSITIVE
4 SPECIES, WAS THE MOST SENSITIVE STUDY.
5 Q OKAY. ALL RIGHT. HOW DID YOU GO ABOUT THIS
6 MODELING EXERCISE?
7 A I USED THE EPA -- I USED THE SAME METHOD
8 THAT EPA USED IN ITS 2010 ACRYLAMIDE RISK ASSESSMENT.
9 Q OKAY. DID YOU USE ANY CRITERIA TO ASSESS
10 GOODNESS OF FIT?
11 A WHEN -- I DID WHEN I WAS DEVELOPING MY
12 INDIVIDUAL MODELS.
13 Q OKAY. TELL US ABOUT THAT. HOW DID YOU GO
14 ABOUT THAT?
15 A I USED THE SAME CRITERIA PRETTY MUCH. THIS
16 IS FROM MY BENCHMARK DOSE SOFTWARE.
17 Q OKAY.
18 A SO I USED THREE CRITERIA. I USED ASTRA
19 GOODNESS OF FIT, WHICH IS A STANDARD STATISTICAL TEST:
20 HOW WELL DID THE DATA FIT THE MODEL -- HOW WELL DOES THE
21 MODEL FIT THE DATA? I'M SORRY.
22 I USED ANOTHER CRITERION, CALLED THE AKAIKE
23 INFORMATION CRITERION, WHICH IS VERY SIMILAR TO THE
24 ASTRA GOODNESS OF FIT EXCEPT THAT IT TAKES ACCOUNT OF
25 THE NUMBER OF PARAMETERS YOU'RE ESTIMATING.
26 AND THIRD, I MEASURED HOW WELL EACH MODEL
27 FIT THE DATA AT EACH ONE OF THE DATA POINTS. AND THAT
28 WAS CALLED A SCALE DEVIATION.

1 Q OKAY. IS THAT SOMETIMES CALLED THE SCALE
2 RESIDUAL TEST?
3 A YES.
4 Q ALL RIGHT.
5 A IT'S NOT A TEST; IT'S JUST A -- NO STATISTIC
6 BEHIND IT.
7 Q ALL RIGHT. HOW DID YOU CHOOSE THE BEST
8 MODEL?
9 A ON THE INDIVIDUAL TUMOR SITES?
10 Q YES.
11 A NUMBER ONE, I RESTRICTED MY MODELS TO
12 LINEARIZED MULTISTAGE MODEL FAMILY; CANCER LINEARIZED
13 MULTISTAGE MODEL FAMILY. AND SO THERE WERE FOUR OF
14 THOSE BECAUSE THERE WERE FOUR POSSIBLE MODELS.
15 AND THEN I CHOSE -- THE SOFTWARE WOULD KICK
16 OUT AND IT WOULD LOOK AT ALL THE MODELS AND FIT ALL THE
17 MODELS. AND IT WOULD SAY, "YEAH, BASED ON CERTAIN
18 TESTS," WHICH I JUST DESCRIBED, "THIS MODEL IS VIABLE,
19 THIS MODEL ISN'T VIABLE, AND THIS MODEL DOESN'T FIT THE
20 DATA WELL."
21 AND SO I LOOKED AT ALL THE MODELS THAT THE
22 COMPUTER GENERATED FOR ME AS VIABLE MODELS. AND THEN
23 FROM THOSE, I CHOSE THE MODEL, ALMOST ALWAYS, WITH THE
24 LOWEST AKAIKE INFORMATION CRITERIA.
25 NOW, THIS IS DIFFERENT FROM WHAT THE
26 COMPUTER USUALLY SAYS. THE COMPUTER WILL USUALLY
27 RECOMMEND A MODEL THAT GIVES THE HIGHEST RISK.
28 Q OKAY.

1 A BUT I FELT THAT WAS UNFAIR IN THIS CASE
2 BECAUSE IF -- BECAUSE NOW THEY'RE GIVING INDIVIDUAL
3 MODELS; RIGHT? BUT I'M INTERESTED IN THE OVERALL RISK,
4 NOT AN INDIVIDUAL MODEL FOR EACH DATASET.
5 AND IF I KEEP CHOOSING -- FOR EACH ONE OF MY
6 INDIVIDUALIZED MODELS, IF I KEEP CHOOSING THE ONE THAT
7 GIVES THE HIGHEST RISK, THEN I'M GOING TO OVERESTIMATE
8 THE RISK.
9 SO I CHOSE THE ONE BASED ON THE LOWEST
10 AKAIKE INFORMATION CRITERIA, WHICH IS A BETTER FITTING
11 MODEL.
12 Q OKAY.
13 A SO I DIDN'T -- I DEVIATED HERE BECAUSE,
14 REMEMBER, I'M TRYING TO COMBINE TUMORS TO GET THE MOST
15 SENSITIVE STUDY. I'M NOT TRYING TO GET THE ONES -- IF I
16 KEEP ADDING MODELS, INDIVIDUAL TUMOR SITES, WITH HIGHER
17 RISKS, I'M GOING TO GET A RISK THAT'S JUST FAR TOO HIGH,
18 I THINK.
19 Q OKAY. AND YOU MENTIONED YOU USED THE -- YOU
20 RELIED ON THE AKAIKE INFORMATION CRITERIA, THE MODEL
21 THAT HAD THE LOWEST VALUE FOR THAT?
22 A YES.
23 Q WHY IS THAT? WHY DID YOU DO THAT?
24 A BECAUSE IT'S A BASICALLY ACCEPTED TEST, AND
25 I HAD -- AND BECAUSE THE COMPUTER GENERATED IT. AND IT
26 WAS -- I CONSIDERED THAT AN UNBIASED TEST OF RISK.
27 Q OKAY.
28 A SO THE AKAIKE INFORMATION CRITERIA WASN'T

1 NECESSARILY RELATED TO THE MODELS THAT GAVE THE HIGHEST
2 RISK, JUST THOSE THAT FIT THE DATA BETTER.
3 Q ALL RIGHT. AND UNDER -- USING THE AKAIKE
4 INFORMATION CRITERIA, WHAT IS THE IMPORT OF THE LOWEST
5 VALUE? WHAT DOES THAT INDICATE?
6 A BETTER FIT.
7 Q BETTER FIT OF WHAT?
8 A THE MODEL TO THE DATA.
9 Q OKAY. AND THAT'S WHAT YOU'RE TRYING TO
10 ACCOMPLISH?
11 A I'M TRYING TO -- WELL, IT'S -- ULTIMATELY,
12 WHAT I'M TRYING TO ACCOMPLISH IS FINDING OUT WHICH
13 ANIMAL STUDY IS THE MOST SENSITIVE.
14 Q ALL RIGHT. NOW, DID YOU PREPARE TABLES OF
15 YOUR CALCULATIONS AND RESULTS FOR THE INDIVIDUAL DOSE-
16 RESPONSE CANCER SLOPES?
17 A YES, I DID.
18 MR. METZGER: ALL RIGHT. COULD WE HAVE --
19 THE WITNESS: YOUR HONOR?
20 THE COURT: YES.
21 THE WITNESS: I'M SORRY, BUT I HAVE TO GO TO THE
22 BATHROOM.
23 THE COURT: ALL RIGHT. WE'RE GOING TO TAKE A
24 FIVE-MINUTE RECESS.
25 THE WITNESS: THANK YOU.
26 MR. METZGER: YOUR HONOR, I'M WONDERING IF --
27 SHOULD WE RECESS FOR LUNCH AT THIS POINT, IF WE'RE GOING
28 TO --

1 THE COURT: OKAY. HOW MUCH LONGER ARE YOU GOING
2 TO BE WITH THE WITNESS?
3 MR. METZGER: OH, IT WILL BE ALL DAY AND INTO
4 TOMORROW. HE HAS TWO QUANTITATIVE CANCER RISK
5 ASSESSMENTS TO PRESENT. WE'RE AT THE BEGINNING OF THE
6 FIRST ONE.
7 THE COURT: IT WILL BE TODAY AND INTO TOMORROW.
8 WHAT TIME TOMORROW DO YOU THINK YOU'RE GOING TO
9 CONCLUDE?
10 MR. METZGER: I HOPE TO BE FINISHED BY NOON, BUT
11 IT COULD TAKE ALL DAY.
12 THE COURT: BY NOON TOMORROW.
13 HOW MUCH CROSS-EXAMINATION?
14 MR. SCHURZ: IF THE WITNESS IS TURNED OVER TO US
15 BEFORE LUNCH TOMORROW, WE'LL CERTAINLY FINISH TOMORROW,
16 YOUR HONOR.
17 THE COURT: THEN THERE'S -- SO PLAINTIFF WILL REST
18 AFTER DR. BAYARD?
19 MR. METZGER: WE WILL REST AFTER YOU HAVE RULED ON
20 THE -- ADMITTED THE TESTIMONY OF THE PMK'S; SO BASED ON
21 YOUR RULINGS. AND WE'VE CLEANED UP A FEW THINGS WITH
22 EXHIBITS. BUT HE'S OUR LAST WITNESS, OUR LAST LIVE
23 WITNESS.
24 THE COURT: OUR LAST WITNESS. OKAY.
25 AND DO YOU HAVE DR. GOODMAN, WHO IS --
26 MR. SCHURZ: YES, WE WILL BE OFFERING DR. JULIE
27 GOODMAN AS OUR SOLE REBUTTAL WITNESS AT THIS TIME.
28 THE COURT: HOW LONG WILL THAT TESTIMONY TAKE?

1 MR. SCHURZ: WE EXCEPT A DAY OR LESS.
2 THE COURT: OKAY.
3 MR. METZGER: COULD I JUST INQUIRE: IS SHE COMING
4 THIS WEEK OR THE FOLLOWING WEEK? I THINK YOU SAID
5 INITIALLY --
6 THE COURT: WE'RE GOING TO FINISH THIS WEEK;
7 RIGHT?
8 MR. SCHURZ: YOUR HONOR, CONSISTENT WITH WHAT WE
9 TOLD YOU EARLIER, DR. GOODMAN HAS BOTH FAMILIAL AND
10 PROFESSIONAL RESPONSIBILITIES IN BOSTON THIS WEEK THAT
11 PRECLUDE HER FROM TRAVELING. SO AS I INDICATED EARLIER,
12 IT WAS OUR HOPE THAT WE WOULD PRODUCE HER ON MONDAY.
13 THE COURT: HOW LONG WILL HER TESTIMONY TAKE?
14 MR. SCHURZ: IT'LL BE OVER IN A DAY. I CAN'T
15 IMAGINE IT WOULD TAKE MORE.
16 MR. METZGER: AND WE HAVE THE MOTION TO EXCLUDE
17 THAT, WHICH I THINK --
18 THE COURT: I KNOW; I KNOW. ALL RIGHT.
19 MR. METZGER: SOUNDS LIKE YOU'RE NOT GOING TO
20 GRANT THAT. OKAY.
21 THE COURT: WE'LL TALK ABOUT THAT.
22 ALL RIGHT, COUNSEL. WE'LL BE IN RECESS,
23 THEN, TILL 1:30 -- 1:45.
24 MR. METZGER: 1:45. THANK YOU, YOUR HONOR.
25 OH, ONE OTHER THING, YOUR HONOR. TODAY WE
26 HAVE FILED A MOTION TO STRIKE PART -- A PART OF DR.
27 MURRAY'S TESTIMONY. I EXPECT YOU'RE GOING TO WANT TO
28 SCHEDULE THAT FOR BRIEFING AND A DECISION DATE.

1 THE COURT: OKAY. I MAY CONSOLIDATE ALL THESE
2 MOTIONS TO STRIKE AND HAVE THEM HEARD AFTER YOU FINISH
3 THE TESTIMONY, BUT I'LL TELL YOU ABOUT THAT.
4 ALL RIGHT. THANK YOU.
5 MR. METZGER: THANK YOU, YOUR HONOR.
6 THE COURT: BE IN RECESS TILL 1:45.
7 (AT 11:48 A.M., A LUNCH RECESS WAS TAKEN
8 UNTIL 1:45 P.M. OF THE SAME DAY.)
9 (TRANSCRIPT CONTINUES ON PAGE 151.)
10
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EXHIBIT “H”

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. STARBUCKS CORPORATION, A CALIFORNIA CORPORATION, ET AL., DEFENDANTS.	CASE NO. BC435759
AND CONSOLIDATED ACTION.	

REPORTER'S TRANSCRIPT OF TRIAL PROCEEDINGS

MONDAY, OCTOBER 27, 2014

AFTERNOON SESSION

APPEARANCES:

FOR THE PLAINTIFF: METZGER LAW GROUP
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FOR THE DEFENDANT: MORRISON & FOERSTER
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CCROLA JOB NO. 114689

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

I N D E X

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

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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?

A YES.
Q AFTER YOUR DEPOSITION, DID YOU MAKE CORRECTIONS TO YOUR DEPOSITION TRANSCRIPT?
A YES, SIR.
Q I WOULD LIKE TO SHOW YOU WHAT HAS BEEN MARKED AS EXHIBIT 130. OTHER THAN THE FIRST PAGE, WHICH IS THE TRANSMITTAL PAGE FOR SENDING YOUR DEPOSITION CORRECTIONS, WOULD YOU CONFIRM THAT THE DOCUMENTS WITHIN THAT EXHIBIT, THE PAGES WITHIN THAT EXHIBIT, ARE CORRECTIONS THAT YOU MADE TO YOUR DEPOSITION?
A THESE ARE MY CORRECTIONS.
Q DID THAT INCLUDE CORRECTIONS TO THE -- TO SOME WORDS AND NUMBERS IN THE TESTIMONY THAT YOU GAVE?
A PLEASE REPEAT THE QUESTION.
Q DID THE CORRECTIONS INCLUDE CORRECTIONS TO SOME WORDS IN THE TESTIMONY THAT YOU GAVE?
A YES.
Q DID IT ALSO INCLUDE SOME CORRECTIONS TO DATA THAT YOU PROVIDED IN THE TABLES IN YOUR REPORT, WHICH IS EXHIBIT 120?
A THE CORRECTIONS WEREN'T TO THE DATA, THEY WERE TO THE RESULTS.
Q FINE. LET ME SEE IF I UNDERSTAND. SO ARE THERE -- THERE ARE TABLES IN EXHIBIT 120, WHICH IS YOUR REPORT; CORRECT?
A YES.
Q AND THEY SET FORTH CERTAIN RESULTS OF CALCULATIONS THAT YOU PERFORMED; CORRECT?

A YES.

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

A YES.

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

MR. SCHURZ: I WILL OBJECT.

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

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

A YES, BUT I THINK IT WAS SLIGHTLY EARLIER.

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

A JUNE 13TH, 2014.

Q ALL RIGHT.

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

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

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

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

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

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

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

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

A ALL THESE MODELS FIT THE DATA EXTREMELY WELL.

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

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

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

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

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

Q AND WHAT DID YOU CONCLUDE FROM THE RESULTS HERE?

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

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?

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

Q WHY NOT?

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

Q OKAY.

A THAT IS A SIMPLE ALGEBRA I PROBLEM.

Q TELL US THAT.

A WELL, THAT WOULD BE IN THE NEXT SLIDE.

Q OKAY.

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

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

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

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

Q OKAY. SLIDE 149.

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.

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

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

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

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

THE COURT: THANK YOU.

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

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

THE COURT: WE WILL RECESS UNTIL TOMORROW MORNING.

MR. METZGER: THANK YOU, YOUR HONOR.

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

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. STARBUCKS CORPORATION, A CALIFORNIA CORPORATION, ET AL., DEFENDANTS. AND CONSOLIDATED ACTION.	CASE NO. BC435759

I, KAREN VILICICH, CSR NO. 7634, OFFICIAL COURT REPORTER OF THE SUPERIOR COURT OF THE STATE OF CALIFORNIA, FOR THE COUNTY OF LOS ANGELES, DO HEREBY CERTIFY THAT THE FOREGOING PAGES 151 THROUGH 225 COMPRISE A FULL, TRUE AND CORRECT TRANSCRIPT OF THE TESTIMONY AND PROCEEDINGS HELD IN THE ABOVE-ENTITLED MATTER ON MONDAY, OCTOBER 27, 2014.

DATED THIS 27TH DAY OF OCTOBER, 2014.

KAREN VILICICH, CSR NO. 7634
OFFICIAL REPORTER PRO TEMPORE

EXHIBIT “I”

Steven P. Bayard
[redacted] Md. [redacted]
Tel. H: 301[redacted] C: 301-[redacted]
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E-mail. spbayard@aol.com

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.

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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.

Bayard S, Jinot J.
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.

Repace J, Jinot J, Bayard S, et al
Air Nicotine and Saliva Cotinine as Indicators of Workplace Passive Smoking Exposure and Risk. Risk Analysis. 1998. Vol. 18, No. 1, pgs. 71-83.

Ward EM, Schulte PA, Bayard S, et al
Priorities for Development of Research Methods in Occupational Cancer. Env. Health Persp. 2003. Vol. 111, No.1, pgs. 1-124

Toraason M, Albertini R, Bayard S, et al
Applying New Buotechnologies 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 Forrest 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.

Bayard SP, Greenberg R, Showalter D, Byar D.
An Analysis of Treatment Data from Patients with Prostatic Cancer Using an Exponential Model Relating Survival to Concomitant Variables. Cancer Chemotherapy Reports. 11-12/74, Vol. 58, No. 6, 845-859.

Greenberg R, Bayard S, Byar D.
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Bayard SP, Statistical Principles in Health Care Information, by S. James Kilpatrick, Jr., A Review. Health Laboratory Science, Oct. 1974, Vol. 11; No. 6.

Sanen FJ, Van Scott E, Bayard SP.
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Osterberg R, Bayard SP, Ulsamer, AG.
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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.
Qualitative and Quantitative Cancer Risk Assessment of 2,3,7,8-TCDD. Presented at the 5th International Symposium on Chlorinated Dioxins and Related Compounds. Bayreuth

(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.
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Hiremath C, Bayard SP.
Cancer Risk Characterization of HxCDD. Presented at the 7th International Symposium on Chlorinated Dioxins and Related Compounds. Las Vegas, Nev., Oct. 1987.

Cote I, Bayard SP.
EPA's Risk Assessment of 1,3-Butadiene. Presented at the Nat. Institute of Environ. Health Sciences' International Symposium on 1,3-Butadiene. RTP, NC, Apr. 1988.

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.

Cote I, Bayard SP.
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.