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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. 5

Dear Ms. Vela:

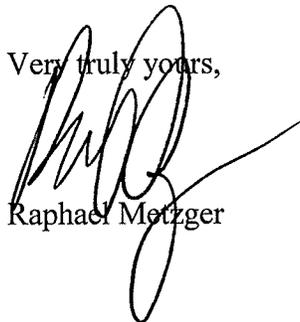
Enclosed herewith are the following documents that are being submitted on behalf of our client, the Council for Education and Research on Toxics (CERT) regarding the Opinions of Dr. James E. Huff Regarding the Carcinogenicity of Acrylamide to Animals and Relevance to Humans.

1. Exhibit A - Opinions of Dr. James E. Huff
2. Exhibit B - Acrylamide, Glycidamide, Methyloacrylamide Carcinogenic Tumor Site Comparisons.
3. Exhibit C - Testimony of Dr. James E. Huff in *CERT v. Starbucks* trial, October 22, 2014 a.m.
4. Exhibit D - Testimony of Dr. James E. Huff in *CERT v. Starbucks* trial, October 22, 2014 p.m.
5. Exhibit E - Biography of Dr. James E. Huff
6. Exhibit F - Publications List of Dr. James E. Huff

Monet Vela
OEHHA
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Kindly include these materials of Dr. James E. Huff in the record for this rulemaking proceeding.

Very truly yours,

A handwritten signature in black ink, appearing to read 'Raphael Metzger', with a long horizontal flourish extending to the right.

Raphael Metzger

RM:ip
encls: as specified

EXHIBIT “A”

OPINIONS OF JAMES HUFF

Value and Public Health Significance of Cancer Bioassays

1. Rodents and humans are mammals, there are more similarities - physiologically, pharmacologically, biochemically, genomically - than differences. (Huff 258, Huff 432)
2. All known human carcinogens that could be tested experimentally are likewise carcinogenic to animals. (Huff 258, Huff 288, Huff 289, Huff 331, Huff 339, Huff 393, Huff 432)
3. Nearly one-third of human carcinogens were first discovered in animal bioassays. (Huff 331, Huff 432)
4. One-third would likely be larger but several human carcinogens were discovered in early industrial times, predating standard, more frequent bioassays, and some human carcinogens are undefined "exposure circumstances" (eg, aluminum production, furniture/cabinet making, rubber industry) not readily testable in animals. (Huff 432)
5. For those chemicals known as both animal and human carcinogens, there is at least one common cancer-induced tissue/organ site between both mammalian species. (Huff 288, Huff 432, Tomatis 1989))
6. Findings from independently conducted bioassays on the same chemicals are consistent, albeit sometimes with additional or different target sites. (Huff 381, Huff 432)
7. Bioassays both predict (prospective: 1,3-butadiene, TCDD, TCE, VCM) or confirm (retrospective: arsenic, benzene) human carcinogenicity. (Huff 432)
8. Most chemicals early studied in animals had an a priori suspicion of being carcinogenic, while later randomly selected chemicals identified fewer carcinogens. (Huff 306, Huff 432)
9. Less than 10% to 15% of all chemicals if evaluated in bioassays would be predicted to be carcinogenic. (Huff 154, Huff 255, Huff 306, Huff 432)
10. No other in vitro assay or in vivo bioassay or combination of tests, or even epidemiology, can claim these collective facts and advantages. (Huff 331, Huff 432)
11. Most chemical carcinogens do not cause cancer only at the highest exposures. (Huff 255)
12. Carcinogenicity findings from experiments on laboratory animals are logical and scientifically reasonable for identifying and predicting potential carcinogenic effects to humans. (Huff 255, Huff 324, NTP RoC 2011, IARC 2014)

Specific Evidence Supporting Extrapolation from Animals to Humans

1. Cancers induced at multiple sites in animals increase the prediction of human cancer. (Huff 155, Huff 206, Huff 255, Huff 331; Rice 2005, NTP 2012)
2. Cancers induced in multiple species increase the prediction of human cancer. (Huff 255, NTP RoC (2012), IARC (2014))
3. Cancers induced in multiple strains of the same species increase the prediction of human cancer. (Huff 432, NTP RoC 2011, IARC 2014)
4. Cancers induced in both sexes of animals increase the prediction of human cancer. (Huff 255)
5. Multiple cancers induced in the same organ or bilaterally (eg both kidneys) increase the prediction of human cancer. (NTP 2012)
6. Cancers that metastasize increase the prediction of human cancer. (NTP 2012)
7. A high tumor incidence rate increases the prediction of human cancer. (Huff 255)
8. A tumor dose-response relationship increases the prediction of human cancer. (Huff 255, NTP 2012)
9. Rare or uncommon cancers in animals increase the prediction of human cancer. (Huff 255, NTP 2012)
10. Cancers in animals induced by a genotoxic mechanism increase the prediction of human cancer. (Huff 331, NTP 2012, IARC Preamble)

Evidence of Carcinogenicity of Acrylamide and its Metabolite

1. Acrylamide and its metabolite glycidamide produce cancer at multiple sites in animals (Huff concordance summary, Rice 2005, NTP 2012, NTP 2013)
2. Acrylamide and glycidamide produce cancer in two species that have been studied - rats and mice. (Johnson 1985, Friedman 1995, NTP 2012, NTP 2013).
3. Acrylamide produces cancers in different strains of mice. (Bull 1984, Bull 1984, Rice 2005)
4. Acrylamide produces cancers in both sexes of rats and mice. (Johnson 1985, Friedman 1995, NTP 2012).
5. Acrylamide and glycidamide produces multiple cancers in the same organ in animals and bilaterally, e.g., the Harderian gland. (Bull 1984, Johnson 1985, NTP 2012, NTP 2013)
6. Acrylamide and glycidamide produce high incidences of induced tumors in animals. (NTP 2012, NTP 2013)

7. Acrylamide and glycidamide produce cancers in animals with a dose-response relationship. (Johnson 1985, Friedman 1995, NTP 2012, NTP 2013).
8. Acrylamide and glycidamide induce rare tumors in animals - e.g., schwannomas of the heart, mesotheliomas of the epididymis (tunica vaginalis tumors), follicular cell thyroid tumors, oral cavity tumors, forestomach tumors, and brain tumors. (NTP 2012, NTP 2013)
9. Acrylamide and glycidamide induce cancers at low exposures of 6-8 ppm. (NTP 2012, NTP 2013)
10. Acrylamide and glycidamide induce cancers in animals by a genotoxic mechanism. (IARC 1994, Rice 2005, NTP 2012, NTP 2013)
11. There is strong concordance between rodent tumors induced by acrylamide and those induced by glycidamide. (Huff concordance summary, NTP 2012, NTP 2013)
12. "[T]hat acrylamide is a multiorgan carcinogen in both rats and mice . . . strongly imply that acrylamide presents a potential carcinogenic hazard to humans." (Rice 2005)

Mechanistic Evidence of Acrylamide Carcinogenicity

1. Acrylamide produces cancer through a genotoxic mechanism - metabolism to glycidamide. (Rice 2005, NTP 2012, NTP 2013, Beland 2013)
2. Chemicals or their metabolites that are carcinogenic to the central nervous system in rodents are usually genotoxic. (Rice 2005) Acrylamide and glycidamide are carcinogenic to the central nervous system of rats. (Johnson 1986, Rice 2005, NTP 2012, NTP 2013)
3. Acrylamide and its metabolite form covalent adducts with DNA in mice and rats. (IARC 1994)
4. Acrylamide and its metabolite form covalent adducts with hemoglobin in humans and rats. (IARC 1994)
5. Acrylamide induces gene mutations and chromosomal aberrations in germ cells of rodents. (IARC 1994)
6. Acrylamide induces chromosomal aberrations in somatic cells of rodents in vivo. (IARC 1994)
7. Acrylamide induces gene mutations and chromosomal aberrations in cultured cells in vitro. (IARC 1994)
8. Acrylamide induces cell transformation in rodent cell lines. (IARC 1994)
9. Structure-activity relationships support the carcinogenicity of acrylamide. (Huff concordance summary)

Relevance of Animal Data to Quantitative Human Cancer Risk

1. Animal bioassay data from rats and mice and other species should be used to assess human cancer risk. (Huff 255, NTP RoC 2011, IARC Preamble 2006, Rice 2005)
2. Data from all tumor sites should be evaluated and those sites showing clear or strong evidence of carcinogenicity should be used for purposes of human cancer risk assessment. (Huff 379, Huff 432, NTP RoC 2011, IARC Preamble 2006)
3. Tumors induced in organs that are unique to rodents (eg forestomach, Harderian gland, preputial gland, Zymbal gland) should not be excluded from human cancer risk assessment because human carcinogens induce these tumors (Huff 263, Huff 432, Rice 2005)
4. Analyses of all tumor sites should be combined for estimating human cancer risk. (Huff 432)
5. Benign tumors induced by chemicals are relevant for judging human carcinogenicity, as few chemicals produce only benign tumors, and most progress to malignancy. (Huff 203, Huff 255, IARC Preamble 2006)
6. Most chemical carcinogens do not cause cancer only at the highest exposures used, and for those few that do there are no scientific reasons that these should not be considered relevant for human cancer hazard identification. (Huff 255)

Carcinogenicity of Coffee and its Constituents

1. The available cancer bioassays of coffee were considered inadequate for evaluating the carcinogenicity of coffee when IARC did its evaluation. (IARC 1991)
2. More recent cancer bioassays of coffee are also inadequate for evaluating the carcinogenicity of coffee.
3. Coffee has been shown to enhance the carcinogenicity of DMBA. (Saroja 2001)
4. Of the more than 1,000 chemicals identified in coffee by 1998 only 32 had been tested for carcinogenicity in animals and 23 (72%) had been found to be carcinogenic. (Gold 2005)
5. The 23 chemical carcinogens that had been identified in coffee by 1998 are acetaldehyde, acrylamide, benzaldehyde, benzene, benzofuran, benzo(a)pyrene, caffeic acid, catechol, 1,2,5,6-dibenzanthracene, ethanol, ethylbenzene, formaldehyde, furan, furfural, hydrogen peroxide, hydroquinone, isoprene, limonene, 4-methylcatechol, styrene, toluene, and xylene. (Ames 1997, Gold 2002)

6. Benzene is a known human carcinogen. (Huff 170, Huff 191, Huff 206, Huff 263, Huff 448, Huff 452, IARC 2012, NTP RoC 2011).
7. Benzo(a)pyrene is a known human carcinogen. (IARC 2012)
8. Formaldehyde is a known human carcinogen. (IARC 2012, NTP RoC 2011)
9. Hydroquinone, a metabolite of benzene, is a carcinogen. (Huff 252, Huff 287)
10. Ethyl benzene, toluene and xylene, congeners of benzene, have been shown to be carcinogenic. (Huff 213, Huff 433)
11. Catechol and phenol, benzene metabolites, are carcinogens. (Huff 206, Huff 318)
12. Eugenol, present in coffee, is a carcinogen. (Huff 139, NTP 1983)
13. Isoeugenol, another constituent of coffee, is a carcinogen. (NTP 2010)
14. Methyl eugenol, another constituent of coffee, is also a carcinogen. (NTP 2000)
15. IQ (2-amino-3-methylimidazo/4,5-f/quinoline) and MeIQ (2-amino-3,4-dimethylimidazo/4,5-f/quinoline) are carcinogens in coffee. (IARC 1993, NTP RoC 2011)
16. Ochratoxin A, a contaminant of green, roasted, instant, and ready-to-drink coffee, is carcinogenic. (Huff 248, IARC 1993, NTP 1989, NTP 2011)
17. Aflatoxin, another contaminant in coffee, is a known human carcinogen. (IARC 2012, NTP RoC 2011)
18. One method for assessing carcinogenic potency is the Human Exposure/Rodent Potency (HERP) Index. Coffee has the highest HERP index of all foods evaluated. (Ames 1987)

Mutagenicity of Coffee

1. "Brewed coffee induced chromosomal aberrations and sister chromatid exchange in cultured human lymphocytes. Sister chromatid exchange was also induced in cultured mammalian cells." (IARC 1991)
2. "In bacteria, [brewed, instant and decaffeinated coffee] was mutagenic, particularly to strains with enhanced sensitivity to oxidative mutagens, and induced DNA damage." (IARC 1991)
3. Instant coffee "induced chromosomal aberrations in cultured human lymphocytes and induced mutations and sister chromatid exchange in cultured mammalian cells." (IARC 1991)
4. "The urine of coffee drinkers was not mutagenic to bacteria but induced chromosomal aberrations [clastogenicity] in cultured mammalian cells." (IARC 1991)

EXHIBIT “B”

Acrylamide [AA], Glycidamide [GA], Methyloacrylamide [MAA]
Carcinogenic Tumor Site Comparisons

	AA [NTP TR 575, July 2012]	GA [NTP TR 588, Oct 2013]	MAA [NTP TR 352, Sept 1989]
Tumor Sites	<ul style="list-style-type: none"> *Clitoral gland fr *Epididymis mr *Forestomach mm, fm **Harderian gl mm, fm *Heart mr, fr Liver fr **Lung mm, fm *Mammary gland fr, fm *Oral mucosa fr Ovary fm Pancreatic islets mr *Skin fr, fm *Testes mr *Thyroid gland mr, fr *Tongue fr 	<ul style="list-style-type: none"> Brain mr, fr *Clitoral gland fr *Epididymis mr *Forestomach fr, mm, fm *Harderian gl mm, fm *Heart mr Leukemia mr?, fr *Lung mm, fm *Mammary gland fr, fm *Oral mucosa mr, fr *Skin mm, fm *Testes mr *Thyroid gland mr, fr *Tongue mr, fr 	<ul style="list-style-type: none"> *Harderian gl mm, fm ***Liver mm, fm *Lung mm, fm ***Ovary fm

Totals 15: mr 5, fr 8, mm 3, fm 6 14: mr 8, fr 8, mm 4, fm 5 4: mm 3, fm 4

=====

mr = male rats; fr = female rats; mm = male mice; fr = female mice

Concordant between: * AA & GA; ** AA, GA, MAA; *** AA, MAA

Virtual concordance of tumor sites in acrylamide and glycidamide bioassays supports the hypothesis that carcinogenic activity of acrylamide likely derives primarily from metabolism to glycidamide in both sexes of both species.

AA Groups of 48 male & 48 female F344/N rats & B6C3F1 mice were offered acrylamide in drinking water for 2 years. Concentrations of 0, 0.0875, 0.175, 0.35, 0.70 mM AA (0, 6.25, 12.5, 25, 50 ppm AA) resulted in average daily consumption of ~ 0.33, 0.66, 1.32, 2.71 mg AA/kg body weight in male F344/N rats, ~0.44, 0.88, 1.84, 4.02 mg/kg in female rats, ~1.04, 2.20, 4.11, 8.93 mg/kg in male mice, ~1.10, 2.23, 4.65, 9.96 mg/kg in female mice.

Exposure to acrylamide was associated with increased incidences of degeneration of the retina and sciatic nerve in male and female rats; preputial gland duct ectasia in male rats; adrenal cortex hypertrophy and cytoplasmic vacuolization, bone marrow hyperplasia, ovarian atrophy, and spleen hematopoietic cell proliferation in female rats; cataracts of the eye, spleen hematopoietic cell proliferation, and forestomach epithelial hyperplasia in male and female mice; preputial gland inflammation and lung epithelial hyperplasia in male mice; and ovarian cysts in female mice.

GA Groups of 48 male & 48 female F344/N NCTR rats & B6C3F1/NCTR mice were offered glycidamide in drinking water for 2 years. Concentrations of 0.0875, 0.175, 0.35, and 0.70 mM GA (0, 7.65, 15.3, 30.6, 61.2 ppm GA) resulted in average daily consumption of ~0.39, 0.79, 1.56, 3.34 mg GA/kg body weight in male rats, ~0.54, 1.08, 2.23, 4.65 mg/kg in female rats, ~1.20, 2.65, 5.13, 9.55 mg/kg in male mice, ~1.37, 2.89, 5.64, 12.99 mg/kg in female mice.

Exposure to glycidamide increased: fibrosis of spleen, exfoliated germ cells within epididymis (males), hepatocyte degeneration (males), liver necrosis (males), bone marrow hyperplasia (females), mesenteric lymph node cellular infiltration (females), pituitary gland (pars distalis) hyperplasia (females), axonal degeneration of lumbar spinal cord (females), and uterine endometrial hyperplasia (females).

In male and female mice, GA increased: cataracts, corneal inflammation, forestomach squamous cell hyperplasia, hematopoietic cell proliferation of spleen, preputial gland lesions (degeneration, ductal dilatation, inflammation) (males), ovarian cysts (females), hepatic angiectasis and necrosis (females), axonal degeneration of cervical spinal cord (females).

MAA 2-year studies were conducted by administering 0, 6, or 12 mg N-methylolacrylamide/kg body weight in water by gavage, 5 days per week for 103 weeks, to groups of 50 male & female F344/N rats and to groups of 50 male & female B6C3F1 mice at 0, 25, or 50 mg MAA/kg.

In rats, because no biologically important toxic effects were attributed to N-methylolacrylamide administration, higher doses could have been used to increase the sensitivity of these studies for determining the presence or absence of a carcinogenic response.

Hence, rat bioassay of MAA considered inadequate.

In female mice, ovarian atrophy was MAA related.

EXHIBIT “C”

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

WEDNESDAY, OCTOBER 22, 2014

MORNING SESSION

APPEARANCES:

FOR THE PLAINTIFF:

METZGER LAW GROUP
BY: RAPHAEL METZGER
KEN HOLDREN
MICHAEL CABRAL
401 EAST OCEAN BOULEVARD
SUITE 800
LONG BEACH, CA 90802
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FOR DEFENDANTS:

MORRISON & FOERSTER
BY: MICHELE B. CORASH
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425 MARKET STREET
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(415) 268-7124

CCROLA JOB
NO. 114675

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

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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 WEDNESDAY, OCTOBER 22, 2014
6 TIME: 9:12 A.M.
7 APPEARANCES: (AS HERETOFORE NOTED.)
8

9 THE COURT: GOOD MORNING, COUNSEL. CALLING THE
10 CASE OF CERT VS. STARBUCKS.

11 COUNSEL, YOUR APPEARANCES, PLEASE.

12 MR. METZGER: GOOD MORNING, YOUR HONOR. RAPHAEL
13 METZGER, KEN HOLDREN, AND MIKE CABRAL FOR THE PLAINTIFF.

14 MR. SCHURZ: GOOD MORNING, YOUR HONOR. JAMES
15 SCHURZ AND MICHELE CORASH ON BEHALF OF THE COFFEE
16 DEFENDANTS.

17 THE COURT: GOOD MORNING. WE'RE HERE TO RESUME
18 THE TRIAL. DR. INFANTE IS ON THE STAND.

19
20 PETER FRANCIS INFANTE,
21 CALLED AS A WITNESS BY THE PLAINTIFF, HAVING BEEN
22 PREVIOUSLY SWORN, TESTIFIED FURTHER AS FOLLOWS:

23 THE COURT: PLEASE RESTATE YOUR NAME FOR THE
24 RECORD.

25 THE WITNESS: PETER FRANCIS INFANTE.

26 THE COURT: DR. INFANTE, YOU UNDERSTAND YOU'RE
27 STILL UNDER OATH?

28 THE WITNESS: YES, YOUR HONOR.

1 THE COURT: AND MR. SCHURZ WAS INQUIRING.

2 COUNSEL, YOU MAY PROCEED.

3 MR. SCHURZ: THANK YOU, YOUR HONOR.

4 AND JUST AS A POINT OF PROCEDURE, BEFORE WE
5 BEGIN, WE'D LIKE TO CONFIRM THAT ALL OF THE NOTES THAT
6 DR. INFANTE HAS, WE ALSO HAVE. WE'VE OBSERVED HIM
7 TAKING NOTES THROUGHOUT THE COURSE OF HIS EXAMINATIONS,
8 AND HE HAS A FILE THERE THAT I DON'T KNOW WHAT THAT IS.
9 BUT I WOULD --

10 THE COURT: WELL, ANY NOTES THAT WERE PRODUCED
11 BEFORE THE EXAMINATION THAT DR. INFANTE IS REVIEWING IN
12 CONNECTION WITH HIS TESTIMONY SHOULD BE PRODUCED, BUT HE
13 DOES NOT HAVE TO PRODUCE NOTES THAT HE'S TAKING WHILE
14 HE'S TESTIFYING.

15 MR. SCHURZ: ALL RIGHT. THANK YOU, YOUR HONOR.

16

17 CROSS-EXAMINATION (RESUMED)

18 BY MR. SCHURZ:

19 Q GOOD MORNING, DR. INFANTE.

20 A GOOD MORNING.

21 Q I'D LIKE TO START THIS MORNING WITH SOME
22 DISCUSSION ABOUT YOUR ANALYSIS OF WHAT YOU DENOMINATED
23 AS THE POTATO STUDIES.

24 AND YOU'LL RECALL YESTERDAY AND THE DAY
25 BEFORE YOUR DISCUSSION WITH MR. METZGER RELATING TO THE
26 POTATO STUDIES AND THE INCREASED INCIDENCE OF CANCER
27 THAT YOU SAW WITH RESPECT TO THOSE STUDIES.

28 DO YOU RECALL THAT DISCUSSION WITH MR.

1 METZGER?

2 A YES.

3 Q AND IF WE COULD TAKE A LOOK AT DEMONSTRATIVE
4 NO. 77. AND HERE YOU IDENTIFY A GROUP OF THE
5 CASE-CONTROL STUDIES THAT YOU EVALUATED RELATING TO
6 POTATO CONSUMPTION.

7 AND YOU'VE SEGREGATED THEM -- OR ORGANIZED
8 THEM BY YEAR, THE FIRST SLIDE INDICATED 1975 TO 2006;
9 AND THE SECOND SLIDE, AT DEMONSTRATIVE 79, THAT REFLECTS
10 THE TIME PERIOD 2007 TO 2014; IS THAT CORRECT?

11 A YES, IT IS.

12 Q OKAY. AND YOU IDENTIFIED THESE STUDIES IN
13 SUPPORT OF YOUR OPINION REGARDING THE HUMAN
14 CARCINOGENICITY OF ACRYLAMIDE; IS THAT CORRECT?

15 A YES.

16 Q AND YOU READ THESE STUDIES?

17 A YES.

18 Q AND YOU EVALUATED THE QUALITY OF THESE
19 STUDIES FOR PURPOSES OF RENDERING YOUR OPINION IN THIS
20 CASE; CORRECT?

21 A YES.

22 Q AND YOU BELIEVE THE POTATO STUDIES ARE
23 RELEVANT TO THE ISSUES BEFORE THE COURT, AS BOTH
24 POTATOES, ON THE ONE HAND, AND COFFEE -- AS WELL AS
25 OTHER FOODS -- CONTAIN ACRYLAMIDE; IS THAT CORRECT?

26 A YES.

27 Q ALL RIGHT. NOW, IN YOUR REVIEW OF THE
28 POTATO STUDIES, DO YOU RECALL THE STUDIES EXPRESSLY

1 ADDRESSING THE QUESTION OF COFFEE CONSUMPTION AND THE
2 RISK OF CANCER?

3 A I'M SORRY. I THOUGHT -- I WAS EXPECTING YOU
4 TO ASK ME A QUESTION ABOUT POTATO CONSUMPTION. NOW,
5 WHAT -- ARE YOU ASKING ABOUT COFFEE?

6 I'M SORRY. COULD YOU REPEAT THE QUESTION?

7 Q YES. NOW, IN YOUR REVIEW AND EVALUATION OF
8 THESE POTATO STUDIES, AS YOU'VE DENOMINATED THEM, DO YOU
9 RECALL THAT MANY OF THEM ADDRESSED THE QUESTION OF
10 COFFEE CONSUMPTION AND THE INCREASED OR DECREASED RISK
11 OF CANCER?

12 A SOME OF THEM MAY HAVE EVALUATED THAT. I
13 DON'T RECALL, AS I SIT HERE, WITHOUT LOOKING THROUGH
14 THEM.

15 Q AND BASED UPON YOUR REVIEW, DID YOU MAKE ANY
16 DETERMINATIONS WITH RESPECT TO WHAT THESE POTATO
17 CONSUMPTION STUDIES THAT YOU'VE EVALUATED -- WHAT THEY
18 HAD TO SAY ABOUT COFFEE?

19 A WELL, I -- MY EXHIBIT 229, THAT WAS PRODUCED
20 AT MY DEPOSITION, CONTAINS THE STUDIES THAT I REVIEWED
21 RELATED TO POTATO CONSUMPTION.

22 NOW, FROM THOSE STUDIES THAT WERE EVALUATED,
23 I THEN COMPILED WHAT'S ON THE SLIDES OF SLIDE 77 AND 79,
24 WHICH ARE THE POSITIVE STUDIES OUT OF THAT.

25 AND THE REASON I DID THIS IS BECAUSE THIS
26 WAS -- I WAS -- WHAT I WAS SHOWING WAS THAT THERE ARE A
27 LARGE NUMBER OF POSITIVE STUDIES WHICH DR. BOFFETTA DID
28 NOT INCLUDE IN HIS 2011 REVIEW.

1 I THINK THERE WERE LIKE 17 -- AT LEAST 23
2 STUDIES THAT HE DID NOT -- BUT 17 THAT WERE AVAILABLE
3 THAT HE DID NOT INCLUDE, THAT DEMONSTRATE A
4 STATISTICALLY SIGNIFICANT INCREASE OF A SPECIFIC CANCER.

5 SO THAT WAS THE PURPOSE OF THESE TWO, WAS TO
6 SHOW THE STUDIES THAT DEMONSTRATE A SIGNIFICANT INCREASE
7 OF SPECIFIC CANCERS THAT HE DID NOT INCLUDE IN HIS 2011
8 REVIEW.

9 AND THEN, ALSO, TO SHOW THE ONES THAT WERE
10 PUBLISHED AFTER HIS 2011 REVIEW THAT ALSO DEMONSTRATE A
11 STATISTICALLY SIGNIFICANT INCREASE.

12 Q ALL RIGHT.

13 A SO THIS IS NOT -- I DID NOT, IN THESE TWO
14 SLIDES, PRESENT NEGATIVE STUDIES. THAT WASN'T THE
15 PURPOSE OF THOSE SLIDES.

16 Q ALL RIGHT. WELL, LET'S EVALUATE,
17 ACTUALLY -- I'M MORE INTERESTED IN THE POTATO STUDIES
18 THAT YOU'VE IDENTIFIED FOR WHAT THEY HAVE TO SAY ABOUT
19 COFFEE THAN WHAT THEY HAVE TO SAY ABOUT POTATOES, IN
20 THIS CASE.

21 SO LET'S TAKE A LOOK AT WHAT A NUMBER OF
22 THESE AUTHORS HAVE TO SAY ABOUT COFFEE AND CANCER IN THE
23 POTATO STUDIES THAT YOU HAVE EVALUATED AND USED AS THE
24 BASIS FOR RENDERING YOUR OPINION.

25 AND I WOULD START WITH EXHIBIT 1022, WHICH
26 IS THE FRANCESCHI 1991 CASE-CONTROL STUDY, WHICH
27 PROVIDES A POOLED ANALYSIS OF FOUR EUROPEAN CASE-CONTROL
28 STUDIES.

1 AND DO YOU RECOGNIZE THIS STUDY?

2 A FRANCESCHI '91, YES.

3 Q AND YOU CONSIDERED AND EVALUATED THIS STUDY
4 IN RENDERING YOUR OPINIONS IN THIS CASE?

5 A YES.

6 Q AND TURNING YOUR ATTENTION IN EXHIBIT 1022
7 TO PAGE NO. 2. AND I'M ON THE RIGHT-HAND COLUMN. AND
8 THE AUTHORS CONCLUDE AND OBSERVE:

9 "FINALLY, AN INVERSE ASSOCIATION OF
10 BORDERLINE STATISTICAL SIGNIFICANCE WAS FOUND
11 FOR COFFEE INTAKE."

12 DO YOU SEE THAT?

13 A (REVIEWS DOCUMENT.)

14 YES.

15 (EXHIBIT 1022 MARKED FOR
16 IDENTIFICATION.)

17 Q BY MR. SCHURZ: AND MEANING THAT WHAT THE
18 FRANCESCHI AUTHORS FOUND IN 1991 WAS A REDUCED RISK OF
19 THYROID CANCER ASSOCIATED WITH COFFEE CONSUMPTION;
20 CORRECT?

21 A WELL, IT SAYS A BORDERLINE INVERSE
22 ASSOCIATION. SO --

23 Q RIGHT. SO YOU'VE CITED --

24 A -- IT WASN'T STATISTICALLY SIGNIFICANT, BUT
25 IT WAS A REDUCTION WITH AN INCREASE OF COFFEE
26 CONSUMPTION.

27 Q SO YOU CITED IT FOR ITS FINDINGS THAT IT IS
28 POSITIVE WITH RESPECT TO POTATOES, BUT IN THIS CASE, IT

1 SHOWS AN INVERSE ASSOCIATION OF BORDERLINE STATISTICAL
2 SIGNIFICANCE FOR COFFEE; CORRECT?

3 A YES.

4 Q OKAY. LET'S TAKE A LOOK AT THE NEXT
5 FRANCESCHI STUDY THAT YOU IDENTIFIED, WHICH IS EXHIBIT
6 1023.

7 AND LOOKING AT EXHIBIT 1023, DR. INFANTE,
8 DID YOU CONSIDER THE FRANCESCHI 1997 CASE-CONTROL STUDY
9 REGARDING COLORECTAL CANCER AS PART OF YOUR REVIEW IN
10 THIS CASE?

11 A FOR POTATOES, YES. FOR COFFEE, NO. BECAUSE
12 I STATED DURING MY DIRECT TESTIMONY THAT I ONLY
13 EVALUATED TWO SITES FOR COFFEE IN ADULTS, PLUS THE
14 CHILDHOOD LEUKEMIA.

15 (EXHIBIT 1023 MARKED FOR
16 IDENTIFICATION.)

17 Q BY MR. SCHURZ: AND SO IN REVIEWING THIS
18 STUDY FOR COFFEE, YOU IGNORED THEIR FINDINGS THAT THE
19 AUTHORS MADE WITH RESPECT -- EXCUSE ME.

20 IN REVIEWING THIS STUDY FOR DATA RELATING TO
21 POTATOES, YOU IGNORED THE AUTHORS' FINDINGS WITH RESPECT
22 TO COFFEE; IS THAT CORRECT?

23 A NO, I DIDN'T IGNORE THE AUTHORS' FINDINGS.
24 I DIDN'T REVIEW ANY DATA ON COFFEE CONSUMPTION AND
25 CANCER OTHER THAN FOR BLADDER CANCER, PANCREATIC CANCER,
26 AND CHILDHOOD LEUKEMIA, BECAUSE THERE WAS TOO MUCH
27 LITERATURE TO LOOK AT. I TRIED TO FOCUS AND ORGANIZE MY
28 REVIEW. AND I STATED THE BASIS FOR MY SELECTION OF

1 THOSE SITES.

2 SO I MEAN, YOU CAN POINT OUT SOME THAT MIGHT
3 NOT SHOW AN INCREASED RISK OF SOME OF THESE OTHER
4 CANCERS, AND I WOULDN'T -- I'M NOT ARGUING WITH THAT,
5 BECAUSE I DIDN'T REVIEW ALL OF THE DATA AT ALL FOR
6 COLORECTAL CANCER OR OTHER CANCERS, OTHER THAN THE TWO
7 THAT I MENTIONED.

8 Q LET'S -- IF I COULD DIRECT YOUR ATTENTION TO
9 THE FINDINGS OF THE FRANCESCHI 1997 WITH RESPECT TO
10 COFFEE, IF YOU TAKE A LOOK AT TABLE NO. 2, WHICH APPEARS
11 ON PAGE 003 OF EXHIBIT 1023, THE AUTHORS REPORT THE
12 RISKS ASSOCIATED WITH COFFEE AND TEA AND COLORECTAL
13 CANCER; CORRECT?

14 A YES.

15 Q AND HERE, AS IT'S INDICATED, WHAT THEY WILL
16 SHOW IS A REDUCED RISK THAT IS STATISTICALLY SIGNIFICANT
17 AT THE HIGHEST CONSUMPTION LEVELS; CORRECT?

18 A AT THE HIGHEST CONSUMPTION LEVEL, YES.

19 Q AND WHAT THE AUTHORS FURTHER OBSERVE ON PAGE
20 004 --

21 A WHICH TABLE?

22 Q IT'S NOT A TABLE. IT'S IN THE TEXT OF
23 EXHIBIT 1023. I'M IN THE LEFT-HAND COLUMN. THE AUTHORS
24 OBSERVE:

25 "WITH RESPECT TO HOT BEVERAGES, COFFEE,
26 BUT NOT TEA, WAS INVERSELY RELATED TO CANCER
27 OF THE COLON."

28 AND WHEN THEY SAY "INVERSELY," THEY MEAN IT

1 SHOWS A REDUCED RISK; CORRECT?

2 A THAT'S WHAT THEY SAY, YES.

3 Q AND THEY FURTHER GO ON TO EXPLAIN, DO THEY
4 NOT, ON PAGE 005 OF EXHIBIT 1023, THAT:

5 "IN SEVERAL STUDIES, SUCH AS THE PRESENT
6 ONE, HIGH COFFEE CONSUMPTION HAS BEEN
7 ASSOCIATED WITH ABOUT 30 PERCENT DECREASES OF
8 CANCER OF THE COLON."

9 DO YOU SEE THAT?

10 A YES.

11 Q OKAY. NOW, YOU ALSO CITED THE HU 1998
12 CASE-CONTROL STUDY AT EXHIBIT 1234.

13 A WHAT WAS THE NAME, PLEASE?

14 Q HU, H-U.

15 AND YOU IDENTIFIED THIS STUDY AS SHOWING A
16 STATISTICALLY SIGNIFICANT INCREASE IN LUNG CANCER, WITH
17 A DOSE RESPONSE, FOR POTATOES; CORRECT?

18 A YES.

19 (EXHIBIT 1234 MARKED FOR
20 IDENTIFICATION.)

21 Q BY MR. SCHURZ: ALL RIGHT. AND YOU
22 CONSIDERED THE HU STUDY, EXHIBIT 1234, IN RENDERING YOUR
23 OPINIONS IN THIS MATTER; CORRECT?

24 A YES. REGARDING POTATOES, YES.

25 Q AND DIRECTING YOUR ATTENTION TO TABLE 3 OF
26 THE HU STUDY, AT PAGE 05, IF YOU LOOK -- HERE THE
27 AUTHORS ARE REPORTING THEIR ODDS RATIOS FOR LUNG CANCER
28 AMONG NONSMOKING WOMEN.

1 AND AT THE BOTTOM WE SEE THE DATA WITH
2 RESPECT TO COFFEE. AND HERE WHAT THE AUTHORS REPORT IS
3 A REDUCED RISK OF LUNG CANCER IN NONSMOKING WOMEN WHO
4 ARE CONSUMERS OF COFFEE, WITH A -- NOT STATISTICALLY
5 SIGNIFICANT PROTECTIVE EFFECTS, BUT A REDUCED RISK.

6 WOULD YOU AGREE?

7 A YES. I WOULDN'T SAY A "PROTECTIVE EFFECT,"
8 BUT THERE'S A REDUCED RISK, IF THE ODDS RATIO IS BELOW
9 1.

10 Q RIGHT. OKAY.

11 LET'S TAKE A LOOK AT ANOTHER ONE OF THE
12 STUDIES THAT YOU'VE IDENTIFIED RELATING TO POTATOES.
13 AND THIS WOULD BE THE LUCENTEFORTE ARTICLE AT EXHIBIT
14 1471.

15 AND DO YOU RECOGNIZE THE LUCENTEFORTE
16 ARTICLE?

17 A YES. YES, I DO.

18 (EXHIBIT 1471 MARKED FOR
19 IDENTIFICATION.)

20 Q BY MR. SCHURZ: AND DID YOU CONSIDER THIS IN
21 DEVELOPING YOUR OPINIONS IN THIS MATTER?

22 A REGARDING POTATOES, YES.

23 Q AND DIRECTING YOUR ATTENTION TO PAGE 03 OF
24 EXHIBIT 1471, AT THE VERY BOTTOM OF THE PAGE ON THE
25 RIGHT-HAND SIDE, DO YOU SEE THE CONCLUSION THAT THE
26 AUTHORS OFFER, WHERE THEY STATE:

27 "OUR STUDY CONFIRMS THAT COFFEE AND
28 BLACK TEA ARE UNRELATED TO STOMACH CANCER

1 RISK"?

2 A YES, I DO.

3 Q OKAY. DO YOU FURTHER IDENTIFY THE BRAVI
4 ARTICLE, WHICH IS EXHIBIT 723 -- AGAIN, ON YOUR CHART
5 RELATED TO POTATOES; AND IN THIS CASE, ENDOMETRIAL
6 CANCER?

7 AND ONCE AGAIN, YOU INDICATE THAT THIS
8 STUDY, THE BRAVI STUDY, EXHIBIT 723, INDICATED AN
9 INCREASED RISK OF ENDOMETRIAL CANCER, WITH BORDERLINE
10 STATISTICAL SIGNIFICANCE; CORRECT?

11 A YES.

12 (EXHIBIT 723 MARKED FOR IDENTIFICATION.)

13 Q BY MR. SCHURZ: AND THE AUTHORS IN BRAVI
14 ALSO PROVIDE INFORMATION WITH RESPECT TO COFFEE
15 CONSUMPTION, DO THEY NOT?

16 A THEY MAY. I DON'T KNOW. I DIDN'T -- AS I
17 SAID, I DIDN'T EVALUATE COFFEE EXCEPT FOR THE TWO SITES
18 THAT I MENTIONED EARLIER, PLUS THE CHILDHOOD LEUKEMIA.
19 SO IT MAY.

20 Q SO LET'S TAKE A LOOK AT WHAT THE BRAVI
21 AUTHORS FOUND WITH RESPECT TO COFFEE. AND I DIRECT YOUR
22 ATTENTION TO PAGE 004.

23 A WHAT TABLE IS THAT?

24 Q AND DO YOU HAVE THE FULL PAGE TABLE IN FRONT
25 OF YOU? IT'S EXHIBIT 723-004.

26 A YES, I DO.

27 Q ALL RIGHT. NOW, AND HERE THE BRAVI AUTHORS
28 PROVIDE THEIR DATA WITH RESPECT TO COFFEE CONSUMPTION IN

1 A SERIES OF QUINTILES; CORRECT?

2 A YES.

3 Q AND THEN FOR EACH OF THESE, THEY PROVIDE A
4 RESPONSE WITH RESPECT TO EACH INDIVIDUAL QUINTILE, AND
5 THE RELATIVE -- EXCUSE ME, THE ODDS RATIO AND THE
6 CONFIDENCE INTERVAL FOR EACH OF THOSE EXPOSURE LEVELS;
7 CORRECT?

8 A YES.

9 Q AND WHAT THEY FOUND, WHAT THE BRAVI
10 INVESTIGATORS FOUND, WAS A REDUCED RISK OF ENDOMETRIAL
11 CANCER AMONG COFFEE CONSUMERS, WITH A REDUCED RISK OF 50
12 PERCENT AMONG THE HIGHEST CONSUMPTION CATEGORY; CORRECT?

13 A YES.

14 Q AND IN THAT HIGHEST CONSUMPTION CATEGORY,
15 THE 50 PERCENT REDUCED RISK IS STATISTICALLY
16 SIGNIFICANT; CORRECT?

17 A YES.

18 Q AND THE AUTHORS FURTHER FOUND THAT THIS DOSE
19 RESPONSE TREND WAS STATISTICALLY SIGNIFICANT; CORRECT?

20 A YES.

21 Q AND YOU TESTIFIED IN YOUR CONVERSATION WITH
22 MR. METZGER THAT DOSE RESPONSE IS A PARTICULARLY
23 MEANINGFUL MEASURE OF A SUBSTANCE'S INFLUENCE; CORRECT?

24 A YES.

25 Q I'D LIKE TO TAKE YOU NOW TO EXHIBIT 877.
26 AND THIS IS A RECENT CASE-CONTROL STUDY BY EDUARDO DE
27 STEFANI, AND THE CO-AUTHORS INCLUDE DR. PAOLO BOFFETTA.
28 AND THIS WAS ONE OF THE STUDIES YOU IDENTIFIED, ONE OF

1 THE RECENT STUDIES THAT YOU INCLUDED IN YOUR CHARTS; IS
2 THAT CORRECT?

3 A YES. THAT HE DID NOT HAVE IN HIS REVIEW;
4 CORRECT.

5 (EXHIBIT 877 MARKED FOR IDENTIFICATION.)

6 Q BY MR. SCHURZ: YEAH. HIS REVIEW, WHICH WAS
7 PUBLISHED IN 2011, DID NOT INCLUDE A PAPER THAT WAS
8 PUBLISHED IN 2014; CORRECT?

9 A NOW, WAIT A MINUTE. WHICH -- I'M SORRY. I
10 HAVE THE WRONG ONE.

11 THIS IS ON ESOPHAGEAL -- YEAH, WELL, IT WAS
12 IN MY CHART, BUT IT WAS INDICATED AS ONE THAT WAS
13 PUBLISHED AFTER HIS REVIEW.

14 Q RIGHT. AND YOU'VE INDICATED AND CITED THIS,
15 AGAIN, FOR THE PROPOSITION THAT POTATO CONSUMPTION SHOWS
16 A STATISTICALLY SIGNIFICANT INCREASED RISK FOR
17 ESOPHAGEAL CANCER; CORRECT?

18 A YES. AND A DOSE RESPONSE; CORRECT.

19 Q RIGHT. AND THE AUTHORS, DRs. DE STEFANI AND
20 BOFFETTA AND OTHERS, ALSO REPORT WITH RESPECT TO COFFEE,
21 DO THEY NOT?

22 A I'LL HAVE TO LOOK TO SEE. LIKE I SAID, I
23 DIDN'T REVIEW THESE FOR COFFEE BECAUSE I WASN'T DOING
24 THAT SITE.

25 Q SO I'LL DIRECT YOUR ATTENTION TO PAGE 3 OF
26 877, IN WHICH THE AUTHORS CONCLUDE -- AND IF YOU TAKE A
27 LOOK, IT'S A CARRYOVER FROM THE LEFT-HAND SIDE TO THE
28 RIGHT-HAND-SIDE COLUMNS. YOU CAN SEE THE AUTHORS

1 REPORT:

2 "COFFEE WAS INVERSELY ASSOCIATED WITH
3 SQUAMOUS CELL ESOPHAGEAL CANCER."

4 DO YOU SEE THAT?

5 A YES.

6 Q AND THEY REPORT AN ODDS RATIO OF 0.61, WITH
7 A CONFIDENCE INTERVAL OF 0.88 TO 0.98; CORRECT?

8 A YES.

9 Q SO ONCE AGAIN, IN THIS STUDY, IN THE DE
10 STEFANI STUDY, THE AUTHORS REPORT A STATISTICALLY
11 SIGNIFICANT REDUCED RISK ASSOCIATED WITH CONSUMPTION OF
12 COFFEE; CORRECT?

13 A IN THIS STUDY, YES.

14 Q OKAY. SO ONE LAST ONE, JUST BY WAY OF
15 EXAMPLE, AND THEN WE CAN FURTHER DISCUSS THIS.

16 THE ISO 2007, WHICH IS AT EXHIBIT 1270, YOU
17 ALSO INCLUDED IN YOUR MATERIALS OF STUDIES PUBLISHED
18 AFTER -- STRIKE THAT -- OF STUDIES CONTAINING
19 INFORMATION RELATING TO POTATO CONSUMPTION?

20 A CORRECT. YES.

21 (EXHIBIT 1270 MARKED FOR
22 IDENTIFICATION.)

23 Q BY MR. SCHURZ: AND YOU CONSIDERED THIS
24 STUDY, DID YOU NOT, IN THE CONTEXT OF THE DATA PROVIDED
25 FOR POTATO CONSUMPTION; CORRECT?

26 A YES.

27 Q AND SPECIFICALLY, FOR COLON CANCER; CORRECT?

28 A YES.

1 Q AND THE ISO INVESTIGATORS ALSO PROVIDED
2 EXTENSIVE INFORMATION, DID THEY NOT, WITH RESPECT TO
3 HAZARD RATIOS FOR COFFEE CONSUMPTION, AS WELL AS OTHER
4 CAFFEINATED BEVERAGES, DID THEY NOT?

5 A WELL, YOU'D HAVE TO POINT IT OUT TO ME.
6 BECAUSE, AS I SAID, I DIDN'T REVIEW THESE -- THESE SITES
7 WERE NOT ON MY SCHEME FOR -- IN RELATION TO COFFEE
8 CONSUMPTION. SO IF YOU WANT TO POINT OUT WHERE IN THE
9 STUDY, THEN I'LL ANSWER YOUR QUESTIONS.

10 Q SURE. IF YOU TAKE A LOOK AT PAGE 43 OF
11 EXHIBIT 1270. AND WE'VE HIGHLIGHTED ON THE MONITOR THE
12 SPECIFIC DATA SETS RELATING TO COFFEE THAT ARE CONTAINED
13 WITHIN TABLE 14 OF EXHIBIT 1270.

14 A OKAY. I'M THERE.

15 Q AND HERE THE AUTHORS PROVIDE INFORMATION
16 WITH RESPECT TO ALL CANCERS, AS WELL AS A TOTAL OF FIVE
17 DIFFERENT INDIVIDUAL CANCERS. DO YOU SEE THAT?

18 A YES.

19 Q AND WITH RESPECT TO -- AND LET'S START WITH
20 THE MEN, WHICH APPEARS AT THE TOP OF THE TABLE 14 OF
21 EXHIBIT 1270. AND THEY PROVIDE FOR ALL CANCERS THERE,
22 IN WHAT IS THE FIFTH COLUMN OVER -- THEY PROVIDE THE
23 DATA AT VARIOUS EXPOSURE INTERVALS FOR ALL CANCERS.

24 DO YOU SEE THAT?

25 A YES.

26 Q AND WHAT THEY SHOW IS A REDUCED RISK OF ALL
27 CANCERS THAT IS STATISTICALLY SIGNIFICANT AT THE HIGHEST
28 LEVEL; CORRECT?

1 A YES.

2 Q ALL RIGHT. AND THEN WITH RESPECT TO THE
3 INDIVIDUAL CANCER SITES, THEY FIND STATISTICALLY
4 SIGNIFICANT REDUCED RISKS FOR ESOPHAGEAL CANCER, COLON
5 CANCER, AND LIVER CANCER; IS THAT CORRECT?

6 A LET ME LOOK AT THE DATA. FOR ESOPHAGEAL
7 CANCER, YES. THE COLON CANCER --

8 Q OH -- SO STRIKE THAT.

9 A I WOULD SAY --

10 Q FOR ESOPHAGEAL CANCER, LIVER CANCER, AND --
11 MR. METZGER: LET HIM ANSWER THE QUESTION.

12 THE COURT: WE CAN'T HAVE TWO PEOPLE TALKING AT
13 THE SAME TIME.

14 DID YOU COMPLETE YOUR ANSWER?

15 THE WITNESS: NO, I DIDN'T.

16 THE COURT: ALL RIGHT. PLEASE COMPLETE YOUR
17 ANSWER.

18 THE WITNESS: FOR COLON CANCER, NO. AND FOR LIVER
19 CANCER, THERE'S A DECLINED RISK, YES.

20 Q BY MR. SCHURZ: THAT ACHIEVES STATISTICAL
21 SIGNIFICANCE AT THE HIGHEST LEVEL, AS WELL AS THE SECOND
22 HIGHEST LEVEL; CORRECT? IN FACT, AT ALL THREE LEVELS.
23 THEY'RE ALL STATISTICALLY SIGNIFICANT; CORRECT?

24 A YES.

25 Q ALL RIGHT. AND IF WE LOOK DOWN AT THE
26 WOMEN, ONCE AGAIN, AT THE "ALL CANCERS," WE SEE A
27 REDUCED RISK THAT IS NOT STATISTICALLY SIGNIFICANT;
28 CORRECT?

1 A CORRECT.

2 Q AND FOR ESOPHAGEAL CANCER, WE FIND A REDUCED
3 RISK OF SOME 82 PERCENT, IT SAYS, AND IT'S AN ODDS RATIO
4 OF 0.17. SO A SUBSTANTIAL DECREASED RISK, BUT IT IS NOT
5 STATISTICALLY SIGNIFICANT; CORRECT?

6 A I'M NOT SEEING THAT ON WHAT -- I CAN'T SEE
7 THAT ON THE MONITOR. I'M SORRY.

8 Q IF YOU TAKE A LOOK, WE'RE FOCUSING ON --
9 THERE IT IS.

10 A ALL RIGHT. I MEAN, IS THAT BASED ON ONE
11 CASE? ARE YOU LOOKING AT THE BOTTOM ENTRY? WITHOUT
12 SEEING THE REST OF IT, I CAN'T READ THE --

13 NOW THAT I KNOW WHERE YOU ARE -- OKAY.
14 ESOPHAGEAL CANCER. NOW I SEE IT. YES.

15 Q ALL RIGHT. AND THE POINT IS, DR. INFANTE,
16 WHAT THE ISO STUDY SHOWS WITH RESPECT TO COFFEE ARE A
17 SERIES OF REDUCED RISKS -- SOME THAT ARE STATISTICALLY
18 SIGNIFICANT, AND IN OTHER CASES, DO NOT ACHIEVE
19 STATISTICAL SIGNIFICANCE -- FOR ALL CANCERS, AS
20 EVALUATED BY THE ISO INVESTIGATORS; CORRECT?

21 A WELL, WHEN YOU EVALUATE THE DATA, YOU SEE A
22 REDUCED RISK OR AN INCREASED RISK FOR ALL CANCERS. THEN
23 THE NEXT THING YOU WOULD LOOK FOR IS TO -- WELL, WHICH
24 CANCERS IS THAT A REFLECTION OF?

25 SO YOU WOULDN'T CONCLUDE THAT A PARTICULAR
26 PRODUCT OR EXPOSURE CAUSES AN INCREASE IN ALL CANCERS OR
27 CAUSES A DECREASE IN ALL CANCERS. THAT ONLY TELLS YOU
28 WHERE TO LOOK, THEN, TO SEE WHICH CANCERS THAT'S A

1 REFLECTION OF.

2 JUST SO YOU WOULDN'T HAVE THE WRONG
3 INTERPRETATION: "YEAH, THIS REDUCES ALL KINDS OF
4 CANCER." IT'S NOT THE CASE. SO WHEN YOU -- THEN YOU
5 LOOK TO SPECIFIC CANCERS, TO SEE WHICH CANCERS THAT'S A
6 REFLECTION OF.

7 Q AND IN THIS CASE, WHAT THE ISO INVESTIGATORS
8 FOUND WAS THE ABSENCE OF ANY INCREASED RISK ASSOCIATED
9 WITH COFFEE INTAKE. AND IN A NUMBER OF CASES, THEY
10 FOUND STATISTICALLY SIGNIFICANT REDUCED RISKS, AND IN
11 OTHER CASES, DECREASED RISKS THAT DID NOT ACHIEVE
12 STATISTICAL SIGNIFICANCE; CORRECT?

13 A WELL, IN GENERAL, GLOSSING OVER EVERYTHING,
14 YES.

15 Q ALL RIGHT. NOW, NONE OF THE 28 STUDIES THAT
16 YOU REVIEWED AND THAT YOU RELIED ON FOR YOUR OPINIONS
17 RELATING TO THE POTATO CONSUMPTION SHOWED ANY INCREASED
18 RISK OF CANCER FROM COFFEE CONSUMPTION, DID THEY?

19 A WELL, I DON'T KNOW BECAUSE I DIDN'T
20 REVIEW -- I DIDN'T REVIEW THEM FOR THAT. YOU'VE POINTED
21 CERTAIN ONES OUT TO ME. I DON'T KNOW WHAT THE OTHER
22 ONES SHOW, BECAUSE THAT WASN'T WHAT I DID FOR THIS CASE.

23 Q NOW, YOU FOUND THESE STUDIES SUFFICIENT, DID
24 YOU NOT, AS A BASIS FOR YOUR OPINION THAT POTATO
25 CONSUMPTION INCREASES THE RISKS FOR MANY CANCERS; IS
26 THAT CORRECT?

27 A I'M SAYING THAT THE STUDIES RELATED TO
28 POTATO SHOWED SIGNIFICANTLY INCREASED RISKS FOR THE

1 SITES THAT I HAVE ON THE CHART, BASED ON THAT STUDY. I
2 DIDN'T CONCLUDE THAT POTATO CONSUMPTION CAUSES --
3 CAUSES -- AN INCREASED RISK OF ALL THESE CANCERS.

4 I'M POINTING OUT THAT IN THOSE STUDIES THAT
5 WERE MISSED BY BOFFETTA -- SINCE HE WAS GOING THROUGH
6 THE LITERATURE AND POINTING OUT POTATO STUDIES, AND
7 WHICH ONES SHOWED AN INCREASED RISK, BY CANCER SITE, AND
8 WHICH ONES DIDN'T -- I'M SAYING, "HEY, HE MISSED LIKE 25
9 STUDIES HERE," FOLLOWING HIS OWN METHODOLOGY.

10 I'M NOT PRONOUNCING AT ALL THAT POTATOES
11 CAUSES AN INCREASED RISK OF ALL THOSE CANCERS.

12 Q OKAY. DR. INFANTE, YOU TESTIFIED ON MONDAY
13 THAT THE CASE-CONTROL STUDIES OF CANCER AND POTATO
14 CONSUMPTION PROVIDE SUPPORTIVE EVIDENCE FOR THE HUMAN
15 CARCINOGENICITY OF ACRYLAMIDE; CORRECT?

16 A CORRECT.

17 Q SO -- AND YOU FOUND THESE STUDIES THAT WE'VE
18 BEEN DISCUSSING SUFFICIENT FOR MAKING A DETERMINATION
19 THAT THESE POTATO CONSUMPTION STUDIES ARE SUPPORTIVE
20 EVIDENCE FOR THE HUMAN CARCINOGENICITY OF ACRYLAMIDE;
21 CORRECT?

22 A THAT'S CORRECT. THERE'S SOME EVIDENCE
23 RELATED TO THOSE SITES AND POTATO CONSUMPTION. THAT
24 DOES NOT MEAN THAT IT'S CONCLUSIVE THAT POTATO
25 CONSUMPTION CAUSES ALL THOSE CANCERS. WHAT I'M SAYING
26 IS, THERE'S SOME EVIDENCE.

27 Q AND WOULD YOU --

28 A IT CERTAINLY DOES NOT INDICATE A LACK OF

1 EVIDENCE.

2 Q I UNDERSTAND.

3 A OKAY.

4 Q AND WOULD YOU ALSO AGREE, DR. INFANTE, THAT
5 THESE STUDIES THAT YOU EVALUATED, THAT YOU FOUND
6 SUFFICIENT FOR FINDING THEM AS EVIDENCE OF THE HUMAN
7 CARCINOGENICITY OF ACRYLAMIDE IN POTATOES, THAT THEY ARE
8 ALSO SUFFICIENT TO SHOW THAT COFFEE DOES NOT INCREASE
9 THE RISK OF CANCER, AS BASED ON THESE SAME STUDIES?

10 A I DIDN'T EVALUATE COFFEE IN THOSE STUDIES
11 BECAUSE I ONLY LOOKED AT TWO SITES, AS I'VE REPEATED
12 SEVERAL TIMES, IN RELATION TO COFFEE.

13 Q WOULD YOU AGREE, BASED ON OUR REVIEW OF THE
14 GROUP OF STUDIES THAT WE'VE JUST NOW DISCUSSED, THAT THE
15 STUDIES THAT YOU'VE CITED -- THAT WHILE THEY INDICATE
16 THAT THEY -- INDICATE OR PROVIDE EVIDENCE THAT POTATOES
17 MAY INCREASE THE RISK OF CANCER, THEY ALSO STAND FOR THE
18 PROPOSITION THAT COFFEE DECREASES THE RISK OF CANCER, AT
19 LEAST AT THE SITES THAT WE'VE BEEN TALKING ABOUT?

20 A I WOULD SAY IN THE STUDIES THAT YOU POINTED
21 OUT TO ME -- IN THOSE STUDIES, AS WE WENT THROUGH THE
22 TABLES, THE SITES THAT YOU POINTED OUT TO ME IN RELATION
23 TO COFFEE -- THAT IN THOSE STUDIES, THE ONES THAT SHOWED
24 REDUCED RISKS, THAT'S WHAT THEY SHOWED. YEAH, I'M NOT
25 ARGUING THAT.

26 Q AND DOES THAT SUGGEST TO YOU, DR. INFANTE,
27 THAT IN EVALUATING A PARTICULAR SUBSTANCE -- IN THIS
28 CASE, COFFEE -- IN THE CONTEXT OF A HEALTH ASSESSMENT,

1 OR WHETHER IT'S COFFEE OR VEGETABLES, THAT IT'S
2 IMPORTANT TO LOOK AT THE FOOD ITSELF, AS OPPOSED TO AN
3 INDIVIDUAL COMPONENT, IN EVALUATING ITS CARCINOGENICITY?

4 A I THINK YOU WOULD DO BOTH.

5 Q OKAY. I'M FINISHED WITH THE POTATO STUDIES.
6 YOU CAN PUT THOSE ASIDE. THANK YOU.

7 NOW, DR. INFANTE, YOU'VE BEEN DEPOSED IN
8 SOME 70 OR 80 CASES IN YOUR CAREER; IS THAT CORRECT?

9 A YOU KNOW, I DON'T KNOW. I HAVEN'T COUNTED
10 THEM. IT'S POSSIBLE. AND I DON'T KNOW IF I'VE DONE
11 THAT MANY MAYBE -- I DON'T KNOW, BUT I'VE BEEN DEPOSED
12 QUITE A NUMBER OF TIMES.

13 Q WOULD YOU LIKE TO GIVE US AN ESTIMATE OF HOW
14 MANY TIMES?

15 A WELL, I DON'T REALLY KNOW. DID I PROVIDE --
16 DID I PROVIDE YOU WITH A LIST FOR THE LAST FOUR YEARS,
17 OR SOMETHING? THAT WOULD INDICATE FOR THE LAST FOUR
18 YEARS.

19 I WAS JUST SAYING, AS I'M SITTING HERE, I
20 CAN'T REMEMBER HOW MANY. BUT THAT WOULD BE THE BEST --
21 THAT WOULD BE THE BEST INDICATOR, WHAT I'VE DONE IN THE
22 LAST FOUR YEARS.

23 Q RIGHT.

24 A AND THAT LIST, I HAVE.

25 Q OKAY.

26 A I THINK I PROVIDED IT.

27 Q DO YOU RECALL AT YOUR DEPOSITION I ASKED YOU
28 IF YOU'VE BEEN DEPOSED ROUGHLY 70 TO 80 TIMES, AND YOU

1 SAID "YES"?

2 A YOU KNOW, I DON'T RECALL THAT; BUT IF THAT
3 WAS THE NUMBERS, WELL, THAT'S POSSIBLE. I'M JUST SAYING
4 I DON'T RECALL WHAT IT IS RIGHT NOW. BUT I WOULDN'T
5 ARGUE THAT.

6 Q OKAY. AND WITH THE EXCEPTION OF YOUR
7 INVOLVEMENT IN A BANKRUPTCY PROCEEDING IN 2009, YOU'VE
8 TESTIFIED ON BEHALF OF THE PLAINTIFF IN EVERY SINGLE ONE
9 OF THOSE 70 TO 80 DEPOSITIONS; CORRECT?

10 A YES. AND I HAVE NEVER BEEN ASKED BY
11 DEFENDANTS TO TESTIFY. I HAVE DONE OTHER RESEARCH FOR
12 DEFENDANTS, THAT THEN I'VE GIVEN THEM EVALUATIONS, BUT
13 THEY'VE NEVER ASKED ME TO TESTIFY.

14 MR. SCHURZ: THANK YOU, DR. INFANTE.

15 THANK YOU, YOUR HONOR.

16 THE COURT: ANY REDIRECT?

17 MR. METZGER: OH, YES, YOUR HONOR.

18 THE COURT: HOW LONG?

19 MR. METZGER: IT'S NOT GOING TO BE VERY LONG.

20 THE COURT: HOW LONG IS "VERY LONG"?

21 MR. METZGER: NO MORE THAN A HALF HOUR, BUT
22 PROBABLY LESS.

23 THE COURT: WE DO NOT HAVE TO REVIEW ANY ARTICLES,
24 AND WE DO NOT HAVE TO REPLAY DIRECT TESTIMONY.

25 MR. METZGER: YES, I UNDERSTAND, YOUR HONOR.

26 THE COURT: ALL RIGHT. LET'S GO FORWARD.

27

28

REDIRECT EXAMINATION

1
2 BY MR. METZGER:

3 Q DR. INFANTE, YESTERDAY MR. SCHURZ SHOWED YOU
4 THE CONCLUSIONS FROM ARTICLES THAT YOU HAD REVIEWED. DO
5 YOU RECALL THAT?

6 A YES.

7 Q NOW, WHEN YOU, AS AN EPIDEMIOLOGIST, ASSESS
8 A RISK OF CANCER BASED UPON EPIDEMIOLOGY STUDIES, DO YOU
9 EVALUATE THE AUTHORS' CONCLUSIONS?

10 A NO.

11 Q WHY DO YOU NOT EVALUATE THE AUTHORS'
12 CONCLUSIONS?

13 A WELL, YOU DO THE EVALUATION OF THE DATA.
14 THAT'S WHAT YOU'RE ASKED TO DO. AND I'VE BEEN ON IARC
15 WORKING GROUPS A NUMBER OF TIMES, AND YOU EVALUATE THE
16 DATA IN THE STUDIES. YOU DON'T EVALUATE THE OPINIONS
17 FROM THOSE STUDIES.

18 Q WHY DO YOU EVALUATE THE DATA AND NOT THE
19 CONCLUSIONS?

20 A BECAUSE THAT'S WHAT YOU'RE ASKED TO DO.
21 THAT'S WHY YOU'RE CALLED THERE AS AN EXPERT GROUP, TO
22 EVALUATE THE DATA AND PROVIDE THAT EVALUATION TO THE
23 WORLD HEALTH ORGANIZATION.

24 YOU DON'T LOOK AT OPINIONS BECAUSE THOSE --
25 YOU'RE NOT REVIEWING OPINIONS; YOU'RE REVIEWING THE
26 DATA. AND THEN YOU'RE DRAWING YOUR OWN -- YOU'RE
27 DRAWING -- THE WORKING GROUP IS DRAWING THE CONCLUSIONS
28 FROM THAT EVALUATION OF THE DATA.

1 BECAUSE OPINIONS -- OR RATHER, CONCLUSIONS
2 FROM STUDIES CAN BE AFFECTED BY THINGS SUCH AS, WELL,
3 WHO IS FUNDING THE STUDIES, FOR EXAMPLE. AND THAT'S NOT
4 OF INTEREST TO IARC OR TO ANYONE THAT'S REVIEWING THE
5 DATA.

6 Q CAN YOU GIVE US AN EXAMPLE OF WHAT YOU MEAN
7 THAT THE CONCLUSION CAN BE AFFECTED BY THE FUNDING OF
8 THE STUDY.

9 A WELL, YEAH. THERE WAS ACTUALLY -- THERE WAS
10 AN ARTICLE IN THE "BRITISH MEDICAL JOURNAL," I THINK, IN
11 FEBRUARY OF THIS YEAR, HAVING TO DO WITH TESTOSTERONE
12 ADMINISTRATION AND RISK OF HEART ATTACKS.

13 AND WHAT WAS REPORTED WAS THAT 17
14 INTERVENTION -- NO, IT WAS 14 INTERVENTION STUDIES BY
15 THE INDUSTRY, WHERE THEY DID CLINICAL TRIALS, SHOWED NO
16 ADVERSE HEALTH EFFECTS.

17 BUT 13 NON-INDUSTRY-FUNDED STUDIES, WHEN YOU
18 PUT THOSE -- A META-ANALYSIS TOGETHER, THERE WAS A
19 TWOFOLD RISK OF HEART DISEASE.

20 AND THE GIST OF THE COMMENTARY WAS THAT
21 PHYSICIANS ARE STILL PRESCRIBING TESTOSTERONE, YET IT
22 CARRIES A TWOFOLD RISK OF HEART DISEASE, WHICH WOULD BE
23 A HIGH RISK IN THE GENERAL POPULATION.

24 SO THAT'S ONE EXAMPLE, OFF THE TOP OF MY
25 HEAD, OF THE INFLUENCE OF WHO FUNDS STUDIES ON THE
26 RESULTS OF THE STUDIES AND INTERPRETATION OF THE
27 STUDIES.

28 Q ALL RIGHT. NOW, REGARDING THE ACRYLAMIDE

1 OCCUPATIONAL EPIDEMIOLOGY STUDIES OF THE ACRYLAMIDE
2 PRODUCTION WORKERS, WHO FUNDED THOSE STUDIES?

3 A WELL, THOSE WERE INDUSTRY-FUNDED STUDIES.

4 Q WERE THOSE STUDIES ACTUALLY DONE BY THE
5 INDUSTRY ITSELF, BY EMPLOYEES OF THE ACRYLAMIDE
6 PRODUCTION INDUSTRY?

7 A THEY WERE DONE BY THE PEOPLE THAT THEY HAD
8 HIRED TO DO THOSE STUDIES.

9 Q OKAY. AND WERE SOME OF THE AUTHORS OF THOSE
10 STUDIES FROM DOW CHEMICAL COMPANY, THE ACRYLAMIDE
11 MANUFACTURER?

12 A WELL, THEY FUNDED -- THEY FUNDED ONE OF THE
13 GROUPS OF STUDIES.

14 Q OKAY. NOW, DR. INFANTE --

15 A AND THE OTHER STUDY WAS FUNDED BY SYNGENTA,
16 OR SOMETHING. I FORGET THE NAME OF THE COMPANY THAT
17 FUNDED IT. BUT IT WAS THEIR EMPLOYEES.

18 Q ALL RIGHT. NOW, WHEN YOU DID YOUR ANALYSIS,
19 YOU DID AN ANALYSIS INCLUDING AN ADJUSTMENT FOR THE
20 "HEALTHY WORKER EFFECT," DIDN'T YOU?

21 A YES, I DID.

22 Q ALL RIGHT. DID ANY -- AND WHAT IS THE
23 EFFECT OF REPORTING DATA AND CONCLUSIONS OF AN
24 OCCUPATIONAL COHORT STUDY WITHOUT MAKING AN ADJUSTMENT
25 FOR THE "HEALTHY WORKER EFFECT"?

26 MR. SCHURZ: OBJECTION; ASKED AND ANSWERED.

27 THE WITNESS: WELL, IF YOU SEE A "HEALTHY WORKER
28 EFFECT" IN THE STUDY --

1 THE COURT: OVERRULED.

2 THE WITNESS: -- THEN YOU SHOULD ADJUST FOR IT.
3 OTHERWISE, YOU'RE GOING TO UNDERESTIMATE THE RISKS OF
4 WHATEVER YOU'RE EVALUATING IN THAT STUDY.

5 Q BY MR. METZGER: DR. INFANTE, HAVE YOU
6 REVIEWED MANY OCCUPATIONAL COHORT STUDIES THROUGHOUT
7 YOUR CAREER?

8 A YES, I HAVE.

9 Q IN ANY STUDY THAT WAS EITHER CONDUCTED OR
10 FUNDED BY THE INDUSTRY PRODUCING THE CHEMICAL, DID ANY
11 OF THE AUTHORS EVER, IN ANY ARTICLE THAT YOU READ, DO
12 THE ADJUSTMENT FOR THE "HEALTHY WORKER EFFECT"?

13 MR. SCHURZ: OBJECTION; OVERBROAD, LACKS
14 FOUNDATION.

15 THE COURT: OVERRULED.

16 THE WITNESS: NOT THAT I'M AWARE OF, NO.

17 Q BY MR. METZGER: OKAY. ALL RIGHT.

18 NOW, YOU MENTIONED THE IARC WORKING GROUP.
19 HAVE YOU EVER BEEN ON -- WELL, YOU HAVE BEEN. EXCUSE
20 ME.

21 YOU HAVE SERVED ON IARC WORKING GROUP PANELS
22 TO EVALUATE THE CARCINOGENIC RISKS OF CHEMICALS TO
23 HUMANS?

24 A YES.

25 MR. SCHURZ: OBJECTION; OVERBROAD, OUTSIDE THE
26 SCOPE. THIS HAS NOTHING TO DO WITH THE CROSS-
27 EXAMINATION AND REDIRECT.

28 THE COURT: OVERRULED.

1 Q BY MR. METZGER: HAVE YOU EVER, IN
2 PARTICIPATING IN THOSE IARC WORKING GROUPS FOR VARIOUS
3 CHEMICALS, HEARD ANY SCIENTIST WHO WAS APPOINTED TO THE
4 WORKING GROUP EVER EVALUATE THE AUTHORS' CONCLUSIONS OF
5 THE STUDIES?

6 A NO, THAT WOULD --

7 THE COURT: WHAT IS THE PURPOSE --

8 WAIT A MINUTE.

9 WHAT IS THE PURPOSE OF THIS INQUIRY?

10 MR. METZGER: THE PURPOSE OF THIS, YOUR HONOR, IS,
11 WHEN MR. SCHURZ READS CONCLUSIONS FROM STUDIES, THAT
12 THOSE ARE NOT DATA, THOSE ARE NOT SCIENTIFIC, AND THEY
13 ARE SUBJECT TO THE BIAS OF FUNDING AND INTERESTS OF THE
14 AUTHORS AND THE PEOPLE WHO HIRE THEM TO DO THE STUDIES;

15 AND THAT THE -- IN SCIENCE AT THE IARC, AT
16 THE INTERNATIONAL AGENCY FOR RESEARCH ON CANCER, THOSE
17 BODIES WHICH ARE SELECTED NOT TO HAVE BIASED PEOPLE ON
18 THEM, WHEN THEY ANALYZE THE CARCINOGENIC RISKS, THEY DO
19 NOT CONSIDER CONCLUSIONS OF AUTHORS.

20 MR. SCHURZ: I'M GOING TO OBJECT AS IRRELEVANT AND
21 OUTSIDE THE SCOPE.

22 THE COURT: OBJECTION SUSTAINED. IF THE QUESTION
23 IS, "HAVE YOU EVER HEARD OF A SCIENTIST WHO WAS A FUNDED
24 WORKING GROUP EVALUATING THE AUTHORS?" -- I MEAN, WHERE
25 IS THAT GOING?

26 OBJECTION SUSTAINED. LET'S MOVE ON.

27 MR. METZGER: ALL RIGHT. VERY GOOD.

28 Q DR. INFANTE, I'D LIKE TO REVIEW A FEW OF THE

1 ARTICLES THAT MR. SCHURZ WENT OVER WITH YOU --

2 THE COURT: WE'VE BEEN BACK AND FORTH THROUGH ALL
3 THESE ARTICLES.

4 MR. METZGER: PARDON?

5 THE COURT: DO WE HAVE TO REVIEW THESE ARTICLES
6 AGAIN? HAVEN'T WE REVIEWED ENOUGH OF THESE ARTICLES?

7 MR. METZGER: I HAVE LIKE THREE POINTS TO MAKE
8 WITH YOU, YOUR HONOR. IT'S NOT GOING TO TAKE LONG. AND
9 I --

10 THE COURT: HOW MANY ARTICLES?

11 MR. METZGER: THERE ARE JUST THREE.

12 THE COURT: THREE ARTICLES. ALL RIGHT. LET'S GO
13 FORWARD.

14 Q BY MR. METZGER: SO ONE OF THE ARTICLES THAT
15 MR. SCHURZ BROUGHT TO YOUR ATTENTION WAS THE TURATI 2011
16 REVIEW ON THE META-ANALYSIS OF COFFEE CONSUMPTION AND
17 PANCREATIC CANCER, EXHIBIT 2083. HE SPECIFICALLY
18 DIRECTED YOUR ATTENTION TO FIGURE 4.

19 ALL RIGHT. AND THIS WAS THE CUMULATIVE
20 META-ANALYSIS FOR HIGHEST VERSUS LOWEST COFFEE-DRINKING
21 IN CASE-CONTROL AND COHORT STUDIES, WITH SMOKING
22 ADJUSTMENT. AND THIS IS FOR PANCREATIC CANCER. OKAY?

23 NOW, ON THE Y AXIS, IS -- IS THAT THE RISK?

24 A THE VERTICAL AXIS, YES.

25 Q THE VERTICAL AXIS. THAT'S A GOOD WAY TO DO
26 IT.

27 AND WHERE MY FINGER IS POINTING -- 1.0 -- IS
28 THAT THE DEMARCATION FOR RISK THAT, ABOVE THAT, THERE IS

1 RISK; AND BELOW THAT, THERE IS NOT?

2 A YES.

3 Q AND DOES ALL OF THE DATA HERE IN THIS CHART
4 INDICATE THAT ALL THESE STUDIES, FROM ALL THESE TIME
5 PERIODS, SHOW AN INCREASED RISK OF PANCREATIC CANCER?

6 A YES.

7 Q AND THAT'S FROM COFFEE CONSUMPTION; CORRECT?

8 A YES.

9 Q NOW, ANOTHER STUDY THAT MR. SCHURZ SHOWED
10 YOU WAS THE GENKINGER POOLED ANALYSIS OF 14 COHORT
11 STUDIES, EXHIBIT 1072.

12 HE SPECIFICALLY DIRECTED YOUR ATTENTION TO
13 FIGURE 1A, WHICH IS THE MULTIVARIANT ADJUSTED RELATIVE
14 RISKS, AT 95 PERCENT CONFIDENCE INTERVALS, FOR
15 PANCREATIC CANCER ACCORDING TO INTAKE OF COFFEE, HIGH
16 VERSUS LOW.

17 NOW, IN THIS GRAPH, RISK IS REFLECTED ON THE
18 HORIZONTAL AXIS. IS THAT HOW THAT WORKS?

19 A YES.

20 Q ALL RIGHT. AND AGAIN, IS 1 THE DEMARCATION
21 BETWEEN NO RISK AND INCREASED RISK?

22 A YES.

23 Q MR. SCHURZ DIRECTED YOUR ATTENTION TO THE
24 IOWA WOMEN'S STUDY, WHICH WAS THIS ONE HERE THAT HAS A
25 BIG SQUARE ON THE LEFT; IS THAT CORRECT?

26 A YES.

27 Q ALL RIGHT. NOW, LOOKING AT THE BULK OF THE
28 DATA POINTS, DOES THIS BODY OF DATA FOR PANCREATIC

1 CANCER IN RELATION TO COFFEE CONSUMPTION OVERALL SHOW AN
2 INCREASED RISK, A DECREASED RISK, OR NO RISK?

3 A WELL, THERE ARE MORE STUDIES THAT REPORT AN
4 INCREASED RISK THAN A DECREASED RISK, AS YOU CAN SEE BY
5 THE LITTLE SQUARES BEING TO THE LEFT OF THE VERTICAL
6 LINE VERSUS BEING TO THE RIGHT.

7 Q NOW, TO THE RIGHT OF THE VERTICAL LINE AT 1,
8 THERE IS A DOTTED LINE. DO YOU SEE THAT?

9 A YES.

10 Q WHAT IS THAT DOTTED LINE INDICATING?

11 A THAT'S THE OVERALL RISK FROM POOLING ALL OF
12 THE STUDY RESULTS.

13 Q DOES THAT MEAN THAT POOLING ALL OF THE
14 STUDIES THAT WERE THE SUBJECT OF THIS REVIEW OR
15 META-ANALYSIS -- THAT ALL OF THE STUDIES POOLED TOGETHER
16 DID SHOW AN INCREASED RISK, REFLECTED BY THE DOTTED LINE
17 TO THE RIGHT OF THE SOLID VERTICAL LINE?

18 A YEAH. THE VERTICAL DOTTED LINE IS TO THE
19 RIGHT, AND THAT SHOWS AN INCREASED RISK.

20 MR. METZGER: ALL RIGHT. I SAID THREE. I'M GOING
21 TO KEEP TO MY WORD, YOUR HONOR.

22 Q THE ZHOU DOSE RESPONSE META-ANALYSIS OF
23 COFFEE CONSUMPTION AND BLADDER CANCER, EXHIBIT 11015,
24 MR. SCHURZ WENT OVER WITH YOU. AND I'D LIKE TO DIRECT
25 YOUR ATTENTION TO FIGURE 2 OF THAT META-ANALYSIS. AND
26 THIS IS FOR BLADDER CANCER.

27 AND ONCE AGAIN, IN THIS ONE, THE RISK IS
28 ALONG THE HORIZONTAL AXIS?

1 A EXCUSE ME. ARE YOU JUST SPEAKING COFFEE AND
2 BLADDER CANCER?

3 Q YES, THIS IS COFFEE AND BLADDER CANCER, THE
4 ZHOU META-ANALYSIS. THE TITLE IS:

5 "A DOSE RESPONSE META-ANALYSIS OF COFFEE
6 CONSUMPTION AND BLADDER CANCER, BY ZHOU, ET
7 AL., 2012."

8 IT'S ONE OF THE STUDIES YOU RELIED ON.

9 NOW, DOES THIS SHOW -- WHAT DOES THIS
10 GRAPHIC PRESENTATION OF ALL OF THE STUDIES INDICATE TO
11 YOU REGARDING RISK FOR COFFEE CONSUMPTION AND BLADDER
12 CANCER?

13 A WELL, IT INDICATES THAT THE MAJORITY OF THE
14 STUDIES ARE TO THE RIGHT OF 1. SO THE MAJORITY OF THE
15 STUDIES INDICATE AN INCREASED RISK. AND THEN THE
16 VERTICAL DOTTED LINE INDICATES THAT THE POOLED ANALYSIS
17 OF ALL THE DATA SHOW AN INCREASED RISK.

18 MR. METZGER: VERY GOOD.

19 JUST ONE MOMENT, YOUR HONOR.

20 NO FURTHER QUESTIONS, YOUR HONOR.

21 THE COURT: ALL RIGHT. THANK YOU. MAY THE
22 WITNESS BE EXCUSED?

23 MR. SCHURZ: NO FURTHER QUESTIONS, YOUR HONOR.

24 THE COURT: ALL RIGHT. DR. INFANTE, YOU MAY BE
25 EXCUSED. THANK YOU.

26 THE WITNESS: YOUR HONOR, I DIDN'T UNDERSTAND THE
27 ISSUE ABOUT MY NOTES. DO YOU WANT MY NOTES OR NOT?

28 THE COURT: NO. JUST LEAVE BEFORE ANOTHER

1 QUESTION COMES. YOU'RE BEING EXCUSED.

2 MR. METZGER, ARE YOU READY TO CALL YOUR NEXT
3 WITNESS?

4 MR. METZGER: YES. HE WENT OUT. WE'LL GO GET
5 HIM, YOUR HONOR. I'M SORRY.

6 (DISCUSSION HELD OFF THE RECORD.)

7 (PAUSE IN PROCEEDINGS.)

8 MR. METZGER: YOUR HONOR, THE PLAINTIFFS CALL DR.
9 JAMES HUFF.

10 THE CLERK: SIR, WILL YOU RAISE YOUR RIGHT HAND TO
11 BE SWORN.

12
13 JAMES EDWARD HUFF,
14 CALLED AS A WITNESS BY THE PLAINTIFF, WAS SWORN AND
15 TESTIFIED AS FOLLOWS:

16 THE CLERK: THANK YOU. PLEASE BE SEATED. AND
17 WILL YOU STATE AND SPELL YOUR NAME FOR THE RECORD.

18 THE WITNESS: JAMES EDWARD HUFF, H-U-F-F.

19 THE CLERK: THANK YOU.

20 THE COURT: ALL RIGHT. GOOD MORNING, DR. HUFF.

21 THE WITNESS: GOOD MORNING, SIR.

22 THE COURT: COUNSEL, MR. METZGER, YOU MAY PROCEED.

23 MR. METZGER: THANK YOU, YOUR HONOR.

24

25 DIRECT EXAMINATION

26 BY MR. METZGER:

27 Q GOOD MORNING, DR. HUFF. HOW ARE YOU?

28 A I'M FINE.

1 Q IS THIS YOUR FIRST TIME TESTIFYING IN COURT?

2 A YES.

3 Q OKAY. DR. HUFF, I WANT TO SHOW YOU WHAT'S
4 BEEN MARKED AS EXHIBIT 182. AND WOULD YOU CONFIRM THAT
5 THIS IS A BIOGRAPHICAL SUMMARY THAT YOU HAVE PREPARED.

6 A (REVIEWS DOCUMENT.)

7 YES, IT IS.

8 Q OKAY. AND I'D LIKE TO SHOW YOU WHAT'S BEEN
9 MARKED AS EXHIBIT 187 AND ASK YOU IF THIS IS YOUR LIST
10 OF PUBLICATIONS.

11 A YES, IT IS.

12 MR. METZGER: OKAY. YOUR HONOR, I WOULD OFFER IN
13 EVIDENCE EXHIBITS 182 AND 187.

14 THE COURT: 182, ANY OBJECTION?

15 MR. SCHURZ: NO OBJECTION, YOUR HONOR.

16 THE COURT: ALL RIGHT. 182 IS IN EVIDENCE.

17 187, ANY OBJECTION?

18 MR. SCHURZ: NO OBJECTION, YOUR HONOR.

19 THE COURT: 187 IS IN EVIDENCE.

20 (EXHIBITS 182 AND 187 MARKED FOR
21 IDENTIFICATION AND RECEIVED IN EVIDENCE.)

22 Q BY MR. METZGER: ALL RIGHT. WHILE WE'RE
23 DOING SOME EXHIBITS, I'LL DO TWO MORE, DR. HUFF. WOULD
24 YOU CONFIRM, PLEASE, THAT EXHIBIT 183 IS A LIST OF THE
25 WRITTEN OPINIONS THAT YOU PREPARED FOR THIS CASE.

26 A YES, IT IS.

27 (EXHIBIT 183 MARKED FOR IDENTIFICATION.)

28 Q BY MR. METZGER: OKAY. DID YOU PRODUCE THIS

1 LIST OF OPINIONS FOR YOUR DEPOSITION?

2 A YES, I DID.

3 Q VERY GOOD. AND I'LL NOW SHOW YOU WHAT'S
4 BEEN MARKED AS EXHIBIT 190. AND IS THIS A TWO-PAGE
5 DOCUMENT THAT YOU PREPARED, IN WHICH YOU PROVIDED A
6 CONCORDANCE FOR THE ANIMAL BIOASSAY STUDIES OF
7 ACRYLAMIDE AND GLYCIDAMIDE?

8 A YES.

9 (EXHIBIT 190 MARKED FOR IDENTIFICATION.)

10 Q BY MR. METZGER: AND DID YOU PRODUCE THIS
11 FOR YOUR DEPOSITION?

12 A YES, I DID.

13 Q LASTLY, I'M JUST GOING TO SHOW YOU WHAT HAS
14 BEEN MARKED AS EXHIBIT 194. AND WOULD YOU CONFIRM THAT
15 THIS IS A POWERPOINT PRESENTATION THAT YOU PREPARED FOR
16 THIS CASE TO ASSIST IN CLARIFYING YOUR TESTIMONY?

17 A YES, IT IS.

18 MR. METZGER: ALL RIGHT. THANK YOU.

19 (EXHIBIT 194 MARKED FOR IDENTIFICATION.)

20 Q BY MR. METZGER: DR. HUFF, WHERE DO YOU
21 LIVE?

22 A I LIVE IN CHAPEL HILL, NORTH CAROLINA.

23 Q AND DID YOU WORK NEARBY THERE FOR SOME TIME?

24 A YES. I WORKED AT THE NATIONAL INSTITUTE OF
25 ENVIRONMENTAL HEALTH SCIENCES, WHICH IS PART OF THE
26 NATIONAL INSTITUTES OF HEALTH, SINCE ROUGHLY 1979, 1980.

27 Q ALL RIGHT. I'D LIKE TO GO BACK IN TIME, AND
28 WOULD YOU TELL THE COURT WHAT -- JUST YOUR COLLEGE

1 EDUCATION, JUST WHAT DEGREES YOU RECEIVED AND FROM WHEN
2 AND WHERE.

3 A I WENT TO THE PHILADELPHIA COLLEGE OF
4 PHARMACY AND SCIENCE IN PHILADELPHIA, PENNSYLVANIA, AND
5 RECEIVED A BACHELOR'S DEGREE IN PHARMACY IN 1963; A
6 MASTER'S IN PHARMACOLOGY AND PHARMACEUTICS.

7 I THEN WENT TO PURDUE UNIVERSITY AND
8 RECEIVED A PH.D. IN 1968 IN BIONUCLEONICS.

9 Q WHAT IS BIONUCLEONICS?

10 A IT'S A -- AT THE TIME, IT WAS A NEW TERM, TO
11 COORDINATE THE EFFECTS OF RADIATION OF ALL TYPES AND
12 BIOLOGICAL SYSTEMS.

13 Q DID YOU DO POSTDOCTORAL WORK?

14 A YES. I WENT TO THE FEDERATION OF AMERICAN
15 SOCIETIES FOR EXPERIMENTAL BIOLOGY IN BETHESDA, MARYLAND
16 FOR ROUGHLY 18 MONTHS, AS A POSTDOC.

17 Q WHAT DID YOU DO NEXT IN YOUR CAREER?

18 A I WENT TO THE UNIVERSITY OF ROCHESTER
19 MEDICAL SCHOOL, TO TEACH PHARMACOLOGY; AND TO DO
20 RESEARCH ON CHOLINESTERASE INHIBITOR PESTICIDES AND
21 HIGH-ALTITUDE EFFECTS ON ERYTHROPOIETIN IN BEAGLE DOGS.

22 Q OKAY. AND WHAT DID YOU DO AFTER YOUR
23 EMPLOYMENT AT ROCHESTER?

24 A I WENT TO THE OAK RIDGE NATIONAL LABORATORY
25 IN KNOXVILLE, TENNESSEE, TO DEVELOP A PROGRAM IN
26 DATABASE GATHERING AND INFORMATION ON THE MAJOR END
27 POINTS IN TOXICOLOGY; THAT IS, TERATOLOGY, MUTAGENESIS,
28 TOXICITY, AND CARCINOGENESIS.

1 Q WERE YOU THE CHIEF OF THE UNITS THAT
2 ASSESSED TOXICOLOGICAL RISKS OF CHEMICALS AND
3 ENVIRONMENTAL EXPOSURES AT OAK RIDGE?

4 A YES.

5 Q AND DURING THIS TIME, DID YOU WORK IN
6 DEVELOPING THE TOXLINE DATABASE, WHICH IS PART OF THE
7 NATIONAL LIBRARY OF MEDICINE?

8 A YES. AND PART OF OUR AGREEMENT WITH THE
9 NATIONAL LIBRARY OF MEDICINE IN BETHESDA, MARYLAND --
10 WHICH IS ALSO PART OF NIH -- THEY WERE AT THE TIME
11 BEGINNING TO DEVELOP A PROGRAM TO COLLABORATE WITH, IF
12 YOU WILL, MEDLINE, WHICH WAS ALREADY RUNNING AS FAR
13 AS -- HUMAN ADVANCEMENTS IN SCIENCE WERE BEING
14 COLLECTED.

15 AND HENRY KISSMAN, WHO WAS AT THE NATIONAL
16 LIBRARY OF MEDICINE, WANTED TO DO A SIMILAR PROGRAM, BUT
17 IN TOXICOLOGY.

18 Q AND DID HE SELECT YOU TO DO THAT?

19 A YES, HE DID.

20 Q ALL RIGHT. HOW LONG WERE YOU AT OAK RIDGE?

21 A FIVE YEARS, PERHAPS.

22 Q OKAY. AND WHERE DID YOU GO NEXT?

23 A I WAS RECRUITED TO GO TO THE INTERNATIONAL
24 AGENCY FOR RESEARCH ON CANCER.

25 Q WHO RECRUITED YOU?

26 A DR. LORENZO TOMATIS.

27 Q WHO IS HE?

28 A HE WAS THE INNOVATOR OF THE IARC MONOGRAPH

1 PROGRAM TO EVALUATE CARCINOGENIC RISK OF CHEMICALS.

2 Q AND WHAT DID DR. TOMATIS ASK YOU TO DO?

3 A HE ASKED ME TO JOIN THEM AND HEAD UP THAT
4 PROGRAM ON THE IARC MONOGRAPHS.

5 Q YOU'RE SAYING "IARC." IS THAT WHAT WE'VE
6 BEEN CALLING DURING THE TRIAL IARC (DIFFERENT
7 PRONUNCIATION)?

8 A I'M SORRY. YES, SIR.

9 Q THAT'S THE INTERNATIONAL AGENCY FOR RESEARCH
10 ON CANCER?

11 A YES, WHICH IS PART OF THE WORLD HEALTH
12 ORGANIZATION.

13 Q AND DID YOU BECOME THE CHIEF OF THE IARC
14 MONOGRAPHS PROGRAM ON THE EVALUATION OF CARCINOGENIC
15 RISKS TO HUMANS?

16 A YES, I DID.

17 Q WILL YOU TELL US WHAT YOU DID WHEN YOU WERE
18 AT IARC.

19 A WELL, THE PROCEDURE WAS -- IN IARC WAS TO
20 HAVE THREE MEETINGS PER YEAR ON VARIOUS CHEMICALS, TO
21 EVALUATE THEIR CARCINOGENICITY IN ANIMALS AND HUMANS.
22 AND AT EACH OF THESE MEETINGS, WE WOULD ENLIST THE HELP
23 OF EXPERTS IN THAT AREA THAT WE WERE LOOKING INTO.

24 AND THESE WOULD BE EVALUATED AND THEN
25 CLASSIFIED AS TO WHETHER THEY WERE ANIMAL CARCINOGENS OR
26 HUMAN CARCINOGENS. AND I WAS IN CHARGE OF THIS PROGRAM.

27 Q ALL RIGHT. NOW, WE'VE HEARD ABOUT THE IARC
28 CLASSIFICATION SYSTEM FOR CARCINOGENIC RISK TO HUMANS.

1 DID YOU HAVE INVOLVEMENT IN THAT?

2 A YES. I BELIEVE IT WAS THE SECOND YEAR OR SO
3 THAT I WAS THERE THAT WE CONVENED A GROUP OF EXPERTS,
4 AND WE PREPARED -- WROTE AND PREPARED THE PREAMBLE TO
5 THE IARC MONOGRAPHS. AND AT THE TIME, WE THEN
6 ESTABLISHED CATEGORIES OF EVIDENCE, WHICH WERE NOT IN
7 PLACE PRIOR TO THAT.

8 Q ALL RIGHT. AND WAS THAT DONE UNDER YOUR
9 DIRECTION, AS THE CHIEF OF THE IARC MONOGRAPHS PROGRAM?

10 A YES, IT WAS.

11 Q ALL RIGHT. HOW LONG DID YOU STAY AT IARC?

12 A JUST A LITTLE UNDER THREE YEARS.

13 Q AND WHAT DID YOU DO NEXT?

14 A I WENT TO THE -- I WAS RECRUITED BY DAVID
15 RALL, WHO WAS THEN THE HEAD OF THE NATIONAL INSTITUTE OF
16 ENVIRONMENTAL HEALTH SCIENCES.

17 AND HE WAS ABOUT TO CREATE, IF YOU WILL, A
18 NEW PROGRAM IN THE DEPARTMENT OF HEALTH, EDUCATION, AND
19 WELFARE -- WHICH IS NOW THE DEPARTMENT OF HEALTH AND
20 HUMAN SERVICES -- TO COORDINATE THE DEPARTMENT'S
21 TOXICOLOGY EFFORTS BECAUSE THEY WERE VERY DISPARATE
22 AMONG THE VARIOUS INSTITUTES WITHIN THAT DEPARTMENT.

23 Q AND WHAT WAS THAT AGENCY THAT WAS CREATED?

24 A THE NATIONAL TOXICOLOGY PROGRAM.

25 Q AND WHAT WAS YOUR INVOLVEMENT IN THAT?

26 A I WAS ACTUALLY THE FIRST PERSON RECRUITED TO
27 ASSIST DAVID RALL IN ESTABLISHING AND PROMOTING THAT
28 PROGRAM. AND IN PARTICULAR, I WAS ASKED TO HEAD UP THE

1 PROGRAM ON CHEMICAL CARCINOGENESIS BIOASSAYS.

2 Q OKAY. WHAT IS A CHEMICAL CARCINOGENESIS
3 BIOASSAY?

4 A THIS IS A STUDY THAT IS DONE TO IDENTIFY
5 CHEMICALS THAT MAY BE CARCINOGENIC TO HUMANS, USING
6 RODENTS -- AND IN OUR CASE, RATS AND MICE; AND MOST
7 OTHER INVESTIGATORS, ALSO;

8 AND BASICALLY, EXPOSING THESE ANIMALS FOR
9 APPROXIMATELY TWO YEARS TO VARIOUS LEVELS OF THE
10 CHEMICAL OF INTEREST, AND AT THE END OF THAT TIME, TO
11 DETERMINE WHETHER IT WAS INDEED A CARCINOGEN OR NOT.

12 Q OKAY. AND DID YOU HEAD UP THIS EFFORT?

13 A YES, I DID.

14 Q TELL US BRIEFLY ABOUT HOW THE NATIONAL
15 TOXICOLOGY PROGRAM EVOLVED UNDER YOUR TENURE.

16 A WELL, IN PARTICULAR, FOR THE CARCINOGENESIS
17 PART OF IT, WE INHERITED THIS PROGRAM FROM THE NATIONAL
18 CANCER INSTITUTE. AND THEY WERE DOING, IF YOU WILL,
19 SINGLE- OR DOUBLE-DOSE STUDIES ON THESE RATS AND MICE
20 FOR THE TWO YEARS, AS I MENTIONED.

21 AND IT WAS MY INTEREST TO INCREASE THAT, TO
22 MAKE IT MORE RELEVANT TO WHAT THE REGULATORY AGENCIES
23 NEEDED TO THEN DEVISE STANDARDS FOR THESE EXPOSURES TO
24 THESE CHEMICALS.

25 AND WHAT WE DID, IN BRIEF, IS TO INCREASE
26 THE NUMBER OF DOSE GROUPS SO THAT WE COULD GET A BETTER
27 EFFORT -- A BETTER HANDLE ON DOSE-RESPONSE
28 RELATIONSHIPS, SO THAT WE COULD -- THESE WOULD BE BETTER

1 SUITED FOR MAKING RISK ASSESSMENTS BY THE REGULATORY
2 AGENCIES.

3 AND WE ALSO DEvised A CATEGORY OF
4 ESTABLISHING WHAT LEVEL OF EVIDENCE THE RESULTS OF THESE
5 STUDIES WOULD ENTAIL. THE NATIONAL CANCER INSTITUTE HAD
6 TWO LEVELS: NONCARCINOGENIC IN ANIMALS AND CARCINOGENIC
7 IN ANIMALS.

8 AND WE THOUGHT THAT THAT WAS A BIT TOO
9 RESTRICTIVE. SO WE DEVELOPED ANOTHER PROGRAM THAT WOULD
10 BE MORE -- WOULD ALLOW US TO BETTER CATEGORIZE THESE
11 AGENTS.

12 AND WE DEVELOPED A PROGRAM -- OR I DEVELOPED
13 THE LEVELS OF EVIDENCE, FIVE CATEGORIES. "CLEAR
14 EVIDENCE OF CARCINOGENICITY" WAS THE TOP CATEGORY.
15 "SOME EVIDENCE" WAS THE NEXT. "EQUIVOCAL" WAS THE
16 THIRD. THE FOURTH WAS "NO EVIDENCE."

17 AND WE CHANGED THAT FROM "NOT CARCINOGENIC"
18 BECAUSE "NOT CARCINOGENIC" IS TOO STRICT A STATEMENT IN
19 THAT WE'VE ONLY STUDIED THESE IN TWO SPECIES.

20 AND THEN WE ADDED ONE CATEGORY THAT
21 HOPEFULLY WE DIDN'T HAVE TO USE, WHAT'S CALLED
22 "INADEQUATE STUDY."

23 Q OKAY. ALL RIGHT. HOW MANY YEARS DID YOU
24 SPEND AT THE NATIONAL TOXICOLOGY PROGRAM?

25 A ROUGHLY 12; 12 YEARS.

26 Q AND DURING YOUR YEARS AT NTP, HOW MANY
27 STUDIES -- CARCINOGENESIS BIOASSAY STUDIES DID NTP
28 UNDERTAKE UNDER YOUR DIRECTION?

1 A I WAS INVOLVED IN, DURING THAT TIME PERIOD,
2 APPROXIMATELY 200 TO 225 STUDIES THAT WERE DESIGNED,
3 CARRIED OUT, AND EVALUATED AND PUBLISHED UNDER MY
4 DIRECTION.

5 Q ALL RIGHT. AND WHAT WAS YOUR INVOLVEMENT IN
6 THESE STUDIES?

7 A I WAS THE CHIEF OF THE ESTABLISHMENT OF
8 DOING THESE STUDIES: DESIGNING THEM, ASSIGNING THEM TO
9 DIFFERENT LABORATORIES -- CONTRACT LABORATORIES, BECAUSE
10 WE DIDN'T HAVE THE SPACE TO DO THAT AT OUR INSTITUTE, OR
11 THE PERSONNEL.

12 AND THEN TO EVALUATE THE RESULTS OF THESE
13 AND PRESENT THEM IN OPEN SESSION TO OUR PEER-REVIEWERS,
14 TO EVALUATE OUR STUDY.

15 Q ALL RIGHT. DURING THE TRIAL, THERE HAS BEEN
16 MENTION MADE OF THE NATIONAL TOXICOLOGY PROGRAM REPORT
17 ON CARCINOGENS. DID YOU HAVE SOME INVOLVEMENT REGARDING
18 THAT REPORT?

19 A YES, I DID. I WAS PART OF THE FIRST GROUP
20 OF, ACTUALLY, THREE SCIENTISTS THAT WERE RESPONSIBLE FOR
21 DECIDING WHICH CHEMICALS WOULD BE ADDED TO THE THEN
22 CONGRESS-MANDATED REPORT ON CARCINOGENS.

23 Q TELL US ABOUT THAT. WHEN YOU SAY "CONGRESS-
24 MANDATED REPORT ON CARCINOGENS," COULD YOU EXPLAIN WHAT
25 YOU MEAN.

26 A APPARENTLY, I WAS -- ARRIVED AFTER THAT WAS
27 DECIDED. BUT FROM WHAT I KNOW, IS THAT CONGRESS DECIDED
28 THAT, AS THERE WAS NO LIST OR GROUP OF CHEMICALS

1 OFFICIALLY SANCTIONED IN THE UNITED STATES AS TO WHAT IS
2 AND WHAT ISN'T A CARCINOGEN, THAT IT STARTED, WITH
3 SEVERAL CONGRESSIONAL MEMBERS, TO ESTABLISH SUCH A
4 LISTING.

5 AND THEY DECIDED AND GOT THE DEPARTMENT
6 HEAD, JOSEPH CALIFANO, TO AGREE TO IT, THAT THERE WOULD
7 BE A LIST OF AGENTS OR CHEMICALS THAT WERE CONSIDERED TO
8 BE EITHER KNOWN TO BE CARCINOGENIC IN HUMANS OR THE
9 SECOND CATEGORY, REASONABLY ANTICIPATED TO BE
10 CARCINOGENIC TO HUMANS.

11 AND THESE WERE THE BASIS OF OUR REPORT.

12 Q ALL RIGHT. I'D LIKE TO ASK YOU ABOUT A FEW
13 OF THE AWARDS THAT YOU'VE RECEIVED. DID YOU RECEIVE AN
14 AWARD FROM THE AMERICAN PUBLIC HEALTH ASSOCIATION?

15 A YES, I DID.

16 Q WOULD YOU TELL US WHAT THAT WAS.

17 A IT WAS ESTABLISHED AT THE -- THIS AMERICAN
18 PUBLIC HEALTH ASSOCIATION, A LARGE ORGANIZATION OF
19 ROUGHLY 50,000 MEMBERS. AND I WAS SELECTED TO RECEIVE
20 THE PUBLIC HEALTH ADVOCACY AWARD IN THE NAME OF -- IN
21 THE HONOR OF DAVID RALL.

22 Q OKAY. AND WAS THAT AWARD MADE TO YOU TO
23 RECOGNIZE INDIVIDUALS WHO HAVE MADE OUTSTANDING
24 CONTRIBUTIONS TO PUBLIC HEALTH THROUGH SCIENCE-BASED
25 ADVOCACY?

26 A YES.

27 Q ALL RIGHT. ARE YOU FAMILIAR WITH THE
28 INTERNATIONAL ORGANIZATION KNOWN AS THE COLLEGIUM

1 RAMAZZINI?

2 A YES.

3 Q WHAT IS THAT ORGANIZATION?

4 A IT'S AN ORGANIZATION THAT WAS FORMED BY
5 MAINLY TWO INDIVIDUALS: IRVING SELIKOFF, WHO WAS
6 INSTRUMENTAL IN ESTABLISHING THE CARCINOGENICITY OF
7 ASBESTOS; AND THEN CESARE MALTONI, FROM ITALY, WHO WAS
8 INSTRUMENTAL IN, AMONG OTHER THINGS, ESTABLISHING THE
9 CARCINOGENICITY OF VINYL CHLORIDE.

10 THOSE TWO INDIVIDUALS, WITH OTHERS LIKE
11 DAVID RALL AND MANY OTHERS, DECIDED THAT THERE SHOULD BE
12 AN INTERNATIONAL ORGANIZATION TO MAKE SURE THAT THE REST
13 OF THE WORLD KNEW ABOUT THESE POTENTIAL ENVIRONMENTAL,
14 OCCUPATIONAL, AND PUBLIC HEALTH HAZARDS FROM CHEMICALS.

15 AND THIS ORGANIZATION IS LIMITED TO 180
16 MEMBERS THROUGHOUT THE WORLD.

17 Q AND WERE YOU INVITED TO BE A MEMBER OF THE
18 COLLEGIUM RAMAZZINI?

19 A YES, I WAS.

20 Q ALL RIGHT.

21 A I WAS THE SECOND, FOLLOWING DR. RALL, FROM
22 THE ENTIRE NATIONAL INSTITUTES OF HEALTH.

23 Q OKAY. I WANT TO ASK YOU ABOUT SOME OF YOUR
24 RESEARCH INTERESTS, WHICH I BELIEVE I GOT FROM ONE OF
25 YOUR DOCUMENTS. HAS YOUR PRIMARY RESEARCH INTEREST OVER
26 YOUR CAREER BEEN CHEMICAL CARCINOGENESIS?

27 A YES.

28 Q ALL RIGHT. AND YOU HAVE YOU -- HAS YOUR

1 RESEARCH FOCUSED ON IMPACTS OF CHEMICALS ON THE
2 ENVIRONMENT, OCCUPATIONAL, AND GENERAL PUBLIC HEALTH?

3 A YES.

4 Q AND HOW MUCH OF YOUR CAREER HAS BEEN
5 INVOLVED IN EITHER DESIGNING, CONDUCTING, OR EVALUATING
6 CHEMICAL CARCINOGENESIS BIOASSAYS?

7 A CLOSE TO ALL OF MY TIME.

8 Q OKAY. AND HAVE YOU -- HAS YOUR RESEARCH
9 ALSO FOCUSED ON EXPLORING MECHANISMS OF CARCINOGENIC
10 ACTIVITY?

11 A YES.

12 Q AND HAS YOUR RESEARCH ADDRESSED TEST METHODS
13 FOR ASSESSING CARCINOGENIC RISKS OF CHEMICALS?

14 A YES.

15 Q AND HAVE YOU UNDERTAKEN ALL OF THOSE
16 ACTIVITIES FOR THE PURPOSE OF IMPROVING PUBLIC HEALTH?

17 A YES, I HAVE.

18 Q ALL RIGHT. NOW, THIS LIST OF PUBLICATIONS
19 OF YOURS, JUST BRIEFLY. YOU HAVE PUBLISHED MORE THAN
20 300 SCIENTIFIC PUBLICATIONS?

21 A ROUGHLY 450, YES.

22 Q OKAY. INCLUDING ARTICLES IN THE PEER-
23 REVIEWED LITERATURE?

24 A MOST ARE IN PEER-REVIEWED LITERATURE, YES.

25 Q OKAY. AND IARC MONOGRAPH DOCUMENTS?

26 A YES.

27 Q OKAY.

28 A I BELIEVE I WAS RESPONSIBLE, WHILE I WAS AT

1 THE INTERNATIONAL AGENCY FOR RESEARCH ON CANCER, FOR
2 NINE OR TEN MONOGRAPHS. I DON'T RECALL EXACTLY.

3 Q OKAY. AND NATIONAL TOXICOLOGY TECHNICAL
4 REPORTS?

5 A YES. I WAS RESPONSIBLE FOR GREATER THAN 200
6 OF THOSE DOCUMENTS -- THOSE STUDIES AND THE DOCUMENTS.

7 Q ALL RIGHT. I JUST WANT TO FINISH THROUGH
8 YOUR CAREER. WHEN DID YOU LEAVE THE NATIONAL TOXICOLOGY
9 PROGRAM?

10 A IT WAS IN THE EARLY 1990S.

11 Q WHERE DID YOU GO?

12 A I STAYED WITHIN THE INSTITUTE, BUT I WENT
13 INTO THE UNIT OF CHEMICAL CARCINOGENESIS, TO PURSUE MANY
14 OF THE THINGS WE'VE ALREADY DISCUSSED, WITHOUT HAVING
15 THE ADMINISTRATIVE LEAD IN THE NATIONAL TOXICOLOGY
16 PROGRAM.

17 AND DR. RALL HAD -- OR WAS RETIRING, AND WE
18 THOUGHT IT AN OPPORTUNE TIME FOR ME TO BE ABLE TO DEVOTE
19 MORE OF MY TIME TO LOOKING INTO THESE ISSUES AND PUBLISH
20 IN PEER-REVIEWED JOURNALS.

21 Q ALL RIGHT. SO YOU LEFT THE NTP BUT STAYED
22 WITHIN THE NATIONAL INSTITUTE FOR ENVIRONMENTAL HEALTH
23 SCIENCES?

24 A YES.

25 Q DID YOU EVENTUALLY RETIRE FROM THAT
26 INSTITUTE?

27 A YES.

28 Q WHAT YEAR?

1 A JANUARY 2013 .

2 Q AND HOW MANY YEARS TOTAL DID YOU SPEND AFTER
3 YOU LEFT NTP UNTIL YOU RETIRED FROM NIEHS?

4 A CLOSE TO 20 .

5 Q OKAY. AND DURING THOSE YEARS, WERE YOU
6 PRIMARILY DOING RESEARCH AND PUBLICATION?

7 A YES .

8 Q ALL RIGHT. SO I'D LIKE YOU TO EXPLAIN TO
9 THE COURT IN A LITTLE BIT OF DETAIL, IF YOU COULD --
10 BEFORE WE GET TO THAT, LET ME ASK YOU: WHAT
11 IS THE PURPOSE OF DOING AN ANIMAL CANCER BIOASSAY?

12 A IN SHORT, THE PURPOSE IS TO IDENTIFY THOSE
13 CHEMICALS THAT MAY CAUSE A CARCINOGENIC RISK TO HUMANS .

14 Q OKAY. AND WOULD YOU TELL THE COURT HOW
15 THOSE HAVE BEEN DONE AT NTP .

16 A YES. IT'S A MANY-YEAR PROJECT. OF COURSE,
17 THE FIRST THING TO DO IS TO DECIDE -- OR SELECT WHICH
18 CHEMICAL OR CHEMICALS ONE NEEDS TO EVALUATE .

19 AND AT THE BEGINNING OF THE PROGRAM, WE TOOK
20 CHEMICALS THAT, SCIENTIFICALLY, FROM OUR EXPERIENCE, WE
21 THOUGHT WOULD BE CARCINOGENIC TO ANIMALS. AND IN
22 EVALUATING THOSE SELECTIONS, WE FOUND THAT OF THE FIRST
23 SEVERAL HUNDRED, THAT APPROXIMATELY 65 PERCENT WERE
24 CARCINOGENIC. SO WE WERE CORRECT IN GUESSING THAT THEY
25 WOULD BE CARCINOGENIC .

26 AND THEN THE PROGRAM SWITCHED TO EVALUATE
27 CHEMICALS THAT WERE, NUMBER ONE, HIGH PRODUCTION
28 CHEMICALS IN THE UNITED STATES; AND NUMBER TWO, A LARGE

1 NUMBER OF PEOPLE WERE EXPOSED. AND IN EVALUATING THOSE,
2 WE FOUND ONLY 10 TO 15 TO 18 PERCENT WERE CARCINOGENIC.

3 SO TO DO THOSE STUDIES, WE HAVE TO BEGIN
4 WITH WHAT ARE CALLED PRE-CHRONIC STUDIES, WHERE YOU USE
5 SMALL GROUPS AND SEVERAL DOSE GROUPS AND CONTROLS, FIVE
6 OR SIX DOSE GROUPS OF MALE RAT, FEMALE RAT, MALE MICE,
7 AND FEMALE MICE.

8 AND WE DO THE TWO SPECIES BECAUSE THEY REACT
9 SOMEWHAT INDEPENDENTLY. THE SAME WITH BOTH SEXES
10 BECAUSE OF HORMONAL INFLUENCES ON CARCINOGENESIS.

11 SO WE DO THESE SHORT-TERM TESTS, 14 DAYS,
12 AND EVALUATE THAT. THEN WE DO 90-DAY STUDIES AND
13 EVALUATE THAT.

14 THEN WE DECIDE HOW TO APPROACH THE DESIGN OF
15 A TWO-YEAR STUDY, EXPOSING THE ANIMALS TO SUFFICIENT
16 AMOUNT OF CHEMICAL FOR -- TO ENSURE THAT WE WOULD HAVE A
17 VALID END POINT AND THAT THE ANIMALS WOULD BE EVALUATED,
18 BOTH POSITIVE OR NEGATIVE, AND THAT WE WOULD BE
19 CONFIDENT IN THOSE FINDINGS.

20 AND SO IN THESE CASES, WE WOULD USE TWO OR
21 THREE DOSE GROUPS, DEPENDING, AND CONTROLS FOR EACH OF
22 THE SEX/SPECIES GROUPS, ROUGHLY 800 TO 1,000 ANIMALS;
23 AND EXPOSE THESE ANIMALS BY VARIOUS ROUTES OF EXPOSURE,
24 WHICH WAS THE MOST RELEVANT TO HUMAN EXPOSURE, FOR THE
25 TWO YEARS.

26 AND THEN WE DID OUR EVALUATIONS IN
27 SUBSEQUENT MONTHS AND YEARS AFTER THEY WERE FINISHED,
28 THE PATHOLOGICAL EVALUATIONS.

1 SO BASICALLY, THAT'S A BIOASSAY.

2 Q ALL RIGHT. SO I'D LIKE TO ASK YOU: ARE
3 PEOPLE RATS?

4 A NO, BUT --

5 THE COURT: ASK ANOTHER QUESTION.

6 MR. METZGER: OKAY.

7 Q ARE PEOPLE LIKE RATS?

8 A YES. THERE ARE CLEARLY MORE PHYSIOLOGIC,
9 BIOCHEMICAL, PHARMACOLOGIC, GENOMIC SIMILARITIES BETWEEN
10 THESE TWO MAMMALIAN SPECIES -- THAT IS, RODENTS AND
11 HUMANS -- THAN THERE ARE DIFFERENCES.

12 AND WE KNOW FROM FORMULATING THE USE OF
13 DRUGS IN ANIMALS TO HUMANS THAT THIS IS A VALID
14 COMPARISON.

15 Q OKAY. AND ARE THESE BIOLOGICAL SIMILARITIES
16 BETWEEN HUMANS AND RODENTS THAT YOU'VE DESCRIBED -- IS
17 THAT OF VALUE FOR PURPOSES OF -- DOES THAT SHOW VALUE OF
18 DOING CANCER BIOASSAYS?

19 A YES.

20 Q OKAY. AND HAVE YOU PUBLISHED TO THAT
21 EFFECT, THAT THE BIOLOGICAL SIMILARITIES BETWEEN HUMANS
22 AND RODENTS IS OF VALUE FOR CONDUCTING CANCER BIOASSAYS?

23 A YES, I HAVE.

24 Q OKAY. NOW, COULD YOU TELL THE COURT IF
25 THERE IS A GENERAL RELATIONSHIP BETWEEN ANIMAL
26 CARCINOGENICITY AND HUMAN CARCINOGENICITY.

27 A WELL, FROM OUR UNDERSTANDING, THE ANSWER IS
28 YES.

1 Q AND WHAT IS THE RELATIONSHIP?

2 A WELL, FROM THE -- SIMPLY FROM AN END POINT
3 OF THE CARCINOGENESIS STUDIES, WE FOUND THAT OF ALL THE
4 KNOWN HUMAN CARCINOGENS -- AND THE NUMBER VARIES, BUT
5 IT'S ROUGHLY 100 OR 105 NOW -- ALL OF THESE THAT WERE
6 TESTED IN ANIMALS WERE ALSO LIKEWISE CARCINOGENIC.

7 AND ALMOST WITHOUT EXCEPTION, THEY SHARED --
8 THE RODENTS AND THE HUMANS SHARED THE ORGAN SITE THAT
9 WAS CARCINOGENIC IN BOTH.

10 Q SO EVERY HUMAN CARCINOGEN -- EVERY CHEMICAL
11 THAT WAS KNOWN TO CAUSE CANCER IN HUMANS, WHEN TESTED IN
12 ANIMALS, WAS ALSO CARCINOGENIC?

13 A YES.

14 Q OKAY. AND HAVE YOU PUBLISHED ARTICLES
15 DEMONSTRATING THAT RELATIONSHIP?

16 A YES, I HAVE.

17 Q ALL RIGHT. ARE THOSE LISTED ON YOUR LIST OF
18 PUBLICATIONS?

19 A YES.

20 Q ALL RIGHT. COULD YOU TELL THE COURT
21 APPROXIMATELY HOW MANY HUMAN CARCINOGENS WERE FIRST
22 DISCOVERED BY DOING ANIMAL CANCER BIOASSAYS.

23 A ROUGHLY 30 TO 40 PERCENT OF THE KNOWN HUMAN
24 CARCINOGENS WERE FIRST IDENTIFIED IN LONG-TERM
25 BIOASSAYS --

26 Q SO THEY --

27 A -- SUCH AS --

28 Q I'M SORRY. SO THEY WERE FIRST IDENTIFIED AS

1 BEING CARCINOGENIC IN ANIMALS?

2 A YES.

3 Q AND THEN SUBSEQUENTLY WERE DETERMINED TO BE
4 HUMAN CARCINOGENS?

5 A YES, SIR. THE BIOASSAY HAS VALUE IN BOTH
6 RETROSPECTIVE DETERMINATION AND ALSO PROSPECTIVE
7 IDENTIFICATION OF CARCINOGENS.

8 Q OKAY. NOW, I WANT TO ASK YOU ABOUT -- I
9 THINK IT'S CALLED REPRODUCIBILITY. HAVE ANIMAL CANCER
10 BIOASSAYS FOR A PARTICULAR CHEMICAL SHOWN
11 REPRODUCIBILITY FROM STUDIES DONE AT ONE LABORATORY TO
12 ANOTHER?

13 A YES.

14 Q AND WHAT IS THE DEGREE OF REPRODUCIBILITY?

15 A IT'S ALMOST TOTALLY CONSISTENT.

16 Q ALL RIGHT. AND HAVE YOU PUBLISHED ARTICLES
17 REACHING THAT CONCLUSION?

18 A YES, I HAVE.

19 Q ALL RIGHT. AND ARE THOSE -- ARE SOME OF
20 THOSE LISTED IN YOUR OPINIONS THAT -- THE LIST OF
21 OPINIONS THAT YOU PREPARED?

22 A YES.

23 Q AND ARE OTHERS ALSO LISTED ON YOUR LIST OF
24 PUBLICATIONS?

25 A YES.

26 Q ALL RIGHT. NOW, COULD YOU TELL THE COURT --
27 WELL, HAVE YOU FORMED AN OPINION -- AND I DON'T MEAN FOR
28 THIS CASE, BECAUSE I KNOW YOU'VE BEEN DOING THIS LONG

1 BEFORE THIS CASE. BUT IN YOUR WORK, DID YOU FORM A
2 CONCLUSION AS TO THE PREDICTIVE ABILITY OF BIOASSAYS TO
3 PROSPECTIVELY IDENTIFY HUMAN CARCINOGENS?

4 A YES, I DID.

5 Q WHAT IS YOUR OPINION?

6 A MY OPINION IS THAT -- OF COURSE, NOT ALL
7 CARCINOGENS ARE EQUAL, AND NOT ALL BIOASSAYS ARE EQUAL.
8 BUT UNDER THE CRITERIA OF BOTH THE IARC AND THE NTP, IF
9 THE RESULTS ON A CARCINOGENESIS BIOASSAY MEET THESE
10 CRITERIA, THEN THEY ARE CONSIDERED TO BE LIKELY TO CAUSE
11 CANCER IN HUMANS.

12 Q AND CAN YOU GIVE US SOME EXAMPLES OF
13 CHEMICALS THAT WERE PREDICTED TO CAUSE CANCER IN HUMANS
14 AND BY BIOASSAYS WHICH ULTIMATELY SHOWED THAT THEY WERE
15 CARCINOGENIC IN HUMANS?

16 A YES. AS I MENTIONED ALREADY, THERE'S 30
17 PERCENT OF HUMAN CARCINOGENS.

18 SOME NOTED EXAMPLES ARE VINYL CHLORIDE,
19 WHICH IS USED IN PLASTICS; 1,3-BUTADIENE, WHICH IS USED
20 IN TIRE MAKING; DIETHYLSTILBESTROL, WHICH IS A HORMONE
21 REPLACEMENT FOR WOMEN;

22 TRICHLORETHYLENE, WHICH IS A SOLVENT USED IN
23 DRY CLEANING AND DECAFFEINATING COFFEE; FORMALDEHYDE,
24 WHICH IS ANOTHER CHEMICAL THAT WAS FIRST SHOWN TO BE
25 CARCINOGENIC IN ANIMALS AND SUBSEQUENTLY IN HUMANS.

26 Q AND HAVE YOU PUBLISHED REGARDING THESE
27 CHEMICALS AS TO THEIR HUMAN CARCINOGENIC PREDICTIVITY IN
28 PEER-REVIEWED PUBLICATIONS?

1 A YES, I HAVE.

2 Q AND COULD YOU GIVE THE COURT AN EXAMPLE OF A
3 CHEMICAL THAT WAS FIRST IDENTIFIED AS A HUMAN CARCINOGEN
4 THROUGH EPIDEMIOLOGIC STUDIES AND THEN SUBSEQUENTLY
5 CONFIRMED THROUGH ANIMAL STUDIES TO BE CARCINOGENIC TO
6 ANIMALS?

7 A YES, THERE'S -- TWO MAJOR EXAMPLES ARE
8 ARSENIC AND BENZENE.

9 Q OKAY. AND HAVE YOU PUBLISHED REGARDING THAT
10 CONFIRMATION FOR HUMAN CARCINOGENICITY TO ANIMAL
11 CARCINOGENICITY?

12 A YES. FOR BOTH OF THOSE, I HAVE.

13 Q OKAY. NOW, ARE THERE OTHER ASSAYS OR TESTS
14 WHICH HAVE BEEN CONSIDERED TO BE USED OR USEFUL FOR
15 ASSESSING HUMAN CARCINOGENICITY OTHER THAN CHRONIC
16 ANIMAL CANCER BIOASSAYS?

17 A YES. AND MUCH OF MY CAREER HAS BEEN DEVOTED
18 TO ATTEMPTING TO VALIDATE THESE ASSAYS. BECAUSE THE
19 CANCER BIOASSAYS THAT I'VE BEEN INVOLVED WITH ARE TIME
20 CONSUMING, COSTLY, AND CAN NOWHERE NEAR STUDY AND
21 EVALUATE THE NUMBER OF CHEMICALS IN THE CONSUMER
22 MARKET -- WHICH VARIES, BUT IT'S ROUGHLY 100,000
23 DIFFERENT CHEMICALS THAT WE'RE EXPOSED TO. AND ONLY
24 2500 TO 3,000 HAVE BEEN EVALUATED FOR CARCINOGENESIS.

25 SO WE STARTED OFF TRYING TO DO WHAT THEY
26 CALL STRUCTURE ACTIVITY RELATIONSHIPS. IN OTHER WORDS,
27 IF YOU KNOW A CHEMICAL IS A CARCINOGEN, WHAT DOES IT
28 LOOK LIKE IN A TWO-DIMENSIONAL AND THREE-DIMENSIONAL

1 STRUCTURAL CONFIGURATION, CHEMICALLY, AND WOULD OTHERS
2 THAT RESEMBLE THAT BE CARCINOGENIC?

3 THIS HAD SOME SUCCESS, BUT IT DIDN'T WORK
4 OUT AS MUCH AS WE HAD HOPED. SO WE HAD TO -- NOT
5 ABANDON IT, BUT WE DIDN'T REPLACE THE BIOASSAY WITH
6 THAT.

7 THEN THERE ARE THE -- WHAT WE CALL THE
8 SHORT-TERM IN VITRO STUDIES, EXEMPLIFIED MAINLY BY THE
9 AMES ASSAY ON SALMONELLA, BY BRUCE AMES.

10 AND THIS WAS AN EXCITING DEVELOPMENT THAT
11 HIS CORRELATIONS BETWEEN HIS RESULTS IN GENE MUTAGENESIS
12 CORRELATED WITH THE KNOWN RESULTS OF HUMAN CARCINOGENS
13 GREATER THAN 90 PERCENT WHEN HE FIRST STARTED.

14 BUT AS TIME WENT BY AND THEY ADDED MORE AND
15 MORE CHEMICALS, THE CORRELATION DECREASED, DECREASED, TO
16 SUCH AN EXTENT THAT WE COULD NOT USE IT AS A PREDICTER.

17 AND THEN THERE WERE OTHER BIOASSAYS, SHORT-
18 TERM BIOASSAYS IN ANIMALS, THAT WERE HOPEFUL BUT DID NOT
19 PAN OUT. THE LAST ONE THAT WE LOOKED AT WAS WHAT THEY
20 CALLED TRANSGENIC ANIMALS, IN WHICH YOU INSERT A GENE
21 INTO THESE ANIMALS THAT IS SENSITIVE TO CARCINOGENIC
22 DEVELOPMENT, CANCER DEVELOPMENT.

23 AND WITHOUT GOING INTO DETAIL, THIS DIDN'T
24 WORK OUT, EITHER. BUT IT WAS A SEVERAL-YEAR EFFORT, AND
25 UNFORTUNATELY, IT DIDN'T WORK.

26 SO IN GENERAL -- OR IN CONCLUSION, THE
27 BIOASSAY REMAINS, IN MY OPINION, THE NUMBER ONE ASSAY TO
28 IDENTIFY HUMAN CARCINOGENS THAT WE HAVE.

1 Q OKAY. AND HAVE YOU PUBLISHED ARTICLES
2 CONCLUDING THAT THE CHRONIC ANIMAL CANCER BIOASSAY IS
3 THE MOST PREDICTIVE TEST FOR HUMAN CARCINOGENICITY?

4 A YES, I HAVE.

5 Q OKAY. NOW, I WANT TO ASK YOU: IN THESE
6 ANIMAL CANCER BIOASSAYS, ARE THE ANIMALS TYPICALLY
7 EXPOSED TO CHEMICALS AT DOSES GREATER THAN THOSE TO
8 WHICH HUMANS ARE EXPOSED TO THE CHEMICALS?

9 A YES, BUT NOT ALWAYS.

10 Q OKAY. AND WHY ARE THE ANIMALS OFTEN GIVEN
11 HIGHER DOSES OF THE CHEMICAL THAN HUMANS RECEIVE IN
12 THEIR DAILY LIFE?

13 A WELL, THE TOP DOSE OF A THREE-DOSE -- OR
14 THREE-EXPOSURE STUDY IS USUALLY CHOSEN TO STRESS THE
15 ANIMALS SUFFICIENTLY SO THAT WHATEVER THE RESULTS COME
16 BACK, WE REALIZE THAT THEY HAVE BEEN ADEQUATELY EXPOSED
17 TO A CHEMICAL. AND SO IF IT WAS NEGATIVE OR POSITIVE,
18 WE WOULD BE CONFIDENT IN THAT. IF IT'S LOWER THAN THAT,
19 YOU WOULDN'T KNOW WHAT IT WOULD HAVE DONE AT HIGHER
20 DOSES.

21 AND THEN WE USE LOWER DOSES, WHICH COME
22 CLOSER TO HUMAN EXPOSURES, TO SEE -- TO HOPE FOR A
23 DOSE-RESPONSE RELATIONSHIP THAT IS MORE RELEVANT AND
24 VALUABLE TO THE RISK ASSESSORS IN THE REGULATORY
25 AGENCIES TO THEN DO THEIR RISK ASSESSMENTS AND ESTABLISH
26 STANDARDS.

27 Q WELL, LET ME ASK YOU ABOUT THAT. WHY -- OR
28 WHAT INFORMATION DOES A DERIVATION OF A DOSE-RESPONSE

1 RELATIONSHIP PROVIDE TO RISK ASSESSORS IN CONDUCTING
2 RISK ASSESSMENTS FOR CHEMICALS AS TO THEIR
3 CARCINOGENICITY TO HUMANS?

4 A WELL, IT GIVES US MORE CONFIDENCE IN BEING
5 ABLE TO EXTRAPOLATE THESE RESULTS TO HUMANS BECAUSE AS
6 YOU GO DOWN -- GO LOWER IN THE DOSE RANGE, IT IS MORE --
7 CONSIDERED FOR RELEVANT TO WHAT WOULD BE THE HUMAN
8 EXPOSURE TO THAT SAME CHEMICAL.

9 Q OKAY. DO THE -- DOES DERIVATION OF A
10 DOSE-RESPONSE RELATIONSHIP PROVIDE THE DATA NECESSARY TO
11 GENERATE WHAT IS CALLED A SLOPE FACTOR?

12 A YES.

13 Q AND WHAT IS THAT?

14 A WELL, THERE'S VARIOUS MATHEMATICAL MODELS
15 THAT UTILIZE THESE RESULTS. AND IF YOU HAVE A MONOTONIC
16 RESPONSE IN DOSE VERSUS CARCINOGENESIS, THIS AGAIN IS
17 MORE CONDUCIVE OF A VALID INTERPRETATION THAT WOULD BE
18 BETTER USED FOR RISK ASSESSMENT.

19 Q OKAY. WHY DOESN'T THE NATIONAL TOXICOLOGY
20 PROGRAM SIMPLY EXPOSE ANIMALS TO CHEMICALS AT THE LEVELS
21 THAT -- TO WHICH HUMANS ARE EXPOSED?

22 A WELL, ACTUALLY, WE DO THAT, BUT IT'S NOT
23 COMMON. BUT THE REASON WE DON'T IS BECAUSE THESE
24 STUDIES ARE VERY LIMITED IN THEIR SENSITIVITY, IN THE
25 SENSE THAT YOU HAVE 50 ANIMALS IN A SEX/SPECIES GROUP
26 THAT HAS TO REPRESENT THE REST OF THE POPULATION.

27 SO WE WANT TO MAKE SURE THAT WE ARE EXPOSING
28 THESE ANIMALS TO SUCH A LEVEL THAT WE WOULD NOT MISS

1 ANYTHING, AS FAR AS CARCINOGENESIS. BECAUSE WE HAVE TO
2 SHOW A 10 PERCENT INCREASE TO EVEN CONSIDER THAT IT
3 WOULD BE A VALID RESPONSE. SO TO USE ONLY 50 ANIMALS,
4 YOU HAVE TO GO TO THE HIGHER DOSES.

5 Q I SEE. IF ONE WANTED TO DO AN ANIMAL CANCER
6 BIOASSAY FOR A CHEMICAL LIKE ACRYLAMIDE, WHERE YOU
7 EXPOSE THE ANIMALS TO ACRYLAMIDE AT THE LEVELS IN THE
8 HUMAN DIET, HOW MANY ANIMALS WOULD YOU NEED TO DO THAT
9 STUDY?

10 A IT WOULD BE HARD TO GUESS, BUT MANY
11 THOUSANDS, I BELIEVE.

12 Q OKAY. AND IS THAT PRACTICABLE TO DO?

13 A NO.

14 Q ALL RIGHT. SO I WANT TO ASK YOU ABOUT THIS
15 ISSUE OF HIGH EXPOSURES. DO MOST CHEMICAL CARCINOGENS
16 ONLY CAUSE CANCER AT THE HIGHEST EXPOSURE LEVELS TO
17 WHICH THE ANIMALS ARE DOSED?

18 A NO.

19 Q AND WHAT IS THE SIGNIFICANCE OF THAT?

20 A WELL, AGAIN, IT GIVES A DOSE RESPONSE THAT
21 IS MORE VALUABLE TO ESTABLISH A RISK ASSESSMENT OF THE
22 PARTICULAR CHEMICAL AND GIVES IT MORE VALIDITY WITH
23 RESPECT TO THE BIOLOGICAL RESPONSES THAT ONE SEES IN
24 THESE BIOASSAYS.

25 Q I SEE. DOES THE ESTABLISHMENT OF A DOSE
26 RESPONSE FOR CARCINOGENIC EFFECTS IN ANIMALS ENABLE RISK
27 ASSESSORS TO EXTRAPOLATE FROM THE ANIMAL DATA TO HUMAN
28 EXPOSURE LEVELS?

1 MR. SCHURZ: OBJECTION; OVERBROAD, LACKS
2 FOUNDATION AS TO THIS WITNESS'S UNDERSTANDING OF RISK
3 ASSESSMENT.

4 THE COURT: OVERRULED.

5 THE WITNESS: YES. THE MORE INFORMATION ONE HAS
6 IN A BIOASSAY, THE BETTER THEY ARE FOR BEING USED TO
7 ESTABLISH -- OR TO ASSIST IN RISK ASSESSMENT.

8 Q BY MR. METZGER: OKAY. WHAT IS YOUR
9 ULTIMATE CONCLUSION REGARDING THE VALUE OF CANCER
10 BIOASSAYS IN ASSESSING AND PREDICTING HUMAN CANCER
11 RISKS?

12 A WELL, I BELIEVE THAT A CARCINOGEN -- OR
13 CHEMICAL THAT CAUSES CANCER IN LABORATORY ANIMALS IS
14 CLEARLY CAPABLE OF CAUSING CANCER IN HUMANS.

15 Q NOW, HAVE ANIMAL CANCER BIOASSAYS BEEN USED
16 TO ASSESS THE CARCINOGENICITY OF INDUSTRIAL CHEMICALS?

17 A YES.

18 Q HAVE THEY BEEN USED TO ASSESS THE
19 CARCINOGENICITY OF PESTICIDES?

20 A YES.

21 Q HAVE THEY BEEN USED TO ASSESS THE
22 CARCINOGENICITY OF DRUGS?

23 A YES.

24 Q ALL RIGHT. SO NOW I'D LIKE TO ASK YOU ABOUT
25 PREDICTIVITY OF SPECIFIC RESULTS IN ANIMAL STUDIES IN
26 ASSESSING HUMAN CARCINOGENICITY.

27 FIRST, HAVE YOU FORMED AN OPINION AS TO THE
28 RELEVANCE OF INDUCTION OF CANCER AT MULTIPLE SITES IN

1 ANIMALS IN RELATION TO HUMAN CANCER?

2 A YES.

3 Q WHAT IS YOUR OPINION?

4 A THAT THESE ARE RELEVANT TO PREDICTIVITY OF
5 CANCERS IN HUMANS.

6 Q ARE YOU SAYING THAT THE MORE SITES THAT A
7 CHEMICAL CAUSES CANCER IN ANIMALS, THE MORE LIKELY THE
8 CHEMICAL WILL BE CARCINOGENIC TO HUMANS?

9 A YES.

10 MR. SCHURZ: OBJECTION; LEADING.

11 THE COURT: OVERRULED.

12 Q BY MR. METZGER: AND HAVE YOU -- IS THAT A
13 CONCLUSION THAT YOU REACHED BEFORE I ASKED YOU TO BE AN
14 EXPERT IN THIS LITIGATION?

15 A OH, YES.

16 Q AND IS THAT A CONCLUSION THAT YOU HAVE
17 PUBLISHED IN THE PEER-REVIEWED LITERATURE BEFORE YOU
18 WERE RETAINED FOR THIS CASE?

19 A YES.

20 Q ALL RIGHT. HAVE YOU FORMED AN OPINION AS TO
21 THE RELEVANCE OF INDUCING CANCERS IN ANIMALS IN MULTIPLE
22 SPECIES AS TO THE PREDICTIVITY OF HUMAN CANCER?

23 A YES.

24 Q WHAT IS YOUR OPINION?

25 A THIS ADDS TO THE PREDICTIVITY THAT IT WOULD
26 LIKELY BE CARCINOGENIC TO HUMANS, CAUSE CANCER IN
27 HUMANS.

28 Q AND HAVE YOU PUBLISHED THAT OPINION IN THE

1 PEER-REVIEWED LITERATURE?

2 A YES.

3 Q HAVE YOU FORMED AN OPINION AS TO THE
4 PREDICTIVITY OF HUMAN CANCER, OF THE INDUCTION OF CANCER
5 IN ANIMALS IN MULTIPLE STRAINS OF ANIMALS?

6 A YES.

7 Q AND WHAT IS YOUR OPINION?

8 A THIS TOO ADDS TO THE PREDICTIVITY OF CANCER
9 IN HUMANS.

10 Q AND WHAT IS THE BASIS FOR THAT OPINION?

11 A WELL, BECAUSE THE KNOWN HUMAN CARCINOGENS
12 ARE USING THAT AS A BACKGROUND AND SUBSTANTIATES THIS
13 VIEW.

14 Q AND HAVE YOU PUBLISHED THAT OPINION IN THE
15 PEER-REVIEWED LITERATURE BEFORE THIS LITIGATION?

16 A YES.

17 Q ALL RIGHT. HAVE YOU FORMED A CONCLUSION AS
18 TO WHETHER CANCER OCCURRING IN BOTH SEXES OF ANIMALS HAS
19 ANY PREDICTIVE ABILITY FOR HUMAN CANCER?

20 A YES.

21 Q WHAT IS YOUR OPINION?

22 A THIS TOO ALLOWS US TO BETTER PREDICT CANCERS
23 IN HUMANS.

24 Q AND HAVE YOU PUBLISHED THAT CONCLUSION, AS
25 WELL?

26 A YES.

27 Q ALL RIGHT. NOW, HAVE YOU FORMED AN OPINION
28 AS TO WHETHER A CHEMICAL THAT CAUSES MULTIPLE TUMORS IN

1 THE SAME ORGAN IN ANIMALS HAS ANY PREDICTIVE EFFECT FOR
2 HUMAN CANCER?

3 A YES, I HAVE.

4 Q WHAT IS YOUR OPINION?

5 A IT GIVES US FURTHER CONFIDENCE IN
6 CONSIDERING THAT THIS WOULD BE CARCINOGENIC TO HUMANS.

7 Q HAVE YOU PUBLISHED THAT?

8 A YES, I HAVE.

9 Q ALL RIGHT. AND DO YOU HAVE AN OPINION AS TO
10 WHETHER CANCERS INDUCED IN ANIMALS THAT METASTASIZE HAVE
11 ANY PREDICTIVE EFFECT FOR HUMAN CANCER?

12 A YES, I HAVE.

13 Q WHAT IS YOUR OPINION?

14 A AGAIN, THIS LEADS TO THE STRENGTH OF THE
15 EVIDENCE, AND IT ADDS TO THE PREDICTIVITY OF CANCER IN
16 HUMANS.

17 Q HAVE YOU PUBLISHED TO THAT EFFECT?

18 A YES, I HAVE.

19 Q ALL RIGHT. DO YOU HAVE AN OPINION AS TO
20 WHETHER A HIGH TUMOR INCIDENCE IN ANIMALS HAS PREDICTIVE
21 ABILITY FOR HUMAN CANCER?

22 A YES.

23 Q AND WHAT IS YOUR OPINION?

24 A THIS ADDS TO OUR ABILITY TO CONSIDER THAT
25 THESE ARE -- WILL CAUSE CANCER IN HUMANS.

26 Q HAVE YOU PUBLISHED THAT CONCLUSION?

27 A YES.

28 Q ALL RIGHT. NOW, WE'VE HEARD A LOT ABOUT

1 DOSE RESPONSE. FIRST OF ALL, WHAT IS DOSE --

2 THE COURT: SINCE WE'RE GOING ON TO A NEW SUBJECT,
3 WE'RE GOING TO TAKE A RECESS AT THIS TIME, AND I'LL CALL
4 A COUPLE OF OTHER CASES.

5 BE IN RECESS FOR ABOUT 15 MINUTES.

6 MR. METZGER: VERY WELL.

7 (RECESS.)

8 THE COURT: GOOD MORNING AGAIN, COUNSEL.

9 BACK ON THE RECORD IN CERT VS. STARBUCKS.
10 ALL COUNSEL ARE PRESENT, AND DR. HUFF IS ON THE STAND.

11 COUNSEL, PLEASE BE SEATED. MAKE YOURSELF
12 COMFORTABLE.

13 MR. METZGER WAS INQUIRING.

14 THE CLERK: SIR, YOU'VE PREVIOUSLY BEEN SWORN.
15 YOU'RE STILL UNDER OATH. PLEASE RESTATE YOUR NAME FOR
16 THE RECORD.

17 THE WITNESS: YES. JAMES EDWARD HUFF, H-U-F-F.

18 THE COURT: THANK YOU.

19 COUNSEL, YOU MAY PROCEED.

20 Q BY MR. METZGER: DR. HUFF, IN CONDUCTING
21 ANIMAL CANCER BIOASSAYS, DOES THE NATIONAL TOXICOLOGY
22 PROGRAM ATTEMPT TO ASCERTAIN A DOSE RESPONSE FOR THE
23 TUMORS IN THE ANIMALS?

24 A YES.

25 Q AND WHY IS THAT DONE?

26 A IT GIVES AN INDICATION OF THE AMOUNT OF
27 CHEMICAL THAT WOULD BE CAUSING THESE TUMORS. AND ONE
28 HOPES THAT IT WOULD BE IN A REASONABLY DOSE-RESPONSE

1 RELATIONSHIP BETWEEN THE TOP, MIDDLE, LOW -- AND LOWER
2 DOSES, SO THAT IT IS MORE USEFUL FOR PREDICTING OR
3 ALLOWING THE REGULATORY AGENCIES TO DO THEIR RISK
4 ASSESSMENTS.

5 Q OKAY. COULD YOU SHOW US WHAT YOU MEAN BY
6 THAT; IN OTHER WORDS, HOW A DOSE RESPONSE -- A TUMOR
7 DOSE RESPONSE IN ANIMALS RELATES TO HUMAN CANCER.

8 A YES.

9 YOUR HONOR, WOULD IT BE OKAY TO USE THE
10 BOARD?

11 THE COURT: YES.

12 THE WITNESS: THANK YOU.

13 THE TYPICAL DOSE RESPONSE IS THE NUMBER OF
14 TUMORS VERSUS INCREASING DOSE. SO INCREASING TUMORS AND
15 INCREASING DOSE.

16 AND IF WE CAN GO TO THE TOP DOSE -- AND THIS
17 IS THE TUMOR RATE THAT ONE SEES IN A BIOASSAY. AND THEN
18 TOWARD THE MIDDLE DOSE, ONE SEES, TYPICALLY, A LOWER
19 TUMOR RESPONSE; AND THEN AT THE LOW DOSE, AN EVEN LOWER
20 RESPONSE.

21 AND WHAT WE HOPE FOR IS THAT THESE WOULD BE
22 MONOTONIC OR LINEAR. THEN WE COULD EITHER EXTRAPOLATE
23 TO WHAT WOULD BE MORE RELEVANT TO THE HUMAN EXPOSURES --
24 AND THESE HAVE AN ARRAY OF SLOPES.

25 AND IN THIS CASE, WE CAN THEN HAVE A BETTER
26 IDEA -- IF THIS WAS ZERO, LET'S SAY -- TO INDICATE MUCH
27 MORE VALUE TO THE RISK ASSESSORS, IF WE HAVE SOME
28 REASONABLE LINEAR RESPONSE IN TUMOR RESULTS.

1 Q LET ME ASK YOU --

2 THE COURT: YOU'RE REFERRING TO A DIRECT DOSE OF
3 ACRYLAMIDE?

4 THE WITNESS: THIS IS CHEMICAL IN GENERAL; BUT
5 YES, ACRYLAMIDE, AS WELL.

6 THE COURT: AND IN THIS CASE, SPECIFICALLY WITH
7 ACRYLAMIDE, MIGHT IT DEAL WITH THE EFFECT OF DILUTION?
8 IN OTHER WORDS, WOULD ONE DROP OF ACRYLAMIDE IN A GALLON
9 OF WATER OR LITER HAVE ANY EFFECT ON THAT? OR ARE WE
10 JUST TALKING ABOUT DIRECT INJECTION OR INGESTION OF SOME
11 ITEM THAT HAS A PARTICULAR DOSAGE OF ACRYLAMIDE OR OTHER
12 CHEMICAL?

13 THE WITNESS: WELL, THIS IS A GENERIC RESPONSE.
14 BUT YOUR HONOR, IN THE CASE OF ACRYLAMIDE, THE LOWEST
15 EXPOSURE THAT WE SAW AN EFFECT WAS OUR LOWEST DOSE. AND
16 SO WE DON'T KNOW WHAT'S GOING TO HAPPEN HERE IF WE DID
17 THIS BIOASSAY AGAIN AT LOWER DOSES.

18 SO THIS IS 6 TO 8 PARTS PER MILLION. AND
19 JUST OFFHAND, IF ONE COULD SAY THAT 1 PART PER MILLION
20 EQUALS ABOUT -- WOULD BE ONE PERSON, LET'S SAY, WOULD
21 EQUAL ABOUT THE NUMBER OF PEOPLE AT TEN ROSE BOWL GAMES.

22 THE COURT: WHEN YOU SAY "1 PART PER MILLION," ARE
23 YOU TALKING ABOUT THE DOSAGE OR THE NUMBER OF PEOPLE
24 TESTED?

25 THE WITNESS: NO, THIS IS THE DOSES THAT THE
26 ANIMALS WERE EXPOSED TO.

27 THE COURT: SO YOU'RE SAYING LIKE 6 TO 8 PARTS PER
28 MILLION. SO IT WOULD BE 1 PART ACRYLAMIDE TO, LET'S

1 SAY -- OR EXCUSE ME, 6 TO 8 PARTS OF ACRYLAMIDE TO 1
2 MILLION PARTS OF WATER?

3 THE WITNESS: YES.

4 THE COURT: IS THAT WHAT YOU'RE TALKING ABOUT?

5 THE WITNESS: YES, SIR.

6 THE COURT: OKAY. THANK YOU.

7 Q BY MR. METZGER: I'D JUST LIKE TO MAKE SURE
8 WE'RE CLEAR HERE.

9 SO YOU HAVE -- ON THIS GRAPH THAT YOU'VE
10 DRAWN, YOU HAVE THREE DOSES: TOP, MIDDLE, AND LOW DOSE
11 THOSE ARE FOR THE ANIMALS; IS THAT CORRECT?

12 A YES, YES.

13 Q ALL RIGHT. AND THE LOWEST DOSE -- SINCE YOU
14 BROUGHT UP ACRYLAMIDE, THE LOWEST DOSE TESTED FOR
15 ACRYLAMIDE WAS AT THE RANGE OF 6 TO 8 PARTS PER MILLION?

16 A YES. THE REASON I SAY "6 TO 8" IS BECAUSE
17 ONE OF THESE IS FOR ACRYLAMIDE AND ONE IS FOR
18 GLYCIDAMIDE, THE MAJOR METABOLITE. THAT'S WHY IT'S 6 TO
19 8.

20 SORRY TO BE CONFUSING.

21 Q NO, THAT'S FINE. THANK YOU, DR. HUFF.

22 BUT AT THAT LOW-DOSE RANGE OF 6 TO 8 PARTS
23 PER MILLION IN ANIMALS, WAS THERE A TUMOR RESPONSE?

24 A YES.

25 Q ALL RIGHT. AND AT 6 TO 8 PARTS PER MILLION,
26 HOW FAR IS THAT LOW DOSE IN ANIMALS TO THE DIETARY LEVEL
27 OF ACRYLAMIDE CONSUMPTION, SAY, FOR EXAMPLE, AT -- FOR A
28 CUP OF COFFEE, AT 8 MICROGRAMS? CAN YOU RELATE THAT TO

1 US?

2 THE COURT: WHAT DOES THAT MEAN, "AT 8
3 MICROGRAMS"?

4 MR. METZGER: A SINGLE CUP OF COFFEE HAS ABOUT 8
5 MICROGRAMS OF ACRYLAMIDE.

6 THE COURT: I DON'T WANT YOU TO TESTIFY. I WANT
7 THE WITNESS TO TESTIMONY.

8 MR. METZGER: NO, THAT'S ALREADY -- THERE'S BEEN
9 TESTIMONY --

10 THE COURT: A CUP OF COFFEE, WITH WHATEVER
11 ACRYLAMIDE IS IN COFFEE.

12 MR. METZGER: RIGHT.

13 THE COURT: SO YOU'RE TELLING ME NOW IT'S 8
14 MICROGRAMS.

15 MR. METZGER: FROM THE EPA DOCUMENT WHICH HAS BEEN
16 JUDICIALLY NOTICED, IT'S ABOUT 8 MICROGRAMS. I THINK WE
17 DON'T EVEN DISPUTE THAT.

18 THE COURT: ALL RIGHT.

19 Q BY MR. METZGER: IN ANY EVENT, CAN YOU GIVE
20 US SOME INDICATION OF HOW THE LOWEST DOSE THAT WAS
21 TESTED IN ANIMALS FOR ACRYLAMIDE OR GLYCIDAMIDE RELATES
22 TO EITHER DIETARY ACRYLAMIDE EXPOSURE OR THE AMOUNT OF
23 ACRYLAMIDE FROM COFFEE?

24 A OFF THE TOP OF MY HEAD, WITHOUT DOING ANY OF
25 THIS, I WOULD SUSPECT THAT IT WOULD BE DOWN IN A MUCH
26 LOWER RANGE OF THIS DOSE-RESPONSE CURVE. I WOULD HAVE
27 TO DO THE CALCULATIONS OF COMPARING IT AND --

28 MR. SCHURZ: I'M GOING TO OBJECT AND MOVE TO

1 STRIKE. THIS CALLS FOR SPECULATION. THIS WITNESS JUST
2 TESTIFIED THAT HE DIDN'T DO THE WORK, AND SO HE'S
3 SPECULATING AS TO WHERE THIS IS GOING.

4 WE'VE NOW HAD TESTIMONY FROM MR. METZGER
5 WITH RESPECT TO CONCENTRATIONS OF ACRYLAMIDE IN COFFEE.
6 IT BEARS NO RESEMBLANCE TO THE LEVELS THAT THESE ANIMALS
7 WERE EXPOSED TO.

8 THE COURT: OKAY. OBJECTION SUSTAINED.

9 LET ME JUST ASK A QUESTION: IF YOU SAY THAT
10 THERE ARE 8 MICROGRAMS OF ACRYLAMIDE IN A CUP OF COFFEE,
11 WHAT WOULD BE THE RATIO OF ACRYLAMIDE TO THE LIQUID IN
12 THAT CUP OF COFFEE?

13 MR. METZGER: IF YOU'RE ABLE TO.

14 THE COURT: IF HE WANTS TO VOLUNTEER.

15 THE WITNESS: I DON'T KNOW.

16 THE COURT: ALL RIGHT.

17 MR. SCHURZ: WELL, I'LL VOLUNTEER, YOUR HONOR.

18 THE COURT: OKAY.

19 MR. SCHURZ: THE WAY WE'VE BEEN --

20 THE COURT: NOW WE HAVE EXPERT SCIENTISTS WHO ARE
21 SITTING AT COUNSEL TABLE.

22 MR. SCHURZ: JUST TO PROVIDE, BY WAY OF CONTEXT --
23 WHEN WE SPEAK ABOUT THE ACRYLAMIDE CONCENTRATIONS IN
24 COFFEE THAT ARE MEASURED BY THE FDA, WE'VE BEEN TALKING
25 ABOUT 3 PARTS PER BILLION. NOW WE'RE TALKING ABOUT 8
26 PARTS PER MILLION.

27 THE COURT: I HEARD THE NUMBER, "6 TO 8 PARTS PER
28 MILLION." THEN COUNSEL SAID "8 MICROGRAMS." I WANT TO

1 KNOW, HOW IS THAT -- PER WHAT OF LIQUID?

2 MR. METZGER: OKAY. SO -- WELL, A SINGLE CUP OF
3 COFFEE, AN EIGHT-OUNCE CUP, HAS ABOUT 8 MICROGRAMS OF
4 ACRYLAMIDE.

5 THE COURT: FINE. HOW MANY MICROGRAMS OF LIQUID?

6 MR. METZGER: I DON'T HAVE THAT CALCULATION RIGHT
7 NOW.

8 THE COURT: ALL RIGHT. SO WE'LL LEAVE THAT FOR
9 HOMEWORK FOR TOMORROW.

10 MR. METZGER: YEAH. THE NEXT WITNESS WILL ADDRESS
11 THAT.

12 Q BUT LET ME JUST ASK YOU CONCEPTUALLY,
13 BECAUSE THAT'S WHAT I WAS TRYING TO GET HERE, DR. HUFF:
14 IS THE CONCEPT THAT IN DOING -- IN ESTABLISHING A
15 DOSE-RESPONSE RELATIONSHIP FOR THE TUMOR RESPONSE IN
16 ANIMALS, USING THE THREE DOSE LEVELS IN ANIMALS, THAT
17 THAT INFORMATION HELPS THE REGULATORS, IN DOING
18 QUANTITATIVE CANCER RISK ASSESSMENTS, TO ASSESS THE
19 CARCINOGENIC RESPONSE -- OR TO ESTIMATE IT AT LOWER
20 DOSES IN HUMANS?

21 MR. SCHURZ: I'LL OBJECT AGAIN WITH RESPECT TO
22 LACKS FOUNDATION AS TO WITHIN THE SCOPE OF WHAT THIS
23 WITNESS HAS TESTIFIED TO.

24 THERE WERE NO OPINIONS RELATED TO THIS THAT
25 DR. HUFF OFFERED IN THE CONTEXT OF HIS DEPOSITION, SO
26 ALL OF THIS WOULD BE BRAND NEW.

27 THE COURT: THE QUESTION IS KIND OF CONVOLUTED.
28 START OVER WITH THE NEXT QUESTION.

1 MR. METZGER: SURE.

2 Q DR. HUFF, IN YOUR WORK AT NTP, HAVE
3 BIOSTATISTICIANS AND RISK ASSESSORS CONTACTED YOU FOR
4 INFORMATION REGARDING ANIMAL TUMOR DOSE RESPONSE TO
5 FACILITATE THEM -- TO ENABLE THEM TO DO THEIR
6 QUANTITATIVE CANCER RISK ASSESSMENTS?

7 A YES.

8 Q ALL RIGHT. AND DO YOU HAVE AN
9 UNDERSTANDING, BASED UPON THAT WORK THAT YOU DID, AS TO
10 WHAT USE THEY PUT THE ANIMAL TUMOR DOSE RESPONSE?

11 MR. SCHURZ: WELL, OBJECTION; CALLS FOR
12 SPECULATION.

13 THE COURT: SUSTAINED.

14 MR. METZGER: OKAY.

15 Q IN YOUR WORK, HAVE YOU USED TUMOR DOSE
16 RESPONSE IN EXTRAPOLATING TO HUMAN CANCER RISK?

17 A YES.

18 Q AND WHAT --

19 A QUALITATIVELY, YES.

20 Q OKAY. AND QUALITATIVELY, HOW -- WHAT DID
21 YOU CONCLUDE FROM THAT?

22 A WELL, IF YOU HAVE A REASONABLE DOSE
23 RESPONSE, WHICH MANY OF OUR STUDIES DO, YOU CAN
24 EXTRAPOLATE EITHER TO THIS POINT, OR HOWEVER THE CURVE
25 OR SLOPE IS TO ZERO, AND GET SOME INDICATION OF WHAT THE
26 TUMOR RESPONSE WOULD BE IF YOU HAD A LEVEL OF HUMAN
27 EXPOSURE ON THE DOSE LEVEL.

28 Q OKAY.

1 A DOSE AXIS.

2 Q ALL RIGHT. THANK YOU VERY MUCH.

3 NOW, HAVE YOU FORMED AN OPINION AS TO THE
4 RELEVANCE OF RARE OR UNCOMMON TUMORS IN ANIMALS TO THE
5 PREDICTIVE HUMAN CARCINOGENIC EFFECT?

6 A YES, I HAVE.

7 Q WHAT IS YOUR OPINION?

8 A MY OPINION IS THAT THESE ARE RELEVANT AND
9 ADD TO THE EVIDENCE OF LIKELY CANCER HAZARD TO HUMANS.

10 Q OKAY. HAVE YOU PUBLISHED TO THAT EFFECT?

11 A YES, I HAVE.

12 Q WHEN YOU SAY "WE," WHO ARE YOU REFERRING TO?

13 A MY COLLEAGUES AND MYSELF.

14 Q AT NTP?

15 A YES.

16 Q OKAY. ALL RIGHT.

17 NOW, HIS HONOR HAS HEARD ABOUT A GENOTOXIC
18 MECHANISM. I WANT TO ASK YOU ABOUT THAT.

19 HAVE YOU FORMED AN OPINION AS TO WHETHER
20 CHEMICALS THAT INDUCE CANCER IN ANIMALS THROUGH A
21 GENOTOXIC MECHANISM OF CARCINOGENESIS HAS PREDICTIVE
22 VALUE FOR HUMAN CANCER?

23 A YES.

24 Q WHAT IS YOUR OPINION?

25 A GENOTOXIC CHEMICALS UTILIZING THE SCOPE OF
26 THE KNOWN HUMAN CARCINOGENS ARE MORE CARCINOGENIC TO
27 HUMANS THAN THE TYPICAL NONGENOTOXIC CARCINOGENS IN
28 ANIMALS.

1 Q ALL RIGHT. IS THERE ANY RELEVANCE OF A
2 CHEMICAL BEING A GENOTOXIC CARCINOGEN FOR EXTRAPOLATION
3 TO HUMANS?

4 A YES.

5 Q WHAT IS THAT?

6 MR. SCHURZ: YOUR HONOR, WE WOULD OBJECT AS
7 CUMULATIVE AND REDUNDANT. WE'VE NOW HEARD FROM -- THIS
8 WILL BE THE FOURTH WITNESS WHO WILL BE TESTIFYING NOW
9 WITH RESPECT TO GENOTOXICITY, THE LINEAR MODEL, ISSUES
10 OF THIS NATURE.

11 WE'VE HEARD FROM DR. RAPPAPORT ON THIS
12 ISSUE. WE'VE HEARD FROM DR. SMITH ON THIS ISSUE. WE'VE
13 HEARD FROM DR. MELNICK ON THIS ISSUE. SO NOW WE'RE
14 HAVING A FOURTH WITNESS COVER THE SAME GROUND.

15 WE PROVIDED YOUR HONOR THIS MORNING WITH AN
16 OBJECTION IN WRITING BASED UPON THE DEMONSTRATIVES THAT
17 WE WERE PROVIDED, IN WHICH IT BECAME CLEAR THAT WE WERE
18 GOING TO BE ADDRESSING A RANGE OF ISSUES THAT HAD
19 PREVIOUSLY BEEN ADDRESSED BY THIS COURT AND FROM WHICH
20 THIS COURT HAS HEARD FROM MULTIPLE WITNESSES.

21 SO WE WOULD -- AT THIS TIME WE WOULD OBJECT
22 AND MOVE TO EXCLUDE FURTHER TESTIMONY AS REDUNDANT AND
23 DUPLICATIVE, UNDER 352.

24 THE COURT: OBJECTION OVERRULED.

25 Q BY MR. METZGER: DR. HUFF, YOU CAN ANSWER
26 THE QUESTION. DO YOU --

27 A YES, I REMEMBER THE QUESTION.

28 GENOTOXIC CHEMICALS ARE TYPICALLY MORE -- OR

1 GENOTOXIC CHEMICALS THAT WE HAVE STUDIED ARE MORE LIKELY
2 TO CAUSE CANCER AT EXTRAPOLATED LOWER DOSES BECAUSE WE
3 KNOW THAT A GENOTOXIC CHEMICAL HAS -- LACKS A THRESHOLD,
4 FOR INSTANCE. AND WE BELIEVE THAT AT ANY DOSE, THIS
5 GENOTOXIC CARCINOGEN WILL, IN FACT, IMPACT DNA
6 ALTERATIONS IN HUMANS.

7 Q ALL RIGHT. WHAT DO YOU MEAN WHEN YOU SAY
8 THAT IT HAS NO THRESHOLD?

9 A IT HAS NO THRESHOLD IN THE SENSE THAT NO
10 MATTER WHAT EXPOSURE ONE HAS, THAT THERE IS SOME
11 BIOLOGICAL RESPONSE HAPPENING.

12 Q AND WHAT IS THE BIOLOGICAL RESPONSE FOR A
13 GENOTOXIC CARCINOGEN?

14 A DNA DAMAGE, MAINLY.

15 Q OKAY. AND ALSO CANCER?

16 MR. SCHURZ: WELL, OVERBROAD, LACKS FOUNDATION,
17 CALLS FOR SPECULATION.

18 THE COURT: OVERRULED.

19 THE WITNESS: YES.

20 Q BY MR. METZGER: OKAY?

21 THE COURT: WHY WOULD GENOTOXIC CHEMICALS BE MORE
22 LIKELY TO CAUSE CANCER AT LOWER DOSES THAN HIGHER DOSES?

23 THE WITNESS: NO, SIR. WHAT I MEANT WAS THAT
24 GENOTOXIC CHEMICALS ARE MORE PRONE TO -- ARE KNOWN TO
25 INTERACT WITH DNA AT LOWER DOSES, BELOW THAT WHICH WE
26 SEE TUMORS IN THE ANIMALS; WHEREAS NONGENOTOXIC
27 CHEMICALS DON'T HAVE THAT SAME ATTRIBUTE, IF YOU WILL.

28 THE COURT: I DON'T UNDERSTAND WHAT YOU MEAN.

1 MAYBE YOU CAN CLARIFY IT. BECAUSE I THOUGHT I HEARD YOU
2 SAY THAT THEY HAVE STUDIED THAT GENOTOXIC CHEMICALS ARE
3 MORE LIKELY TO CAUSE CANCER AT EXTRAPOLATED LOWER DOSES.
4 WHAT DOES THAT MEAN?

5 THE WITNESS: WHAT I MEANT WAS, IT WOULD BE MORE
6 LIKELY TO ALSO -- IN ADDITION TO THE OTHER EXPOSURE
7 LEVELS, AT HIGHER EXPOSURE LEVELS, THAT THERE IS THE
8 CHANCE OF CAUSING CANCER AT LOWER LEVELS BECAUSE IT HAS
9 NO THRESHOLD, AND IT WILL INTERACT WITH DNA IN HUMANS AS
10 IT DOES IN ANIMALS, VERSUS A CHEMICAL THAT IS
11 NONGENOTOXIC.

12 THE COURT: NEXT QUESTION.

13 Q BY MR. METZGER: ALL RIGHT. HAVE YOU
14 ASSESSED, IN THIS CASE, THE CARCINOGENICITY IN ANIMALS
15 OF ACRYLAMIDE AND GLYCIDAMIDE?

16 A YES.

17 Q ALL RIGHT. COULD YOU PUT UP SLIDE 38,
18 PLEASE.

19 AND IS THIS A SLIDE THAT YOU PREPARED, DR.
20 HUFF?

21 A YES.

22 Q AND I SEE TWO CHEMICAL STRUCTURES. WILL YOU
23 TELL US WHAT THOSE REPRESENT.

24 A WELL, THE ONE ON THE LEFT IS THE -- THESE
25 ARE VERY MINIMAL STRUCTURES. THE ONE ON THE LEFT IS
26 ACRYLAMIDE. AND AS IT IS METABOLIZED, YOU SEE ON THE
27 RIGHT THAT THE TRIANGLE IS EPOXIDATION OF ACRYLAMIDE
28 INTO GLYCIDAMIDE. AND THIS IS THE MAJOR PATHWAY FOR

1 ACRYLAMIDE IN RATS AND MICE AND HUMANS.

2 Q OKAY. THE PATHWAY FOR WHAT?

3 A THE METABOLIC PATHWAY.

4 Q ALL RIGHT. AND YOU HAVE "CAS" NUMBERS. ARE
5 THOSE CHEMICAL ABSTRACT SERVICE REGISTRY NUMBERS FOR THE
6 CHEMICALS?

7 A YES.

8 Q ALL RIGHT. AND YOU THEN HAVE -- WELL, TELL
9 US ABOUT THE STRUCTURE OF THE CHEMICALS, HOW THEY'RE
10 SIMILAR OR HOW THEY COMPARE.

11 A YES. WELL, AS YOU CAN SEE, AT LEAST IN A
12 TWO-DIMENSIONAL STRUCTURE, THEY'RE VERY SIMILAR EXCEPT
13 FOR THE ATTACHMENT OF THE DOUBLE BOND ON THE LEFT OF THE
14 ACRYLAMIDE. IT CYCLIZES INTO AN EPOXIDE BY OXIDATION
15 AND RESULTS IN GLYCIDAMIDE.

16 Q OKAY. AND YOU HAVE LISTED "MW." WHAT IS
17 THAT?

18 A I'M SORRY. THAT'S THE MOLECULAR WEIGHT OF
19 ACRYLAMIDE AND THE MOLECULAR WEIGHT EMPIRICAL FORMULA
20 FOR GLYCIDAMIDE.

21 Q OKAY. SO BOTTOM LINE, WHAT IS -- ARE THESE
22 TWO CHEMICALS RELATED; AND IF SO, HOW?

23 A WELL, THEY'RE VERY RELATED, EXCEPT FOR THE
24 CHANGE DURING METABOLISM. SO THE STRUCTURES ARE QUITE
25 SIMILAR, WITH THE OBVIOUS DIFFERENCE IN THE EPOXIDE ON
26 THE RIGHT-HAND SIDE, WITH GLYCIDAMIDE.

27 Q OKAY. NOW, IN EXHIBIT 190 THAT YOU
28 PREPARED, WOULD YOU TELL US WHAT -- ESSENTIALLY WHAT,

1 OVERALL -- JUST OVERALL, GENERALLY -- WHAT IS IT THAT
2 YOU ATTEMPTED TO DO IN PREPARING EXHIBIT 190?

3 A WELL, THE THEORY IS THAT MOST OF THE
4 CARCINOGENICITY OF ACRYLAMIDE IS DUE TO THE EPOXIDATION
5 TO GLYCIDAMIDE.

6 AND WHAT I WANTED TO DO WAS TO COMPARE THE
7 TUMOR RESPONSES IN BOTH OF THESE STUDIES -- THAT WERE
8 DONE BY THE NATIONAL TOXICOLOGY PROGRAM, OF WHICH I HAD
9 NOTHING TO DO WITH THESE STUDIES; BUT MY OWN EVALUATION.

10 AND ONE CAN SEE THAT WITH ACRYLAMIDE, IT
11 HAS --

12 Q DOCTOR, HOLD ON JUST A MINUTE. WE'RE GOING
13 TO GET TO IT SO WE CAN SEE.

14 SO WHAT YOU WERE ATTEMPTING TO DO WAS
15 COMPARE THE TUMOR RESPONSE AND SITE RESPONSE OF THE
16 ANIMALS FROM THE NTP ACRYLAMIDE BIOASSAY, COMPARING THAT
17 TO THE NTP GLYCIDAMIDE BIOASSAY?

18 A YES, THAT'S RIGHT.

19 Q ALL RIGHT. NOW, WHEN WERE THESE BIOASSAYS
20 OF ACRYLAMIDE AND GLYCIDAMIDE DONE BY THE NATIONAL
21 TOXICOLOGY PROGRAM?

22 A WELL, BOTH OF THESE STARTED ROUGHLY FIVE
23 YEARS AGO. AND THERE WAS A DELAY BETWEEN THESE TWO OF
24 SEVERAL MONTHS, TO START THEM.

25 Q AND WHEN WERE THEY PUBLISHED?

26 A I BELIEVE THAT THE ACRYLAMIDE WAS PRESENTED
27 AT PEER REVIEW IN PUBLIC SESSION IN 2011 AND PUBLISHED
28 IN 2012. AND THE GLYCIDAMIDE WAS DONE IN 2012 AND HAS

1 NOT BEEN PUBLISHED YET AS FAR AS A TECHNICAL REPORT, BUT
2 THE REPORT THAT WAS PRESENTED TO PEER REVIEW IS
3 AVAILABLE.

4 Q AND WERE THESE STUDIES CANCER BIOASSAYS,
5 ACRYLAMIDE AND GLYCIDAMIDE OF THE NTP, DONE AFTER YOU
6 HAD LEFT THE NTP AND WERE WITH NIEHS?

7 A YES.

8 Q DID YOU REVIEW THE DATA FROM THESE CANCER
9 BIOASSAYS FOR THIS CASE?

10 A YES.

11 Q ALL RIGHT. COULD WE SHOW SLIDE 50.

12 ALL RIGHT. AND IT'S A LITTLE BIT DIFFICULT
13 TO SEE BECAUSE IT'S CUT OFF, BUT IS THE LEFT COLUMN THE
14 ANIMAL CANCER DATA FROM THE NTP STUDY FOR ACRYLAMIDE?

15 A YES.

16 Q IS THE MIDDLE COLUMN THE NTP DATA FOR THE
17 GLYCIDAMIDE BIOASSAY?

18 A YES.

19 Q AND WHAT IS THE RIGHT COLUMN?

20 A THIS IS A STUDY THAT WAS DONE PRIOR TO BOTH
21 OF THOSE, AND IT IS THE METHYL HYDROXY DERIVATIVE OF
22 ACRYLAMIDE. AND UNFORTUNATELY, IT WAS ONLY DONE IN
23 MICE, BUT I THOUGHT IT WOULD BE OF USE TO COMPARE THE
24 TUMOR PATTERNS WITH THAT CHEMICAL WITH THE OTHER TWO.

25 Q ALL RIGHT. SO WOULD YOU TELL US HOW YOU
26 WENT ABOUT STRUCTURING THIS TABLE.

27 A WELL, IN LOOKING AT THE FINDINGS OF THE
28 TUMOR PATTERNS, IT SEEMED STRIKING TO ME THAT THEY WERE

1 QUITE SIMILAR. SO I JUST THOUGHT TO PUT THESE IN A
2 SIDE-BY-SIDE AT THE SAME TUMOR SITE.

3 THAT'S WHY YOU SEE SOME BLANKS THAT -- FOR
4 INSTANCE, ON THE LEFT-HAND SIDE, THE FIRST BLANK:
5 UNDERNEATH "HEART," THERE'S A BLANK. AND THEN IN THE
6 RIGHT, FOR GLYCIDAMIDE, THERE'S LEUKEMIA. AND THAT'S
7 BECAUSE GLYCIDAMIDE INDUCED LEUKEMIAS IN RATS BUT
8 ACRYLAMIDE DID NOT.

9 Q ALL RIGHT. LET'S JUST DEFINE SOME TERMS.
10 YOU HAVE SOME ABBREVIATIONS. "MR" AND "FR," WHAT IS
11 THAT?

12 A EXCUSE ME. THE "FR" ARE FEMALE RATS, THE
13 "MR" ARE MALE RATS, "MM" IS MALE MICE, AND "FM" IS
14 FEMALE MICE.

15 Q ALL RIGHT. AND I SEE THAT YOU HAVE SOME
16 ASTERISKS ON THIS CHART. WOULD YOU TELL US WHAT THE
17 ASTERISKS MEAN.

18 A THE ASTERISKS INDICATE WHERE THERE IS
19 CONCORDANCE WITH THE TUMOR SITE, ACROSS. AND I DON'T
20 KNOW IF I EVEN NEEDED THAT, SINCE IT'S QUITE OBVIOUS.
21 YOU CAN SEE. I THINK I HAD THOSE ON BEFORE I MADE SURE
22 THAT THEY WERE SIDE BY SIDE.

23 Q ALL RIGHT. START -- AND THE TUMOR SITES ARE
24 LISTED ALPHABETICALLY?

25 A YES.

26 Q ALL RIGHT. THE VERY FIRST ONE IS BRAIN
27 TUMORS, FOR MALE RATS AND FEMALE RATS, FOR GLYCIDAMIDE;
28 IS THAT CORRECT?

1 A YES.

2 Q AND WHAT YOU'RE -- WHAT ARE YOU -- SO ARE
3 YOU SAYING THAT IN THE NTP GLYCIDAMIDE BIOASSAY, THEY
4 FOUND BRAIN CANCER IN THE MALE RATS AND THE FEMALE RATS?
5 IS THAT WHAT THIS MEANS?

6 A THAT'S WHAT THEY FOUND; RIGHT.

7 Q LET ME ASK YOU: IS THERE ANY SIGNIFICANCE
8 TO YOU, BASED UPON THE WORK THAT YOU'VE DONE, THAT
9 GLYCIDAMIDE INDUCED BRAIN TUMORS IN THE ANIMALS?

10 A WELL, YES. THERE ARE PUBLICATIONS THAT
11 INDICATE THAT THIS -- WHEN A CHEMICAL CAUSES TUMORS ON
12 THE CENTRAL NERVOUS SYSTEM, IT IS ALMOST INVARIABLY
13 GENOTOXIC. AND THAT'S THE CASE HERE.

14 Q AND IS THAT WHAT YOU FOUND FROM THE MULTIPLE
15 CANCER BIOASSAYS THAT YOU DIRECTED WHEN YOU WERE AT NTP?

16 A YES.

17 Q ALL RIGHT. AND ARE THERE ARTICLES WHICH
18 MAKE THAT CONCLUSION, THAT A SO-CALLED NEUROCARCINOGEN
19 IS ALSO A GENOTOXIC CARCINOGEN?

20 A YES.

21 Q COULD YOU TELL US ONE OF THOSE.

22 A YES. I HAVEN'T DONE THAT, BUT DR. JERRY
23 RICE HAS PUBLISHED A PAPER ON ACRYLAMIDE WHERE HE
24 SPECIFIES -- OR WHERE HE SPECIFICALLY SAYS THAT.

25 Q ALL RIGHT. AND THEN THE NEXT CANCER IS THE
26 CLITORAL GLAND, WHICH WAS APPARENTLY FOUND IN BOTH --
27 FOR BOTH ACRYLAMIDE AND GLYCIDAMIDE IN THE FEMALE RATS?

28 A THAT'S CORRECT.

1 Q ALL RIGHT. WHAT IS THE EPIDIDYMIS?

2 A THAT'S PART OF THE TESTICULAR SYSTEM THAT IS
3 ORGAN SPECIFIC WITH RESPECT TO THE TESTES AND THE
4 EPIDIDYMIS.

5 Q ALL RIGHT. SO TUMORS OF THE EPIDIDYMIS WERE
6 FOUND FOR BOTH ACRYLAMIDE AND GLYCIDAMIDE IN MALE RATS?

7 A YES.

8 Q ALL RIGHT. THE NEXT ONE IS FORESTOMACH.
9 WHAT IS A FORESTOMACH?

10 A RODENTS HAVE, IF YOU WILL, A DOUBLE STOMACH.
11 THE FORESTOMACH IS THE FIRST, AND THEN IT LEADS INTO THE
12 GLANDULAR STOMACH, AND THEN THROUGH THE INTESTINAL
13 TRACT.

14 Q SO WHAT DID YOU FIND FOR ACRYLAMIDE AND
15 GLYCIDAMIDE AS FAR AS FORESTOMACH TUMORS FROM THE NTP
16 STUDIES?

17 A WELL, THERE WAS CONCORDANCE TOTALLY WITH
18 RESPECT TO MALE MICE AND FEMALE MICE AND BOTH CHEMICALS.
19 AND GLYCIDAMIDE ALSO HAD FORESTOMACH TUMORS IN FEMALE
20 RATS.

21 Q ALL RIGHT. THE NEXT TUMOR YOU'VE LISTED IS
22 THE HARDERIAN GLAND. WHAT IS THIS?

23 A THAT'S A TEAR DUCT GLAND IN THE ANIMALS
24 THAT -- OR A SEBACEOUS GLAND IN THE EYE OF ANIMALS THAT
25 HUMANS HAVE A COUNTERPART IN THEIR TEAR DUCT GLANDS.

26 Q AND WHAT DID YOU FIND REGARDING THE
27 HARDERIAN GLAND TUMORS?

28 A THESE HAD THE HIGHEST TUMOR RATE OF THE REST

1 OF THE TUMOR SITES. AND THERE WAS CONCORDANCE NOT ONLY
2 WITH THESE STUDIES BUT WITH EARLIER STUDIES THAT AREN'T
3 ON THIS CHART.

4 Q AND WERE ALL OF THOSE TUMORS FOUND IN MICE?

5 A YES.

6 Q I SEE THAT THAT WAS ALSO FOUND IN THE NTP
7 STUDY FOR METHYL -- HOW DO YOU SAY THAT?

8 A METHYLOLACRYLAMIDE.

9 Q ALL RIGHT. AND THAT'S ANOTHER STRUCTURALLY
10 SIMILAR CHEMICAL?

11 A YES. I DON'T HAVE THE STRUCTURE ON THAT UP
12 WITH THESE.

13 Q THE NEXT TUMOR SITE THAT YOU HAVE LISTED IS
14 HEART, FOR RATS. AND WOULD YOU TELL US WHAT YOU FOUND
15 FOR THAT.

16 A THIS IS ONE OF THE UNUSUAL TUMORS -- IN
17 ADDITION TO THE HARDERIAN GLAND; BUT THIS IS AN UNUSUAL
18 TUMOR IN RATS. AND IT WAS QUITE SIGNIFICANT TO FIND
19 THAT THIS WAS IN BOTH ACRYLAMIDE AND IN GLYCIDAMIDE, YET
20 IT WASN'T IN THE FEMALE RAT FOR THE GLYCIDAMIDE.

21 Q OKAY. YOU HAVE NEXT LISTED LEUKEMIA FOR
22 GLYCIDAMIDE. TELL US WHAT THAT -- WHAT WAS FOUND.

23 A WELL, HERE AGAIN, THERE'S SOME CONCORDANCE
24 WITH BOTH SEXES OF RAT. THIS DID NOT OCCUR IN MICE, AND
25 IT DID NOT OCCUR IN ACRYLAMIDE. SO THIS LENDS SOME
26 CREDIBILITY TO THE NOTION THAT -- OR TO THE REALIZATION
27 THAT GLYCIDAMIDE IS THE ULTIMATE CARCINOGEN OF THESE
28 TWO, YET NOT TOTALLY SO.

1 Q OKAY. NEXT YOU HAVE LUNG TUMORS, WHICH
2 WAS -- THOSE WERE FOUND FOR ALL THREE CHEMICALS?

3 A YES. AGAIN, ONLY IN MICE.

4 Q OKAY. AND NEXT IS MAMMARY GLAND, WHICH WAS
5 FOUND WHERE?

6 A THIS IS FOUND IN FEMALE RAT, FEMALE MICE.
7 IT'S VERY RARE TO FIND A MAMMARY GLAND TUMOR IN MALES.
8 SO THE SIGNIFICANCE HERE IS THAT IT WAS IN BOTH SPECIES
9 OF FEMALE ANIMALS, SO IT OBVIOUSLY GOT OUR ATTENTION.

10 Q WHY SO?

11 A BECAUSE, NUMBER ONE, IT'S CORRELATIVE
12 BETWEEN THE TWO CHEMICALS THAT NTP TESTED. AND NUMBER
13 TWO, IT WAS CONSIDERABLY SIGNIFICANT IN FEMALES OF BOTH
14 SPECIES, WHICH OBVIOUSLY -- WHICH LENDS CREDIBILITY TO
15 THIS FINDING.

16 Q OKAY. ALSO, BOTH CHEMICALS INDUCED ORAL
17 MUCOSA TUMORS IN RATS?

18 A YES. WELL, FEMALE RATS FOR ACRYLAMIDE AND
19 BOTH SEXES FOR GLYCIDAMIDE.

20 AND THE INTERESTING THING HERE IS THAT WE
21 SPOKE OF THE FORESTOMACH BEFORE, WHICH IS SIMILAR TO --
22 OR SORRY, THE SAME CELLULAR TYPE AS THE ORAL CAVITY AND
23 THE ESOPHAGUS OF HUMANS. SO HERE WE HAVE FORESTOMACH
24 AND ORAL MUCOSA IN RATS, WHICH HUMANS DO HAVE.

25 Q OKAY. WHAT ABOUT THE OVARY?

26 A AGAIN, IT WAS -- WE DIDN'T FIND THAT IN THE
27 GLYCIDAMIDE, BUT WE FOUND IT IN THE ACRYLAMIDE AND THE
28 HYDROXYMETHYL ACRYLAMIDE.

1 Q NEXT YOU HAVE PANCREATIC ISLETS IN MALE
2 RATS. WHAT WAS THAT?

3 A THAT WAS INTERESTING BECAUSE ONCE YOU HAVE
4 THE EPOXIDE FORMED, FOR SOME REASON, WE DIDN'T SEE IT
5 IN -- OR NTP DID NOT SEE IT IN THE GLYCIDAMIDE. SO THAT
6 INDICATES TO US THAT ACRYLAMIDE HAS SOME CARCINOGENIC
7 ACTIVITY ON ITS OWN, WITHOUT METABOLIC -- WHAT WE CALL
8 METABOLIC ACTIVATION.

9 AND FURTHERMORE, THERE IS SOME -- ACCORDING
10 TO IARC, THERE'S SOME -- BUT NOT SUFFICIENT -- EVIDENCE
11 THAT THE PANCREAS MAY BE A TARGET SITE IN HUMANS.

12 Q OKAY. THE NEXT YOU HAVE IS SKIN. TELL US
13 WHAT YOU CONCLUDED ABOUT THAT.

14 A WELL, IT'S NOT A TOTAL CORRELATION, BUT IT
15 IS A CORRELATION. AND WHAT'S INTERESTING ABOUT THIS IS
16 THAT YOU DON'T SEE TUMORS OF THE SKIN, ORDINARILY, WHEN
17 YOU GIVE CHEMICALS ORALLY.

18 IF YOU PAINT IT ON THEIR SKIN -- WHICH THERE
19 ARE OTHER STUDIES ON ACRYLAMIDE THAT HAVE SKIN PAINTED,
20 WHICH ARE CARCINOGENIC, AS WELL. SO THAT WAS THE
21 SIGNIFICANCE HERE: AN ORALLY-ADMINISTERED CHEMICAL
22 CAUSED TUMORS OF THE SKIN.

23 Q OKAY. NEXT YOU HAVE THE TESTES IN MALE
24 RATS?

25 A YES, WE SPOKE OF THAT WITH THE EPIDIDYMISS.
26 THESE ARE MESOTHELIOMAS. AND AGAIN, THESE ARE NOT --
27 THESE ARE UNCOMMON TUMORS IN THE RATS USED IN THESE
28 STUDIES.

1 Q NEXT IS THE THYROID GLAND. WHAT WERE YOUR
2 FINDINGS AND CONCLUSIONS REGARDING THAT?

3 A WELL, THIS WAS SIGNIFICANT, AGAIN, BECAUSE
4 IT CAUSED THYROID GLAND TUMORS IN BOTH SEXES OF RATS.
5 AND IT TOO IS NOT A COMMON TUMOR.

6 WE DIDN'T -- AS YOU SEE FROM ITS ABSENCE, WE
7 DIDN'T SEE THIS IN MICE. THEY SEEMED SOMEHOW TO BE MORE
8 RESISTANT TO CHEMICALS THAT INDUCED THYROID GLAND
9 TUMORS.

10 Q OKAY. AND LASTLY IS THE TONGUE?

11 A YES. AND THAT IT GOES ALONG WITH THE ORAL
12 MUCOSA AND THE FORESTOMACH, SO IT'S KIND OF A CONTINUUM;
13 AND AGAIN, SUBSTANTIATES THAT TARGET SITE OF THE CELLS.

14 Q ALL RIGHT. SO ONCE YOU PUT THIS TUMOR
15 CONCORDANCE TABLE TOGETHER, WHAT DID YOU CONCLUDE
16 REGARDING TUMOR SITE CONCORDANCE FOR ACRYLAMIDE AND
17 GLYCIDAMIDE?

18 A WELL, FIRST OF ALL, WE WERE QUITE TAKEN WITH
19 THE NUMBER OF TUMOR SITES IN THESE ANIMALS AT THESE --
20 EVEN AT THESE 6 PART AND 8 PART PER MILLION DOSES, WHICH
21 IS NOT TYPICAL OF A LONG-TERM STUDY. ONE DOESN'T
22 ORDINARILY SEE THIS NUMBER OF TUMOR SITES. SO THIS SAYS
23 TO US THAT THIS IS A REALLY POTENT CARCINOGEN IN THESE
24 ANIMALS.

25 Q AND WHAT DID YOU CONCLUDE REGARDING THE
26 CONCORDANCE BETWEEN ACRYLAMIDE AND GLYCIDAMIDE?

27 A WELL, THIS TOO WAS SPECTACULAR AND LED TO
28 THE HYPOTHESIS THAT GLYCIDAMIDE IS THE ULTIMATE

1 CARCINOGEN; EXCEPT, AS I SAID, THAT ACRYLAMIDE HAS SOME
2 OF ITS CARCINOGENIC ACTIVITY ON ITS OWN.

3 SOME PEOPLE HAVE CALLED IT A PRO-CARCINOGEN,
4 WHICH MEANS IT NEEDS METABOLISM TO BE A CARCINOGEN, BUT
5 THAT'S NOT SUBSTANTIATED BY OUR DATA. IT SHOWS THAT
6 THERE IS CARCINOGENIC ACTIVITY IN ACRYLAMIDE BY ITSELF.

7 AND IT'S HARD TO SEPARATE OUT WHAT IS DUE TO
8 GLYCIDAMIDE OR WHAT'S DUE TO ACRYLAMIDE. SO I MEAN,
9 THEY'RE BOTH PRETTY POTENT CARCINOGENS.

10 Q ALL RIGHT. HAVE YOU FORMED AN OPINION AS TO
11 THE RELEVANCE OF ACRYLAMIDE AND GLYCIDAMIDE PRODUCING
12 TUMORS AT MULTIPLE SITES WITH RESPECT TO HUMAN
13 CARCINOGENICITY?

14 A WELL, NOT ONLY THE MULTIPLE SITES, BUT IT
15 CAUSES TUMORS IN BOTH SEXES OF BOTH SPECIES. IT CAUSES
16 TUMORS IN DIFFERENT STRAINS OF ANIMALS. AND IT GIVES US
17 CONFIDENCE THAT THIS QUITE CLEARLY MAY BE A CANCER
18 HAZARD TO HUMANS FROM THESE CHEMICALS.

19 Q ALL RIGHT. NOW, LET ME ASK YOU ABOUT THE
20 HARDERIAN GLAND. I THINK YOU INDICATED THAT THE
21 STRONGEST TUMOR RESPONSE WAS FOUND IN THE HARDERIAN
22 GLAND?

23 A YES, SIR, AS I RECALL.

24 Q ALL RIGHT. AND IS IT CORRECT THAT HUMANS DO
25 NOT ACTUALLY HAVE A HARDERIAN GLAND BUT RATHER HAVE
26 SOMETHING SIMILAR?

27 A YES.

28 Q ALL RIGHT. WHAT IS THE SIGNIFICANCE TO YOU

1 OF A STRONG TUMOR RESPONSE FOR ACRYLAMIDE AND
2 GLYCIDAMIDE IN THE HARDERIAN GLAND OF THE ANIMALS -- OF
3 THE MICE, WITH RESPECT TO HUMAN CARCINOGENICITY?

4 A WELL, IT'S A CARCINOGENIC RESPONSE,
5 REGARDLESS OF THE TARGET SITE. FOR INSTANCE, BENZENE
6 CAUSES TUMORS OF THE ZYMBAL GLAND IN ANIMALS, WHICH --
7 WE ONLY HAVE SEBACEOUS GLANDS IN OUR EAR, WHERE THE
8 ZYMBAL GLAND RESIDES IN RATS AND MICE.

9 SO THEY'RE RELEVANT TO -- IN MY OPINION, TO
10 RISK ASSESSMENT, REGARDLESS OF THE ONE-TO-ONE
11 CORRELATION BETWEEN RODENTS AND HUMANS.

12 Q HAVE ANY HUMAN CARCINOGENS BEEN FOUND TO
13 CAUSE HARDERIAN GLAND TUMORS IN ANIMALS?

14 A YES.

15 Q ALL RIGHT. NOW, WHAT DID YOU CONCLUDE
16 REGARDING THE INCIDENCE OF TUMOR RESPONSE FROM
17 ACRYLAMIDE AND GLYCIDAMIDE?

18 A WELL, I DON'T -- MY CONCLUSION WAS THAT IT
19 WAS A SPECTACULAR RESPONSE, BUT I DON'T HAVE MEMORY OF
20 ALL THE NUMBERS INVOLVED. BUT LIKE I SAID, HARDERIAN
21 GLAND WAS -- 70, 85 PERCENT OF THE ANIMALS HAD THESE
22 TUMORS, WHICH IS UNHEARD OF IN CANCER BIOASSAYS.

23 Q OKAY. AND OF WHAT RELEVANCE IS IT TO YOU,
24 REGARDING HUMAN CARCINOGENICITY, THAT BOTH ACRYLAMIDE
25 AND GLYCIDAMIDE PRODUCED A HIGH INCIDENCE OF TUMORS IN
26 THE ANIMALS?

27 A WELL, AGAIN, IT GIVES US CONFIDENCE THAT
28 THIS WOULD BE CARCINOGENIC TO HUMANS.

1 AND MIND YOU, THESE FINDINGS WERE SUBSEQUENT
2 TO BOTH IARC CALLING THIS A "PROBABLE HUMAN CARCINOGEN"
3 AND NTP CALLING THIS A "REASONABLY ANTICIPATED TO BE A
4 CARCINOGEN." AND THESE DATA ARE SUPPLEMENTAL TO THAT,
5 WHICH CERTAINLY SOLIDIFIES THEIR CONCLUSION.

6 AND IN FACT, IARC HAS PUT THIS ON THEIR
7 PRIORITY LIST TO REEVALUATE, GIVEN ALL THAT HAS BEEN
8 PRESENTED AT THIS HEARING -- BOTH EPIDEMIOLOGICALLY,
9 MECHANISTICALLY, AND TUMORIGENICALLY.

10 Q ALL RIGHT. SO --

11 MR. SCHURZ: I'M GOING TO OBJECT AND MOVE TO
12 STRIKE AS LACKS FOUNDATION AS TO THIS WITNESS'S
13 KNOWLEDGE AS TO WHAT IARC'S THINKING IS; AND
14 SPECIFICALLY, HIS STATEMENT THAT, "GIVEN ALL THAT HAS
15 BEEN PRESENTED AT THIS HEARING."

16 I THINK WE CAN BE ABSOLUTELY CERTAIN IARC
17 DOESN'T HAVE A CLUE WHAT'S BEEN PRESENTED AT THIS
18 HEARING.

19 THE COURT: ALL RIGHT. THAT'S OVERRULED.

20 Q BY MR. METZGER: DR. HUFF, WHAT DID YOU NOTE
21 REGARDING THE ANIMAL WHAT BIOASSAYS FOR ACRYLAMIDE AND
22 GLYCIDAMIDE WITH RESPECT TO DOSE RESPONSE?

23 A WELL, IN MANY CASES -- I CAN'T NAME WHICH
24 ONES, BUT IN MANY CASES THESE HAD A VERY NICE THREE-
25 LEVEL DOSE RESPONSE FOR THEIR TUMOR PATTERNS. THEIR
26 INCIDENCE RATES WERE QUITE NICELY PLOTTED ON A SIMPLE
27 TUMOR RESPONSE VERSUS DOSE.

28 Q OKAY. WERE THOSE RESPONSES ESSENTIALLY

1 LINEAR?

2 A FOR SOME, YES.

3 Q OKAY. AND WHAT IS THE SIGNIFICANCE OF THAT
4 TO YOU WITH RESPECT TO THE PREDICTION OF HUMAN CANCER OR
5 RISK ASSESSMENTS OF HUMAN CARCINOGENICITY?

6 A WELL, IT SPEAKS TO THE GENOTOXICITY OF BOTH
7 OF THESE -- THE PARENT COMPOUND AND THE METABOLITE AND
8 IS MORE LIKELY TO GIVE SOME INDICATION -- OR TO GIVE
9 INDICATION OF WHAT ONE WOULD EXPECT AT LOWER DOSES.

10 Q OKAY. WHICH OF THE TUMORS THAT ARE LISTED
11 IN YOUR TABLE DO YOU CONSIDER TO BE RARE TUMORS?

12 A WELL, I THINK THE CLITORAL GLAND, THE
13 EPIDIDYMISS, THE FORESTOMACH, THE HARDERIAN, THE HEART.
14 THE SKIN, IN AN ORAL STUDY. THE TESTES AND THE THYROID
15 GLAND.

16 Q OKAY. AND WHAT IS THE SIGNIFICANCE TO YOU,
17 WITH RESPECT TO HUMAN CARCINOGENIC POTENTIAL, THAT
18 ACRYLAMIDE AND GLYCIDAMIDE INDUCED SO MANY RARE TUMORS
19 IN THE ANIMALS?

20 A IT AGAIN ADDS TO THE ABILITY TO EXTRAPOLATE
21 THAT THIS WOULD LIKELY BE CARCINOGENIC IN HUMANS.

22 Q OKAY. NOW, YOU ALSO DID MENTION THAT THESE
23 TWO CHEMICALS INDUCED TUMORS IN ANIMALS AT DOSES OF
24 ABOUT 6 AND 8 PARTS PER MILLION. WHAT IS THE
25 SIGNIFICANCE OF THAT LOW DOSE TUMORIGENICITY IN THE
26 ANIMALS TO YOU WITH RESPECT TO HUMAN CARCINOGENICITY?

27 A WELL, AGAIN, IT GIVES US CERTAINLY MORE
28 EVIDENCE THAT AT LOWER DOSES --

1 BECAUSE WE DON'T KNOW IN THESE STUDIES THE
2 LOWEST DOSE THAT THIS CAUSED THE CARCINOGENIC RESPONSE.
3 AND IN FACT, IF THE STUDY WERE REDONE, WE WOULD FIND
4 THAT OUT; BUT WE DON'T KNOW WHAT WOULD HAPPEN AT LOWER
5 DOSES.

6 BUT CONSIDERING THAT THIS IS GENOTOXIC,
7 CONSIDERING THAT THIS HAS DOSE-RESPONSE RELATIONSHIPS,
8 CONSIDERING THAT WE DON'T KNOW WHAT'S GOING TO HAPPEN AT
9 LOWER THAN 6 PARTS PER MILLION, IT'S CERTAINLY LOGICAL,
10 IN MY VIEW, TO THINK THAT THIS WOULD BE A CARCINOGENIC
11 HAZARD TO HUMANS AT LOWER DOSES THAN 6 PARTS PER
12 MILLION.

13 MR. METZGER: OKAY.

14 THE COURT: WE'RE GOING TO RECESS AT THIS TIME.
15 WE'LL RESUME AT 2:30.

16 (AT 12:17 P.M., A LUNCH RECESS WAS TAKEN
17 UNTIL 1:30 P.M. OF THE SAME DAY.)

18 (TRANSCRIPT CONTINUES ON PAGE 151.)

19

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EXHIBIT “D”

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SUPERIOR COURT OF THE STATE OF CALIFORNIA

FOR THE COUNTY OF LOS ANGELES

DEPARTMENT 323

HON. ELIHU M. BERLE, JUDGE

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

PLAINTIFF,)

VS.)

CASE NO.
BC435759

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

DEFENDANTS.)

AND CONSOLIDATED ACTION.)

REPORTER'S TRANSCRIPT OF TRIAL PROCEEDINGS

WEDNESDAY, OCTOBER 22, 2014

AFTERNOON SESSION

APPEARANCES:

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CCROLA JOB
NO. 114686

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

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I N D E X

WEDNESDAY, OCTOBER 22, 2014 (P.M.)

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1 CASE NUMBER: BC435759
2 CASE NAME: CERT VS. STARBUCKS
3 LOS ANGELES, CALIFORNIA WEDNESDAY, OCTOBER 22, 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: BACK ON THE RECORD IN THE CASE OF CERT
12 VERSUS STARBUCKS. ALL COUNSEL ARE PRESENT. DR. HUFF IS
13 ON THE STAND.

14 DR. HUFF, YOU UNDERSTAND YOU ARE STILL UNDER
15 OATH?

16 THE WITNESS: YES, SIR.

17 THE COURT: PLEASE RESTATE YOUR NAME FOR THE
18 RECORD.

19 THE WITNESS: JAMES EDWARD HUFF, H-U-F-F.

20 THE COURT: THANK YOU. MR. METZGER WAS INQUIRING.
21 COUNSEL WILL PROCEED.

22 MR. METZGER: THANK YOU, YOUR HONOR.
23

24 DIRECT EXAMINATION

25

26 BY MR. METZGER:

27 Q DR. HUFF, I WOULD LIKE TO NOW TURN TO THE
28 TOPIC OF THE USE OF ANIMAL DATA FOR HUMAN CANCER RISK

1 ASSESSMENT.

2 HAVE YOU FORMED AN OPINION AS TO WHETHER
3 ANIMAL CANCER BIOASSAY DATA FROM RATS AND MICE SHOULD BE
4 USED TO ASSESS HUMAN CANCER RISK?

5 A YES.

6 Q WHAT IS YOUR OPINION?

7 A I THINK THAT THESE ARE VALID USE OF ANIMAL
8 BIOASSAY DATA FOR RISK ASSESSMENT.

9 Q DO ANY INTERNATIONAL AGENCIES OR
10 GOVERNMENTAL BODIES HOLD THAT VIEW THAT ANIMAL CANCER
11 BIOASSAYS SHOULD BE USED FOR HUMAN CANCER RISK
12 ASSESSMENT?

13 A YES.

14 Q WHAT ARE THEY?

15 A IN PARTICULAR, THE INTERNATIONAL AGENCY FOR
16 RESEARCH ON CANCER HAS BEEN DOING THIS SINCE THE EARLY
17 70S, AND HAS CONTINUOUSLY ENDORSED THIS PROCEDURE AND
18 CONCEPT. I DON'T KNOW OF ANY INTERNATIONAL OR NATIONAL
19 ORGANIZATIONS THAT DOESN'T CONSIDER THESE RELEVANT TO
20 HUMAN RISK ASSESSMENT, INCLUDING N.T.P., F.D.A. AND
21 E.P.A., ET CETERA.

22 Q NOW, HAVE YOU FORMED AN OPINION AS TO
23 WHETHER DATA FROM ALL ANIMAL TUMOR SITES SHOULD BE
24 EVALUATED FOR PURPOSES OF HUMAN CANCER RISK ASSESSMENT?

25 A YES.

26 Q WHAT IS YOUR OPINION?

27 A I BELIEVE THAT SINCE THIS IS A CARCINOGENIC
28 RESPONSE FROM THE CHEMICAL IN QUESTION OR A CHEMICAL IN

1 QUESTION, THAT THE TRUER EVALUATION OF THE POTENTIAL RISK
2 TO HUMANS IS NOT ONLY TO SINGLE OUT ONE TARGET SITE, BUT
3 TO COMBINE THEM TO GIVE AN OVERALL VIEW OR AN ESTIMATE OF
4 THE TRUE CARCINOGENIC RISK FOR THAT PARTICULAR CHEMICAL.

5 Q DO YOU HAVE AN OPINION AS TO WHETHER THOSE
6 SITES IN ANIMALS THAT SHOW CLEAR OR SUFFICIENT EVIDENCE
7 OF CARCINOGENICITY SHOULD ACTUALLY BE USED IN THE HUMAN
8 CANCER RISK ASSESSMENT?

9 A YES.

10 Q WHAT IS YOUR OPINION?

11 A I BELIEVE THE MOST SIGNIFICANT RESULT IS THE
12 PRIME TUMOR SITE TO USE IN RISK ASSESSMENT PROCEDURES.

13 Q DO YOU MEAN BY THAT THOSE SITES THAT SHOW
14 CLEAR OR SUFFICIENT EVIDENCE?

15 A YES. CLEAR OR SUFFICIENT, YES.

16 Q DO ANY AGENCIES OR INTERNATIONAL BODIES
17 SHARE THAT ASSESSMENT?

18 A YES, THEY DO.

19 Q WHAT ARE THEY?

20 A IARC AGAIN AND N.T.P. F.D.A., IN
21 PARTICULAR, UTILIZES CLEAR EVIDENCE OR SUFFICIENT
22 EVIDENCE IN THEIR EVALUATIONS.

23 Q OKAY. IS THIS AN OPINION THAT YOU HAVE
24 EXPRESSED AND PUBLISHED BEFORE THIS CASE?

25 A YES.

26 Q DO YOU HAVE AN OPINION WHETHER TUMORS
27 INDUCED IN ORGANS THAT ARE UNIQUE TO RODENTS, SUCH AS THE
28 FORE-STOMACH, THE HARDERIAN GLAND, SHOULD BE INCLUDED IN

1 HUMAN CANCER RISK ASSESSMENT?

2 A YES.

3 Q WHAT IS YOUR OPINION?

4 A I -- REGARDLESS OF THE TUMOR SITE, WHETHER
5 IT IS UNIQUE TO RODENTS OR NOT, I BELIEVE THAT THE
6 RESPONSE OF THESE ORGANS TO A CHEMICAL ARE INDICATIVE OF
7 A CARCINOGENIC EFFECT AND SHOULD NOT BE IGNORED WHEN
8 ATTEMPTING TO PROTECT THE PUBLIC HEALTH FROM A CANCER
9 HAZARD.

10 Q IS THAT AN OPINION THAT YOU PUBLISHED BEFORE
11 THIS CASE?

12 A I HAVE.

13 Q ARE THERE OTHER AUTHORITIES THAT SUPPORT
14 THAT CONCLUSION?

15 A YES.

16 Q WHAT ARE THEY?

17 A IARC, N.T.P., E.P.A., TO NAME THREE.

18 Q OKAY. DO YOU HAVE AN OPINION WHETHER
19 ANALYSES OF ALL TUMOR SITES IN ANIMALS SHOULD BE COMBINED
20 FOR ESTIMATING HUMAN CANCER RISK?

21 A YES.

22 Q WHAT IS YOUR OPINION?

23 A MY OPINION IS THAT THE MORE TRUE RESPONSE IN
24 AN ANIMAL IS GATHERED BY COMBINING ALL TUMOR SITES TO GET
25 A TRUER ESTIMATE OF THE TOTAL CARCINOGENIC EFFECT OF A
26 CHEMICAL.

27 Q IS THAT AN OPINION THAT YOU HAVE HELD BEFORE
28 THIS CASE?

1 A YES.

2 Q IS IT AN OPINION THAT YOU HAVE PUBLISHED?

3 A YES.

4 Q IS IT AN OPINION THAT OTHERS HAVE PUBLISHED?

5 A YES.

6 Q DO YOU HAVE AN OPINION AS TO WHETHER BENIGN
7 TUMORS INDUCED BY CHEMICALS IN ANIMALS ARE RELEVANT FOR
8 JUDGING HUMAN CARCINOGENICITY?

9 A YES.

10 Q WHAT IS YOUR OPINION?

11 A SINCE MOST CARCINOGENS RARELY INDUCE ONLY
12 BENIGN TUMORS, AND SINCE BENIGN TUMORS TYPICALLY PROGRESS
13 TO MALIGNANCY, I HAVE COME TO THE CONCLUSION THAT THESE
14 ARE RELEVANT TO HUMAN CANCER RISK ASSESSMENT.

15 Q IS THAT AN OPINION THAT YOU HAVE PUBLISHED
16 BEFORE THIS CASE?

17 A YES.

18 Q IS THAT AN OPINION THAT IS HELD BY
19 GOVERNMENT AND INTERNATIONAL AGENCIES?

20 A YES.

21 Q WHAT ARE THEY?

22 A N.T.P., THE NATIONAL TOXICOLOGY PROGRAM, THE
23 FOOD AND DRUG ADMINISTRATION, ENVIRONMENTAL PROTECTION
24 AGENCY, INTERNATIONAL AGENCY FOR RESEARCH ON CANCER.
25 THE -- AND OTHERS.

26 Q DO YOU HAVE AN OPINION AS TO WHETHER TUMORS
27 THAT ARE INDUCED IN ANIMALS ONLY AT THE HIGHEST
28 EXPERIMENTAL DOSE SHOULD BE INCLUDED IN AN ASSESSMENT OF

1 HUMAN CANCER RISK?

2 A YES.

3 Q WHAT IS YOUR OPINION?

4 A FIRST, THIS RARELY HAPPENS THAT THERE IS NOT
5 SOME SUPPORTING EVIDENCE AT THE LOWER DOSES. WHETHER
6 THEY ARE SIGNIFICANT OR NOT, THERE IS TRENDS OVER THE
7 ENTIRE DOSE RANGE. SO IT DOESN'T ORDINARILY HAPPEN THAT
8 ONLY A HIGH DOSE EFFECT IS SEEN. EVEN IF IT WERE, THESE
9 ARE STILL CARCINOGENIC RESPONSES AND RELEVANT TO THE
10 CARCINOGENIC RISK EVALUATION.

11 Q WHAT DOES THE TERM "MOST SENSITIVE TUMOR
12 SITE" MEAN?

13 A USUALLY IT MEANS THE SITE THAT HAS THE
14 LARGEST NUMBER OF ANIMALS WITH THE TUMOR AT THAT SITE.

15 Q DO YOU HAVE AN OPINION AS TO THE USE OF MOST
16 SENSITIVE TUMOR SITES IN HUMAN CANCER RISK ASSESSMENT?

17 A YES.

18 Q WHAT IS YOUR OPINION?

19 A I THINK THAT THEY SHOULD CONTINUE TO BE USED
20 TO MAKE THE EVALUATION OF THE POTENTIAL HUMAN RISK
21 ASSESSMENT.

22 Q TELL THE COURT, IF YOU WOULD, YOUR OVERALL
23 CONCLUSION REGARDING THE UTILITY OF ANIMAL CANCER
24 BIOASSAY DATA IN INFORMING HUMAN CANCER RISK ASSESSMENT?

25 A WELL, OVER DECADES, THESE HAVE BEEN USED TO
26 EVALUATE THE CARCINOGENIC RISK POTENTIAL OF CHEMICALS TO
27 HUMANS. THE -- THIS IS SUPPORTED BY THE KNOWN HUMAN
28 CARCINOGENS THAT ARE ALSO CARCINOGENIC IN ANIMALS. THIS

1 IS SUPPORTED FURTHER BY A THIRD OR MORE OF THE KNOWN
2 HUMAN CARCINOGENS THAT WERE ACTUALLY FIRST DISCOVERED TO
3 BE CARCINOGENIC IN ANIMALS. THE CANCER PROCESS ITSELF
4 BETWEEN THE DIFFERENT MAMMALS IS THE SAME. SO THERE IS
5 NOT A DIFFERENT CANCER THAT DEVELOPS IN ANIMALS THAT
6 WOULD BE DIFFERENT THAN HOW A CANCER DEVELOPS IN HUMANS.

7 SO I THINK THAT THESE THINGS TAKEN TOGETHER,
8 WITH THE BIOLOGICAL PROCESS, INDICATES THAT ANIMAL DATA
9 ARE RELEVANT TO HUMAN RISK ASSESSMENT.

10 Q HAVE YOU FORMED AN OPINION AS TO WHETHER IT
11 IS GENERALLY ACCEPTED IN THE SCIENTIFIC COMMUNITY THAT
12 ANIMAL CANCER BIOASSAY DATA CAN AND SHOULD BE USED IN
13 HUMAN CANCER RISK ASSESSMENT?

14 A YES.

15 Q WHAT IS YOUR OPINION?

16 A I THINK IT IS UNIVERSALLY ACCEPTED THAT THIS
17 IS THE BEST EVALUATION THAT WE HAVE TO PREDICT A
18 POTENTIAL CARCINOGENIC RISK TO HUMANS. IT IS SUPPORTED
19 BY THE HISTORICAL USE OF IT AND THE RELEVANCE AS JUST
20 MENTIONED.

21 Q LET ME ASK YOU, ARE THERE ANY ADVANTAGES OF
22 USING CANCER BIOASSAY DATA FOR HUMAN CANCER RISK
23 ASSESSMENT AS OPPOSED TO HUMAN EPIDEMIOLOGIC DATA?

24 A YES.

25 Q WHAT ARE THEY?

26 A IN PARTICULAR, THE EXPOSURE ASSESSMENT IN
27 ANIMALS IS SIGNIFICANTLY MORE ACCURATE WITH RESPECT TO
28 TUMOR INDUCTION THAN ARE STUDIES IN HUMANS BECAUSE ONE OF

1 THE MOST DIFFICULT THINGS IN HUMANS IS TO GET AN ACCURATE
2 ASSESSMENT OF THE AMOUNT OF EXPOSURE THAT HUMANS HAVE
3 BEEN SUBJECT TO FROM A PARTICULAR CHEMICAL. BECAUSE
4 ANIMALS GET ONE CHEMICAL ONLY, ANIMALS GET A SPECIFIC
5 EXPOSURE LEVEL AT TWO OR THREE DIFFERENT DOSE LEVELS, AND
6 WE KNOW FOR CERTAIN WHAT THEY ARE EXPOSED TO, VERSUS
7 HUMANS, WHO WE LIVE IN A SEA OF DIFFERENT CHEMICALS AND
8 -- SO IT IS -- ANIMALS ARE MUCH MORE SPECIFIC.

9 Q COULD YOU TELL THE COURT HOW THE
10 DIFFERENTIAL EXPOSURE BETWEEN THE EXPOSED ANIMALS AND THE
11 CONTROL ANIMALS COMPARES TO DIFFERENTIAL EXPOSURE IN
12 EXPOSED AND COMPARISON GROUPS IN EPIDEMIOLOGIC STUDIES?

13 MR. SCHURZ: OBJECTION. LACKS FOUNDATION AS TO
14 THIS WITNESS'S KNOWLEDGE OR UNDERSTANDING WITH RESPECT TO
15 EPIDEMIOLOGICAL STUDIES.

16 THE COURT: LAY THE PROPER FOUNDATION.

17 MR. METZGER: SURE.

18 Q DR. HUFF, IN YOUR WORK, HAVE YOU ASSESSED
19 EPIDEMIOLOGIC STUDIES?

20 A YES.

21 Q HAVE YOU PUBLISHED REGARDING EPIDEMIOLOGIC
22 STUDIES?

23 A YES.

24 Q COULD YOU EXPLAIN THE DIFFERENCE, IF ANY,
25 BETWEEN THE EXPOSURE TO ANIMALS AND CONTROLS IN CANCER
26 BIOASSAYS VERSUS EXPOSED POPULATIONS AND COMPARISON
27 POPULATIONS IN EPIDEMIOLOGIC STUDIES?

28 MR. SCHURZ: I WILL RESTATE THE OBJECTION. LACKS

1 FOUNDATION WITH RESPECT TO THIS WITNESS'S UNDERSTANDING
2 OF THE EPIDEMIOLOGICAL STUDIES, DIFFERENTIAL
3 CLASSIFICATIONS, AND THEIR USE.

4 THE COURT: WELL, THE OBJECTION TO THIS QUESTION IS
5 SUSTAINED (SIC), BUT WE WILL TAKE THE QUESTIONS ONE AT A
6 TIME AND WITHOUT PREJUDICE TO ANYONE OBJECTING.

7 GO AHEAD.

8 MR. METZGER: DID YOU SAY "SUSTAINED"?

9 THE COURT: IT IS OVERRULED, BUT WE WILL SEE WHERE
10 YOU GO WITH THIS.

11 Q BY MR. METZGER: YOU CAN ANSWER THE
12 QUESTION, DR. HUFF.

13 A WELL, IN ANIMAL STUDIES, AS MENTIONED, THE
14 EXPOSURE LEVELS THROUGH THE DIFFERENT EXPOSURE GROUPS IS
15 WELL KNOWN AND -- WELL KNOWN AND INDIVIDUAL ANIMALS, WHAT
16 THEY RECEIVE, IS ACCURATELY RECORDED. THE CONTROL GROUPS
17 RECEIVE NO CHEMICAL, BUT THEY RECEIVE EVERYTHING ELSE
18 THAT THE TREATED GROUP RECEIVES.

19 FOR INSTANCE, IF ANIMALS RECEIVE THEIR
20 CHEMICAL IN FOOD, THEN THE FOOD THAT THE CONTROL GROUP
21 HAS IS THE SAME, BUT WITHOUT THE CHEMICAL. SIMILARLY
22 WITH THE OTHER ROUTES OF EXPOSURE.

23 IN HUMANS, EXCEPT FOR SOME -- EXCEPT FOR
24 OCCUPATIONAL STUDIES, IT IS OFTEN DIFFICULT TO FIND A
25 CONTROL GROUP THAT HAS NO EXPOSURE TO THE PARTICULAR
26 AGENT THAT WE ARE LOOKING AT IN EPIDEMIOLOGICAL STUDIES.

27 FOR INSTANCE, DIETARY STUDIES ARE NOTORIOUS
28 FOR HAVING DIFFICULTY IN FINDING TRUE CONTROL GROUPS,

1 ESPECIALLY IN THE ADJACENT POPULATIONS.

2 MR. SCHURZ: YOUR HONOR, WE WILL OBJECT AND MOVE TO
3 STRIKE AS TO LACKS FOUNDATION. THESE ARE ALL NEW
4 OPINIONS. THIS WITNESS HAS NO BASIS TESTIFYING WITH
5 RESPECT TO DIETARY EPIDEMIOLOGY OR ANYTHING RELATING TO
6 EPIDEMIOLOGY. IT IS OUTSIDE OF THE SCOPE OF HIS
7 DESIGNATION. THERE IS NOTHING IN HIS WRITTEN OPINIONS
8 THAT HE HAS BEFORE HIM AND THAT HE HAS PROVIDED AND
9 NOTHING IN THE SCOPE OF HIS DEPOSITIONS THAT INDICATE ANY
10 OPINIONS THAT HE HAS WITH RESPECT TO THE USE OF
11 EPIDEMIOLOGIC LITERATURE.

12 THE COURT: THE OBJECTION OVERRULED. NEXT
13 QUESTION.

14 Q BY MR. METZGER: DR. HUFF, WHY NOT DO A
15 HUMAN STUDY WHERE YOU GIVE HUMANS -- ONE GROUP OF HUMANS
16 CERTAIN DOSES OF ACRYLAMIDE AND THE OTHER GROUP YOU DON'T
17 GIVE THEM ACRYLAMIDE?

18 A WHY NOT?

19 Q WELL, LIKE YOU DO IN ANIMALS, HAS THAT BEEN
20 DONE?

21 A LONG-TERM STUDIES SUCH AS THAT, NO. BUT WAY
22 BACK IN THE DAY, TOXICOLOGY STUDIES WERE DONE ON HUMAN
23 VOLUNTEERS FOR SHORT-TERM EXPOSURES TO POTENTIALLY
24 HAZARDOUS AGENTS, BUT IT IS, IN MY OPINION, UNETHICAL TO
25 EXPOSE HUMAN BEINGS TO KNOWN CARCINOGENS, OR AT LEAST
26 KNOWN RODENT CARCINOGENS.

27 Q CHANGING TOPICS. DID I ALSO ASK YOU, AS
28 PART OF THIS CASE, TO ASSESS THE -- OR TO IDENTIFY

1 CARCINOGENS THAT ARE PRESENT IN COFFEE?

2 A YES.

3 Q DID YOU DO THAT?

4 A YES.

5 Q ALL RIGHT. CAN YOU GIVE US AN OVERVIEW --
6 FIRST OF ALL, APPROXIMATELY HOW MANY CHEMICALS ARE THERE
7 IN COFFEE, IN BREWED COFFEE?

8 A ANYWHERE FROM 500 TO 1,000 TO 1,500
9 CHEMICALS. IT HASN'T BEEN TOTALLY DETERMINED.

10 Q DO YOU HAVE AN ESTIMATE OF HOW MANY OF THOSE
11 CHEMICALS HAVE ACTUALLY BEEN TESTED FOR CARCINOGENICITY?

12 A YES.

13 Q WHAT IS IT?

14 A WELL, A REASONABLE ESTIMATE IS ROUGHLY 50 OF
15 THOSE CHEMICALS HAVE BEEN EVALUATED IN LONG-TERM
16 BIOASSAYS.

17 Q AND COULD YOU INFORM THE COURT OF --
18 GENERALLY OF THOSE 50 OUT OF SAY 1,000 THAT ARE IN
19 COFFEE, WHAT PERCENTAGE OF THOSE CHEMICALS THAT HAVE BEEN
20 TESTED THAT ARE PRESENT IN COFFEE ARE FOUND TO BE
21 CARCINOGENIC?

22 A APPROXIMATELY TWO-THIRDS TO THREE-QUARTERS
23 HAVE SHOWN CARCINOGENIC ACTIVITY IN ANIMAL STUDIES.

24 Q CAN YOU TELL US WHAT SOME OF THOSE CHEMICALS
25 ARE?

26 A WELL, WE KNOW THAT HUMAN -- SOME HUMAN
27 CARCINOGENS ARE IN COFFEE LIKE AFLATOXIN. IT ESCAPES ME
28 RIGHT NOW WHAT SOME OF THE OTHERS ARE.

1 BENZENE IS IN COFFEE. ANOTHER HUMAN
2 CARCINOGEN. FORMALDEHYDE, IS IN COFFEE. ANOTHER HUMAN
3 CARCINOGEN.

4 Q OKAY. LET ME ASK YOU ABOUT CERTAIN
5 CHEMICALS AND I WILL GO ONE BY ONE. ARE YOU FAMILIAR
6 WITH A CHEMICAL KNOWN AS BENZOPYRENE?

7 A YES.

8 MR. SCHURZ: OBJECT AS LEADING. THE WITNESS HAS
9 GIVEN AN ANSWER WHICH HE BELIEVES THE CARCINOGEN IS IN
10 COFFEE. NOW COUNSEL WANTS TO GO THROUGH A LIST AND FEED
11 HIM THE ANSWERS.

12 THE COURT: WELL, I WILL HOLD OFF WITH A DECISION
13 TO SEE WHERE THIS GOES. WE ARE NOT GOING TO GO THROUGH
14 100 CHEMICALS. THE WITNESS SAYS HE IS FAMILIAR WITH
15 BENZENE.

16 NEXT QUESTION?

17 Q BY MR. METZGER: WHAT IS THE CLASSIFICATION
18 OF BENZOPYRENE?

19 A IT IS A KNOWN HUMAN CARCINOGEN.

20 Q IS IT A CHEMICAL THAT IS PRESENT IN COFFEE?

21 A YES.

22 Q YOU MENTIONED FORMALDEHYDE AND YOU MENTIONED
23 BENZENE. ARE THERE CERTAIN METABOLITES OF BENZENE THAT
24 ARE ALSO CARCINOGENS THAT ARE PRESENT IN COFFEE?

25 A YES.

26 THE COURT: THE OBJECTION IS SUSTAINED. THE ANSWER
27 IS STRICKEN. THIS HAS NOTHING TO DO WITH THIS CASE.
28 LET'S MOVE ON.

1 MR. METZGER: I THINK IT HAS MUCH TO DO WITH THE
2 CASE.

3 THE COURT: WHAT DOES IT HAVE TO DO WITH THIS CASE?

4 MR. METZGER: WELL, THE CONCEPT IS THAT SINCE THERE
5 ARE MULTIPLE CARCINOGENS IN COFFEE, THAT LENDS SUPPORT TO
6 THE CONCEPT THAT COFFEE HAS CARCINOGENIC POTENTIAL TO
7 HUMANS.

8 THE COURT: WHAT DOES THAT HAVE TO DO WITH THE
9 CLAIM OF ACRYLAMIDE?

10 WE ARE TALKING ABOUT -- WE ARE GOING TO TRY
11 A CASE ABOUT BENZENE AND FORMALDEHYDE?

12 MR. METZGER: NO. NO. HERE IS WHAT IT HAS TO DO:
13 I PERSONALLY DON'T THINK THAT THIS CASE IS ABOUT COFFEE
14 AT ALL. IN MY VIEW, AND IN THE PLAINTIFF'S VIEW, COFFEE
15 IS MERELY THE VEHICLE THAT IS DELIVERING ACRYLAMIDE TO
16 CALIFORNIANS WITHOUT A WARNING. BUT THE DEFENDANT'S VIEW
17 OF THE WORLD IS THAT THE CASE IS ABOUT COFFEE AND THAT
18 COFFEE IS A PANACEA TO ALL HUMAN ILLS.

19 SO I THINK I AM TOTALLY ENTITLED TO REBUT
20 THEIR POSITION AND THEIR EVIDENCE ABOUT THE BENEFICIAL
21 EFFECTS OF COFFEE BY SHOWING THAT COFFEE IS REplete WITH
22 KNOWN HUMAN CARCINOGENS.

23 THE COURT: WELL, THE DEFENDANT CAN CORRECT ME IF I
24 AM WRONG, BUT I UNDERSTOOD THE DEFENDANT'S POSITION TO BE
25 THAT THE CASE IS NOT ABOUT COFFEE PER SE, BUT ABOUT
26 ACRYLAMIDE IN COFFEE AND THAT THERE ARE OTHER CHEMICALS
27 IN COFFEE THAT COUNTERACT ANY NEGATIVE EFFECT OF
28 ACRYLAMIDE.

1 IS THAT DEFENDANT'S POSITION?

2 MR. SCHURZ: YES, YOUR HONOR.

3 THE COURT: OKAY.

4 MR. METZGER: WELL, IF THAT IS THEIR POSITION,
5 THEIR EXPERT DID NOT DO A QUALITATIVE RISK ASSESSMENT OF
6 ACRYLAMIDE IN COFFEE.

7 THE COURT: AND YOU CAN MAKE AN ARGUMENT AT THE END
8 OF THE CASE. WE ARE NOT GOING TO GO THROUGH EVERY OTHER
9 CHEMICAL. IF YOU WANT TO FOCUS ON ANY EVIDENCE WITH
10 REGARD TO COUNTERVAILING FACTORS IN OTHER CHEMICALS, YOU
11 CAN DO SO, BUT WE ARE NOT GOING TO GO THROUGH A LIST OF
12 100 CHEMICALS AND FIND OUT WHAT EFFECT, IF ANY, THEY HAVE
13 ON THE INGESTION OF COFFEE.

14 MR. METZGER: ALL RIGHT. THEN I WILL LEAVE IT WITH
15 YOUR ESTIMATE THAT YOU GAVE OF THE NUMBER OF CARCINOGENS
16 IN COFFEE AND I WILL MOVE ON.

17 THE COURT: ALL RIGHT.

18 Q BY MR. METZGER: DR. HUFF, ARE YOU FAMILIAR
19 WITH DR. BRUCE AMES?

20 A YES, I AM.

21 Q WHO IS BRUCE AMES?

22 A BRUCE AMES IS A MICROBIOLOGIST WHO HAS
23 DEVELOPED THE SHORT-TERM TEST USING SALMONELLA
24 TYPHIMURIUM TO IDENTIFY CHEMICALS THAT ARE MUTAGENIC IN
25 ITS SYSTEM. HIS HYPOTHESIS WAS THAT THIS WOULD BE A
26 PREDICTOR OF CANCER IN ANIMALS. THAT IS WHAT BRUCE AMES
27 DEVELOPED.

28 Q DID BRUCE AMES ALSO DEVELOP SOMETHING THAT

1 IS CALLED THE HUMAN EXPOSURE RODENT POTENCY OR HERP
2 INDEX?

3 A YES, HE DID.

4 Q ARE YOU FAMILIAR WITH THAT?

5 A YES.

6 Q BEFORE WE GET INTO THAT, DID BRUCE AMES
7 EXPRESS HIS OWN VIEWS OR CONCLUSIONS IN A NUMBER OF
8 ARTICLES REGARDING HIS VIEWS ON HUMAN CANCER RISK
9 ASSESSMENT?

10 A YES.

11 Q AND IF MR. SCHURZ READS CONCLUSIONS FROM
12 THOSE ARTICLES TO YOU, CAN YOU TELL US PROSPECTIVELY
13 WHETHER YOU AGREE OR DISAGREE WITH THEM?

14 MR. SCHURZ: WELL, I AM GOING TO OBJECT. THIS
15 CALLS FOR SPECULATION. LACKS FOUNDATION. DR. HUFF
16 DOESN'T KNOW WHAT WE ARE GOING TO TALK ABOUT.

17 CALLS FOR SPECULATION.

18 THE COURT: YES, I DON'T THINK HE CAN TO TESTIFY
19 WHAT MR. SCHURZ IS GOING TO DO IN CROSS-EXAMINATION.

20 Q BY MR. METZGER: LET ME ASK IT THIS WAY: DO
21 YOU AGREE WITH BRUCE AMES'S VIEWS OF HUMAN CANCER RISK
22 ASSESSMENT?

23 A NO.

24 Q OKAY. NOW, REGARDING THE HERP INDEX THAT HE
25 DEVELOPED, HAVE YOU EVALUATED THAT IN ANY WAY FOR THIS
26 CASE?

27 A YES.

28 Q WOULD YOU TELL US WHAT YOU USED THE HERP

1 INDEX FOR IN YOUR WORK IN THIS CASE?

2 A WELL, I THOUGHT IT WAS INTERESTING AND
3 PREPARED SOME TYPE OF CHART TO INDICATE THAT OF THE
4 AGENTS THAT HE HAS CALCULATED A HERP INDEX FOR, AND THAT
5 IS HUMAN EXPOSURE RODENT POTENCY, BASED ON BIOASSAY
6 RESULTS AND HIS VIEW, HIS ANALYSIS OF THE HUMAN EXPOSURE
7 PIECE OF THAT, AND COMPARED VARIOUS AGENTS, VARIOUS FOOD
8 CONSTITUENTS USING HIS MATHEMATICAL MODEL, THE HERP
9 INDEX.

10 Q LET ME ASK YOU FIRST: WHAT IS THE HERP
11 INDEX?

12 COULD YOU EXPLAIN WHAT THAT HUMAN EXPOSURE
13 RODENT POTENCY INDEX IS TO THE COURT GENERALLY.

14 A WELL, IT IS HIS COMBINATION OF USING WHAT IS
15 KNOWN ABOUT THE HUMAN EXPOSURE TO A PARTICULAR CHEMICAL
16 AND HIS AWARENESS OF THE CANCER -- AVAILABLE CANCER DATA
17 FROM RODENTS. HE THEN USES THIS IN HIS MODEL TO COME OUT
18 WITH A NUMBER THAT IS AN INDICATION OF THE POTENTIAL, IN
19 HIS VIEW, HAZARD OF THAT AGENT. AND HE DOES THIS FOR
20 MANY, MANY CHEMICALS. HE USES IT TO COMPARE ONE TO THE
21 OTHER BY THE SIZE OF THE HERP INDEX.

22 Q ALL RIGHT. HAVE YOU LOOKED AT THE HERP
23 INDEX VALUES THAT DR. AMES AND HIS COLLEAGUES DETERMINED
24 FOR DIFFERENT FRUITS AND VEGETABLES, INCLUDING COFFEE?

25 A YES, I DID.

26 Q DID YOU COMPILE THOSE INTO A CHART OR A
27 GRAPH TO VISUALLY DEPICT THE DIFFERENTIAL HERP INDEX
28 VALUES FOR THE FRUITS AND VEGETABLES IN COFFEE?

1 A YES.

2 Q COULD WE DISPLAY, PLEASE, EXHIBIT -- I'M
3 SORRY, SLIDE 86.

4 IS THIS THE GRAPH THAT YOU PREPARED?

5 A YES, IT IS.

6 Q WOULD YOU EXPLAIN IT TO THE COURT, PLEASE.

7 A WELL, THIS WAS ACTUALLY TAKEN FROM
8 DR. AMES'S STUDIES WHERE HE RANKED IN HIS MODEL THE
9 POSSIBLE CARCINOGENIC HAZARDS OF ANY NUMBER OF AGENTS. I
10 THOUGHT IT WAS PARTICULARLY INTERESTING AND STRIKING TO
11 ME THAT OF THE FOODS THAT I HAVE LISTED HERE, THAT COFFEE
12 IS FAR AND AWAY MORE THAN DOUBLE THE NEXT HIGHEST LEVEL
13 ACCORDING TO THE HERP INDEX.

14 Q SO YOU COMPARED COFFEE TO LETTUCE, TOMATO,
15 APPLE, CARROT, CELERY, PLUM AND PARSNIP, AND THAT IS WHAT
16 THAT GRAPH DEPICTS?

17 A YES, SIR.

18 Q AND COFFEE HAS A HERP INDEX OF 0.10 AND THE
19 NEXT HIGHEST FRUIT OR VEGETABLE IS LETTUCE AT 0.04?

20 A YES.

21 Q GOING DOWN TO PARSNIPS AT 0.0007?

22 A YES.

23 THE COURT: LET ME ASK YOU, THESE OTHER PRODUCTS --
24 FIRST OF ALL, WHEN IT SAYS "COFFEE," IS THAT JUST COFFEE,
25 COFFEE BEANS, OR IS IT SOME PROCESS LIKE BREWING COFFEE
26 AND BOILING COFFEE?

27 THE WITNESS: YOUR HONOR, MAINLY IT IS DR. AMES
28 LOOKED AT THE CHEMICAL COMPOSITION OF ALL OF THESE FRUITS

1 AND VEGETABLES AND COFFEE, AND THEN DID A COMBINED HERP
2 INDEX ON THE COLLECTION OF THOSE CHEMICALS THAT HAVE BEEN
3 STUDIED FOR CANCER AND --

4 THE COURT: MY QUESTION WAS: WHEN YOU SAY
5 "COFFEE," IS IT RAW COFFEE BEANS OR IS IT COFFEE THAT HAS
6 BEEN PROCESSED IN SOME WAY?

7 THE WITNESS: I MUST SAY I DON'T RECALL THAT.

8 THE COURT: BECAUSE MY NEXT QUESTION WAS GOING TO
9 BE: CAN YOU -- THESE OTHER FRUITS AND VEGETABLES, ARE
10 THEY RAW FRUITS AND VEGETABLES OFF THE VINE OR HAVE THEY
11 BEEN PROCESSED, LIKE BOILED OR SOME OTHER COOKING
12 PROCESS?

13 BECAUSE IF NOT, THEN ARE WE COMPARING APPLES
14 TO ORANGES?

15 ARE WE TALKING ABOUT SOMETHING IN WHICH
16 THERE IS A PROCESS, A HUMAN OR INDUSTRIAL PROCESS,
17 COMPARED TO SOMETHING WHICH IS JUST RAW, NOT PROCESSED AT
18 ALL?

19 THE WITNESS: YOUR HONOR, IT IS MY RECALL THAT WHAT
20 DR. AMES DID WAS HE DID NOT TEST THESE FRUITS AND
21 VEGETABLES AS THEY ARE IN A BIOLOGICAL SYSTEM. WHAT HE
22 DID WAS, THROUGH THE LITERATURE, FIND OUT WHAT CHEMICALS
23 ARE IN THESE FRUITS AND VEGETABLES, AND THEN PUT THOSE
24 VALUES, THOSE CHEMICALS INTO HIS MODEL OF THE HERP INDEX,
25 AND CAME OUT WITH THIS COMPOSITE RESULT.

26 THE COURT: I WANT TO MAKE SURE WE ARE COMPARING
27 THE SAME THING. SO IS THAT ALSO TRUE OF COFFEE, THAT THE
28 BEANS WERE TAKEN AS THEY WERE, WITHOUT PROCESSING?

1 THE WITNESS: I BELIEVE SO.

2 THE COURT: ALL RIGHT. COUNSEL.

3 Q BY MR. METZGER: ALL RIGHT, DR. HUFF. NEW
4 TOPIC.

5 CAN YOU TELL US WHAT THE ARYL HYDROCARBON
6 RECEPTOR IS?

7 MR. SCHURZ: YOUR HONOR, WE WOULD OBJECT AS THE
8 OPINIONS THAT ARE NOW BEING SOUGHT TO BE ELICITED ARE
9 ENTIRELY NEW, UNDISCLOSED, AND WERE NOT PART OF
10 DR. HUFF'S EARLIER TESTIMONY. THIS IS THE FIRST WE HAVE
11 HEARD OF IT. WE SAW IT IN THE DEMONSTRATIVE THAT WAS
12 PROVIDED TO US YESTERDAY. THIS IS THE FIRST TIME THAT WE
13 HAVE SEEN OR HEARD ABOUT THIS PARTICULAR MEDIATED GENE.
14 YOU WILL SEE IT AS ONE OF THE ELEMENTS THAT WAS SUBMITTED
15 WITH OUR WRITTEN OBJECTION.

16 THE COURT: MR. METZGER, WHAT ABOUT THAT?

17 MR. METZGER: WELL, DR. HUFF DID TESTIFY AT HIS
18 DEPOSITION REGARDING THE GENOTOXICITY, MUTAGENICITY,
19 CLASTOGENICITY, CHROMOSOMAL ABERRATIONS, AND ALSO -- JUST
20 A MOMENT -- THE -- THE CELL TRANSFORMATION, ALL THESE
21 GENETIC EFFECTS OF COFFEE, AND THE ARYL HYDROCARBON
22 RECEPTOR, AS MR. SCHURZ INDICATES, IS A REPORTER GENE.

23 IN PARTICULAR, WHAT I AM -- DR. HUFF HAS AN
24 ARTICLE THAT WAS JUST RECENTLY PUBLISHED REGARDING THE
25 ARYL HYDROCARBON RECEPTOR GENE AND COFFEE. THAT IS WHAT
26 I WOULD LIKE HIM TO TESTIFY ABOUT.

27 THE COURT: WAS THAT DISCUSSED IN HIS DEPOSITION?

28 MR. METZGER: THE ARTICLE WAS JUST PUBLISHED. SO

1 NO, IT WAS NOT.

2 THE COURT: THEREFORE, WHAT?

3 MR. METZGER: WELL, IT COULD NOT HAVE BEEN
4 DISCUSSED AT HIS DEPOSITION BECAUSE IT DID NOT EXIST AT
5 THAT TIME.

6 THE COURT: ALL RIGHT. SO IS THAT A REASON TO
7 ALLOW IT NOW?

8 SHOULD WE RECESS FOR A NEW DEPOSITION?

9 MR. METZGER: I THINK SCIENCE IS ALWAYS DEVELOPING
10 AND THAT DR. -- THE DEFENSE EXPERTS TESTIFIED ABOUT
11 RECENT ARTICLES THAT THEY DID NOT TESTIFY ABOUT AT THEIR
12 DEPOSITION AND I DID NOT OBJECT BECAUSE SCIENCE IS
13 DEVELOPING. IF YOU DON'T FEEL IT IS APPROPRIATE FOR
14 DR. HUFF TO TESTIFY ABOUT THIS NEW DEVELOPMENT IN
15 SCIENCE, THEN HE WON'T BE ABLE DO THAT.

16 THE COURT: ALL RIGHT. THE OBJECTION IS SUSTAINED.

17 MR. METZGER: OKAY.

18 THE COURT: INCIDENTALY, I THINK WHAT WE OUGHT TO
19 DO IN CONNECTION -- THERE HAVE BEEN A NUMBER OF
20 OBJECTIONS DURING THE COURSE OF THE TRIAL ON BOTH SIDES
21 WITH REGARD TO BEYOND THE SCOPE OF THE EXPERT'S PREVIOUS
22 TESTIMONY. I THINK WHAT WE OUGHT TO DO AT THE CONCLUSION
23 IS TO HAVE, ALONG WITH THIS MOTION, INSTEAD OF DOING THIS
24 PIECEMEAL, THE REQUEST OF THE PARTIES TO EXCLUDE
25 EVIDENCE, IN CONNECTION WITH THAT MOTION, THERE SHOULD BE
26 A CHART OF WHAT THE PARTY CONTENDS IS THE NEW OPINIONS
27 THAT WAS NOT PREVIOUSLY DISCLOSED IN ONE COLUMN, AND THE
28 OTHER COLUMN, THE NOTATION OF WHAT WAS DISCUSSED IN THE

1 DEPOSITION.

2 FOR EXAMPLE, THE WITNESS HAS BEEN TESTIFYING
3 AS TO CANCER ABC, AND THEN AT HIS DEPOSITION, HE
4 TESTIFIED ABC WAS SOMETHING ELSE. THEN YOU COULD ALWAYS
5 REPRESENT THAT IN CONNECTION WITH THE MOTION.

6 WE WILL DISCUSS THAT AT THE END. I WANT TO
7 TRY TO FINISH THE TESTIMONY.

8 GO AHEAD, MR. METZGER.

9 MR. METZGER: OKAY. NO FURTHER QUESTIONS.

10 THE COURT: THANK YOU.

11 MR. SCHURZ.

12 MR. METZGER: THANK YOU, DR. HUFF.

13 THE WITNESS: THANK YOU.

14

15 CROSS-EXAMINATION

16

17 BY MR. SCHURZ:

18 Q GOOD AFTERNOON, DR. HUFF.

19 A GOOD AFTERNOON.

20 Q LET'S START, IF WE MIGHT, ABOUT SOME OF THE
21 TESTIMONY THAT YOU OFFERED, DR. HUFF, REGARDING YOUR
22 OPINIONS THAT ANIMAL BIOASSAYS ARE PREDICTIVE OF HUMAN
23 CANCER RISK.

24 WOULD YOU AGREE, DR. HUFF, THAT HUMAN DATA
25 PROVIDE THE MOST DIRECT EVIDENCE OF ADVERSE HEALTH
26 EFFECTS IN HUMANS?

27 A YES.

28 Q AND YOU EARLIER TESTIFIED ABOUT YOUR --

1 DR. TOMATIS AT IARC; IS THAT CORRECT?

2 A YES.

3 Q AND YOU PUBLISHED WITH HIM BEFORE, I TAKE
4 IT?

5 A YES.

6 Q AND IF I COULD SHOW YOU NOW EXHIBIT 192.

7 DR. HUFF, WE DISCUSSED THIS DOCUMENT AT YOUR
8 DEPOSITION; DID WE NOT?

9 MR. METZGER: OBJECTION, YOUR HONOR, AS TO WHETHER
10 DR. HUFF READ, REVIEWED AND CONSIDERED THIS FOR THIS CASE
11 OR WHETHER MR. SCHURZ JUST SHOWED IT TO HIM AT THE
12 DEPOSITION.

13 THE COURT: ALL RIGHT. NOW, THIS IS A PREFATORY
14 QUESTION. IT IS MEANINGLESS WHETHER THEY DISCUSSED IT OR
15 NOT. LET'S MOVE ON TO SOMETHING SUBSTANTIVE.

16 LET'S GO TO THE NEXT QUESTION.

17 Q BY MR. SCHURZ: TURNING YOUR ATTENTION TO
18 PAGE 6 OF EXHIBIT 192.

19 MR. METZGER: OBJECTION, YOUR HONOR. 721(B).
20 THERE IS NO FOUNDATION.

21 THE COURT: WE HAVE NOT HEARD THE QUESTION YET.

22 MR. METZGER: THE QUESTION I WOULD LIKE TO HEAR IS
23 HAS HE READ, REVIEWED OR CONSIDERED THIS.

24 THE COURT: I ASSUME THERE WILL BE A QUESTION.

25 MR. METZGER: NOT THAT ONE.

26 THE COURT: WELL, LET'S SEE.

27 Q BY MR. SCHURZ: DID WE REVIEW THIS DOCUMENT
28 AT YOUR DEPOSITION, DR. HUFF?

1 MR. METZGER: OBJECTION; RELEVANCE.

2 THE COURT: OVERRULED.

3 MR. METZGER: IT IS A "YES" OR "NO" QUESTION.

4 MR. SCHURZ: I AM GOING TO OBJECT TO MR. METZGER
5 COACHING WITNESSES.

6 MR. METZGER: EXCUSE ME, YOUR HONOR. I APOLOGIZE.

7 THE COURT: MR. METZGER, IF YOU HAVE AN OBJECTION,
8 JUST STATE AN OBJECTION. DON'T GIVE ADVICE TO THE
9 WITNESS FROM THE COUNSEL TABLE.

10 MR. METZGER: YES, I DO APOLOGIZE. IT WAS TOTALLY
11 IMPROPER.

12 THE WITNESS: YES, I RECALL READING ONE SENTENCE IN
13 THE DOCUMENT.

14 Q BY MR. SCHURZ: DIRECTING YOUR ATTENTION TO
15 PAGE 6 OF EXHIBIT 192 WHERE DR. TOMATIS STATES --

16 MR. METZGER: OBJECTION; 721(B).

17 THE COURT: WE WILL FIND OUT IF HE READ IT OR
18 STUDIED IT. LET'S HEAR THE QUESTION.

19 MR. METZGER: HE IS NOT ASKING IT.

20 THE COURT: MR. SCHURZ, GO AHEAD.

21 MR. SCHURZ: THANK YOU, YOUR HONOR.

22 Q WHERE DR. TOMATIS STATES, QUOTE:
23 "EPIDEMIOLOGICAL STUDIES PROVIDES THE ONLY" --

24 THE COURT: MR. METZGER.

25 MR. METZGER: OBJECTION.

26 THE COURT: MR. METZGER, PLEASE WAIT UNTIL THE END
27 OF THE QUESTION.

28 MR. METZGER: BUT HE IS READING FROM A DOCUMENT

1 THAT IS INADMISSIBLE HEARSAY. THAT IS NOT THE WAY IT IS
2 SUPPOSED TO BE.

3 THE COURT: IT IS A QUESTION. NOTHING IN A
4 QUESTION IS ADMISSIBLE EVIDENCE. THERE IS A QUESTION AND
5 IT SEEMS LIKE A PREFACE TO A QUESTION.

6 MR. METZGER: NO, IT IS NOT.

7 THE COURT: MR. SCHURZ, GO AHEAD.

8 MR. METZGER: HE WANTS TO READ HEARSAY INTO THE
9 RECORD. THAT IS ALL THAT IS GOING ON HERE, JUDGE.

10 THE COURT: ALL RIGHT. THANK YOU, MR. METZGER, FOR
11 YOUR OBSERVATION.

12 COUNSEL.

13 MR. SCHURZ: THANK YOU, YOUR HONOR.

14 Q DR. HUFF, DIRECTING YOUR ATTENTION TO THE
15 FIRST SENTENCE OF EXHIBIT 192 WHERE DR. TOMATIS STATES:

16 "EPIDEMIOLOGICAL STUDIES
17 PROVIDE THE ONLY DEFINITIVE
18 INFORMATION ON THE DEGREE OF CANCER
19 RISK TO MAN."

20 MR. METZGER: OBJECTION; 721(B).

21 THE COURT: OVERRULED.

22 Q BY MR. SCHURZ: IS THIS A SENTENCE WE
23 DISCUSSED AT YOUR DEPOSITION?

24 MR. METZGER: SAME OBJECTION.

25 THE COURT: OVERRULED.

26 THE WITNESS: I RECALL WE DID, YES.

27 Q BY MR. SCHURZ: I TAKE IT YOU DISAGREE WITH
28 DR. TOMATIS'S STATEMENT HERE AS EXPRESSED IN EXHIBIT 192?

1 A NO, I DON'T.

2 Q THANK YOU. MOVING ON.

3 MR. METZGER: YOUR HONOR, I WOULD MOVE TO STRIKE
4 THAT ENTIRE LINE OF QUESTIONING AND ANSWERS PURSUANT TO
5 EVIDENCE CODE SECTION 721(B) BECAUSE NO PROPER FOUNDATION
6 WAS LAID FOR THAT TYPE OF EXAMINATION.

7 THE COURT: OBJECTION OVERRULED.

8 Q BY MR. SCHURZ: DR. HUFF, YOU HAVE TESTIFIED
9 WITH RESPECT TO THE USE OF ANIMAL BIOASSAYS OR TWO-YEAR
10 ANIMAL STUDIES FOR THEIR USE IN QUANTITATIVE RISK
11 ASSESSMENT.

12 DO YOU RECALL THAT TESTIMONY?

13 A YES.

14 Q YOU HAVE OFFERED A NUMBER OF OPINIONS ABOUT
15 HOW DATA SHOULD BE USED IN THOSE QUANTITATIVE RISK
16 ASSESSMENTS; CORRECT?

17 A YES.

18 Q NOW, IN TERMS OF YOUR OWN EXPERIENCE,
19 DR. HUFF, YOU HAVE NEVER ACTUALLY PERFORMED A
20 QUANTITATIVE RISK ASSESSMENT; HAVE YOU?

21 A NO, I HAVE NOT.

22 Q YOU ARE NOT AN EXPERT IN SELECTING TUMORS
23 FOR PURPOSES OF PERFORMING A QUANTITATIVE RISK
24 ASSESSMENT; ARE YOU?

25 A YES, I AM.

26 Q YOU ARE?

27 A YES.

28 Q AND YOUR WORK AT N.T.P. DID NOT INVOLVE

1 QUANTITATIVE RISK ASSESSMENTS; DID IT?

2 A NO.

3 Q IN FACT, N.T.P. DOESN'T DO QUANTITATIVE RISK
4 ASSESSMENTS; DOES IT?

5 A THEY HAVE DONE SOME, YES.

6 Q IN YOUR WORK AT N.T.P., WERE YOU INVOLVED IN
7 THE PERFORMING OF ANY QUANTITATIVE RISK ASSESSMENTS?

8 A NO.

9 Q IN FACT, YOU DO NOT CONSIDER YOURSELF AN
10 EXPERT IN HUMAN CANCER RISK ASSESSMENT; DO YOU?

11 A YES, I DO.

12 Q ALL RIGHT. COULD WE HAVE DR. HUFF'S
13 TRANSCRIPT, PLEASE.

14 I WOULD READ FROM PAGE -- THE DEPOSITION
15 TRANSCRIPT, PAGE 172, LINES 18 THROUGH 25.

16 MR. METZGER: JUST A MOMENT, PLEASE.

17 MR. SCHURZ: 18 THROUGH 20.

18 MR. METZGER: NO OBJECTION.

19 THE COURT: ALL RIGHT. YOU MAY READ IT.

20 Q BY MR. SCHURZ:

21 "QUESTION: NOW, DO YOU
22 CONSIDER YOURSELF AN EXPERT IN HUMAN
23 CANCER RISK ASSESSMENT?

24 "ANSWER. NO."

25 LET ME TURN TO A NEW TOPIC.

26 DR. HUFF, YOU TESTIFIED THAT ALL HUMAN
27 CARCINOGENS THAT HAVE BEEN TESTED ON ANIMALS CAUSE CANCER
28 IN ANIMALS; IS THAT CORRECT?

1 A THAT'S CORRECT.

2 Q SO ALL KNOWN HUMAN CARCINOGENS HAVE ALSO
3 BEEN FOUND TO BE CARCINOGENIC IN ANIMALS; CORRECT?

4 A ALL CHEMICALS -- ALL HUMAN CARCINOGENS THAT
5 COULD BE TESTED ADEQUATELY ON ANIMALS HAVE BEEN SHOWN TO
6 BE CARCINOGENIC IN ANIMALS, YES.

7 Q BUT THE CONVERSE IS NOT TRUE; CORRECT?
8 NAMELY, THERE ARE ANIMAL CARCINOGENS THAT DO
9 NOT CAUSE CANCER IN PEOPLE; CORRECT?

10 A AS FAR AS WE KNOW. IF AN EPIDEMIOLOGICAL
11 STUDY HASN'T BEEN DONE, THEN HOW CAN WE MAKE THAT
12 CONCLUSION?

13 Q I THINK MY QUESTION IS A LITTLE SIMPLER.

14 A OKAY.

15 Q SO LET ME TRY IT AGAIN.

16 A ALL RIGHT.

17 Q THERE ARE ANIMAL CARCINOGENS THAT DO NOT
18 CAUSE CANCER IN PEOPLE; CORRECT?

19 A THERE ARE -- FOR SOME ANIMAL CARCINOGENS
20 THAT HAVE NOT HAD EPIDEMIOLOGICAL STUDIES ON THEM, THEN
21 WE HAVE TO ASSUME THAT THEY HAVE NOT BEEN SHOWN TO BE
22 CARCINOGENIC IN HUMANS, YES.

23 Q IN OTHER INSTANCES, THERE ARE KNOWN ANIMAL
24 CARCINOGENS THAT HAVE BEEN FOUND NOT TO BE CARCINOGENIC
25 IN HUMANS; CORRECT?

26 A YES.

27 Q NOW, PHYSIOLOGICALLY, RATS --

28 A I'M SORRY, MAY I -- IT DEPENDS ON WHAT YOU

1 MEAN BY "CARCINOGENIC IN ANIMALS." IF YOU ARE USING THE
2 CRITERIA OF N.T.P. AND IARC, THAT IS ONE THING. IF ARE
3 YOU USING THE CRITERIA THAT IT HAS CAUSED CANCER IN A
4 SINGLE ORGAN IN A SINGLE SEX OF A SINGLE SPECIES, THEN I
5 DON'T HAVE A DIFFERENCE OF OPINION WITH YOU.

6 Q THANK YOU.

7 NOW, SACCHARINE WOULD BE SUCH AN EXAMPLE OF
8 A SUBSTANCE THAT HAS BEEN FOUND TO BE CARCINOGENIC IN
9 ANIMALS, BUT NOT FOUND TO BE CARCINOGENIC IN HUMANS;
10 CORRECT?

11 A NO.

12 Q IS SACCHARINE BELIEVED TO BE CARCINOGENIC IN
13 HUMANS?

14 A SACCHARINE HAS BEEN SHOWN TO BE CARCINOGENIC
15 IN A COHORT OF WOMEN WHO -- FOR URINARY BLADDER CANCER
16 SOME MANY YEARS AGO BY -- I CAN'T COME UP WITH THE NAME
17 AT N.C.I., HOOVER PERHAPS. SACCHARINE WAS PLACED IN THE
18 REPORT ON CARCINOGENS, AND THEN IT WAS SUBSEQUENTLY
19 REMOVED.

20 Q YES. THAT IS MY POINT, SACCHARINE WAS
21 DELISTED AS A HUMAN CARCINOGEN; CORRECT?

22 A YES.

23 Q ALL RIGHT.

24 A NO, IT WAS NEVER LISTED AS A HUMAN
25 CARCINOGEN.

26 Q SACCHARINE DOES NOT APPEAR ON THE LIST OF
27 HUMAN CARCINOGENS; CORRECT?

28 MR. METZGER: OBJECTION; 352, YOUR HONOR.

1 THE COURT: OVERRULED.

2 THE WITNESS: IT IS CORRECT BECAUSE IT HAS NEVER
3 BEEN LISTED AS A HUMAN CARCINOGEN IN THE REPORT ON
4 CARCINOGENS OR ANYWHERE ELSE.

5 Q BY MR. SCHURZ: NOW, RETURNING TO YOUR
6 TESTIMONY RELATING TO RATS AND THEIR SIMILARITY TO
7 HUMANS. IT IS THE CASE THAT PHYSIOLOGICALLY RATS AND
8 MICE ARE MORE SIMILAR TO EACH OTHER THAN EITHER IS TO
9 HUMANS; CORRECT?

10 A I DON'T AGREE WITH THAT.

11 Q AND YOU WOULD EXPECT TO SEE 80 TO 85 PERCENT
12 CONCORDANCE BETWEEN THE TWO RODENT SPECIES; CORRECT?

13 A YES.

14 Q NOW, EARLIER TODAY, WE WALKED THROUGH A
15 CHART IN WHICH YOU DISCUSSED SOME OF THE TUMOR SITE
16 CONCORDANCE AS BETWEEN ACRYLAMIDE AND GLYCIDAMIDE AND
17 METHYLACRYLAMIDE.

18 DO YOU RECALL THAT DISCUSSION?

19 A YES.

20 Q AND I WOULD LIKE TO ADDRESS A SLIGHTLY
21 DIFFERENT TOPIC, AND NAMELY, THAT IS WITH RESPECT TO
22 SPECIES CONCORDANCE, AND NAMELY AS BETWEEN RATS AND MICE
23 AND WHAT CONCORDANCE YOU SAW IN VARIOUS TUMOR SITES BASED
24 UPON YOUR REVIEW OF THE N.T.P. STUDIES. OKAY?

25 A YES.

26 Q BASED UPON YOUR REVIEW OF THE N.T.P. STUDIES
27 OF ACRYLAMIDE AND GLYCIDAMIDE, THE SITES FOR WHICH THERE
28 WAS CONCORDANCE AS BETWEEN RATS AND MICE WERE TWO,

1 MAMMARY AND SKIN; CORRECT?

2 A I WOULD HAVE TO LOOK AT THE CHART IF I HAVE
3 IT HERE.

4 WHICH I DON'T.

5 NO, I DON'T.

6 THE COURT: LET'S COME BACK TO IT LATER. IF THE
7 WITNESS -- HOW MUCH LONGER ARE YOU GOING TO BE WITH THE
8 WITNESS?

9 MR. SCHURZ: NOT LONG, YOUR HONOR.

10 THE COURT: WILL YOU FINISH TODAY?

11 MR. SCHURZ: ABSOLUTELY. YES.

12 THE COURT: BY WHAT TIME?

13 MR. SCHURZ: NOW YOU ARE -- I BELIEVE WE WILL BE
14 DONE BY 4:00 O'CLOCK.

15 THE COURT: OKAY. I JUST WANTED TO ASK YOU
16 BECAUSE --

17 MR. SCHURZ: YOU HAVE GOT PEOPLE WAITING?

18 THE COURT: OTHER CASES.

19 MR. SCHURZ: I APPRECIATE THAT. WE ONLY HAVE A
20 COUPLE OF TOPICS THAT WE ARE GOING TO BE ADDRESSING WITH
21 DR. HUFF.

22 THE COURT: OKAY.

23 Q BY MR. SCHURZ: NOW, WITH THE HARDERIAN
24 GLANDS WHICH YOU TESTIFIED EARLIER WAS THE STRONGEST
25 APPEARANCE, THAT ONLY APPEARED IN THE MICE STUDIES;
26 CORRECT?

27 IT DID NOT APPEAR IN RATS?

28 A YES.

1 Q SO LET'S NOW TALK A LITTLE BIT ABOUT THE
2 TUMORS THAT YOU IDENTIFIED THAT ARE SPECIFIC TO RODENTS.

3 NOW, JUST BY WAY OF CLARIFYING, PEOPLE DO
4 NOT HAVE HARDERIAN GLANDS; CORRECT?

5 A THEY DO NOT HAVE HARDERIAN GLANDS AS AN
6 EXACT MIMIC OF WHAT ARE IN THE RODENTS, CORRECT.

7 Q PEOPLE DO NOT HAVE FORE-STOMACHS; DO THEY?

8 A NO, BUT THEY HAVE AN ORAL CAVITY AND
9 ESOPHAGUS, WHICH ARE THE SAME CELLULAR TYPE.

10 Q AND MICE ALSO HAVE ESOPHAGUS; CORRECT?
11 ESOPHAGI?

12 A YES.

13 Q PEOPLE DO NOT HAVE PREPUTIAL GLANDS; DO
14 THEY?

15 A THEY HAVE GLANDS OF THE PENIS THAT ARE
16 EXACTLY COMPARATIVE TO THE PREPUTIAL GLAND OF THE
17 RODENTS.

18 Q NOW, LET'S TALK A LITTLE BIT ABOUT THE HERP
19 INDEX AND THE CHART THAT YOU DISCUSSED WITH MR. METZGER.

20 IF WE COULD TAKE A LOOK, PLEASE, AT THE
21 DEMONSTRATIVE 86.

22 THIS IS A CHART YOU PREPARED, DR. HUFF,
23 IDENTIFYING CANCER RISK OF COFFEE COMPARED TO OTHER
24 FRUITS AND VEGETABLES; CORRECT?

25 A YES.

26 Q AND THIS WAS BASED UPON YOUR REVIEW OF THE
27 BRUCE AMES ARTICLE RANKING POSSIBLE CARCINOGENIC HAZARDS
28 THAT YOU HAVE IDENTIFIED HERE AT THE BOTTOM OF

1 DEMONSTRATIVE 86; CORRECT?

2 A YES.

3 Q SHOWING YOU NOW WHAT IS EXHIBIT 2517.

4 YOU HAVE RELIED ON THIS ARTICLE BY BRUCE
5 AMES IN PREPARING YOUR OPINIONS IN THIS MATTER; IS THAT
6 CORRECT?

7 A PARDON ME?

8 WOULD YOU REPEAT THAT.

9 Q HAVE YOU REVIEWED THIS ARTICLE AS PART OF --
10 IN DEVELOPING YOUR OPINIONS IN THIS MATTER?

11 A SOMETIME AGO I DID.

12 Q ALL RIGHT. NOW, YOU STATED THAT COFFEE HAS
13 THE HIGHEST HERP INDEX OF ALL FOODS EVALUATED; CORRECT?

14 A YES.

15 Q LET'S TAKE A MOMENT AND TAKE A LOOK AT TABLE
16 NO. 1 THAT APPEARS ON PAGE 3 OF EXHIBIT 2517.

17 DO YOU HAVE THAT IN FRONT OF YOU?

18 A YES.

19 Q DIRECTING YOUR ATTENTION DOWN TO THE MIDDLE
20 OF THE PAGE, DO YOU SEE THE VALUES THAT ARE REPORTED HERE
21 FOR THE POSSIBLE HAZARD HERP INDEX FOR BASIL, ONE GRAM OF
22 DRIED LEAF?

23 DO YOU SEE THAT?

24 A YES.

25 Q THAT INCLUDES A VALUE OF 0.10; CORRECT?

26 A 0.1, YES.

27 Q WHICH IS THE SAME VALUE AS COFFEE THAT YOU
28 HAVE REPORTED IN YOUR DEMONSTRATIVE 86; CORRECT?

1 A YES, THAT IS CORRECT.

2 Q IF WE TAKE A LOOK AT THE NEXT VALUE, IT
3 INDICATES THE HERP INDEX FOR ONE NEW MUSHROOM.
4 DO YOU SEE THAT?

5 A YES.

6 Q IT ALSO LISTS A HERP INDEX OF 0.10. THE
7 SAME VALUE THAT YOU HAVE CITED FOR COFFEE; CORRECT?

8 A YES.

9 Q SO IF WE GO DOWN, WE CAN LOOK FURTHER AND WE
10 CAN SEE A VALUE FOR WINE, FOR EXAMPLE, THAT INCLUDES A
11 VALUE -- A HERP INDEX VALUE OF 4.7; CORRECT?

12 A UH-HUH.

13 Q SO THAT WOULD BE 47 TIMES THE VALUE FOR
14 COFFEE; CORRECT?

15 A ACCORDING TO THE HERP INDEX, YES.

16 Q IF WE GO -- IF WE LOOK UP THE CHART, THERE
17 ARE A VARIETY OF OTHER FOODS, BUT LET'S TAKE A LOOK AT
18 THE VALUE FOR CONVENTIONAL HOME AIR AS REPORTED BY
19 DR. AMES.

20 HERE, HE LISTS A HERP INDEX OF 0.6 FOR
21 CONVENTIONAL HOME AIR MEASURED AT 14 HOURS PER DAY.

22 DO YOU SEE THAT?

23 A YES, WHERE HE BASES THIS ON THE
24 FORMALDEHYDE/BENZENE CONTENT.

25 Q AND THIS WOULD BE SIX TIMES THE VALUE THAT
26 YOU HAVE CITED FOR COFFEE; CORRECT?

27 A YES.

28 Q SO ROUGHLY TWO HOURS OF CONVENTIONAL HOME

1 AIR WOULD HAVE THE SAME CARCINOGENIC HERP INDEX AS A CUP
2 OF COFFEE; CORRECT?

3 A SAME HERP, YES.

4 NOW, THE REASON I DID NOT PUT THESE IN IS
5 BECAUSE I WAS CONCENTRATING ON FRUITS AND VEGETABLES. I
6 WASN'T CONCENTRATING ON THE AGENTS YOU HAD ALREADY -- YOU
7 HAD MENTIONED.

8 Q ALTHOUGH BASIL AND MUSHROOMS WOULD CERTAINLY
9 FALL WITHIN THAT CATEGORY; WOULD THEY NOT?

10 WE DON'T NEED TO ARGUE ABOUT FOODS. I AM
11 NOT INTERESTED. LET'S MOVE ON.

12 DIRECTING YOUR ATTENTION TO PAGE 1 OF THE
13 AMES ARTICLE RANKING POSSIBLE CARCINOGENIC HAZARDS, I
14 DIRECT YOUR ATTENTION TO THE ABSTRACT FIRST IN WHICH THE
15 AUTHORS INDICATE:

16 "THIS REVIEW DISCUSSES REASONS
17 WHY ANIMAL CANCER TESTS CANNOT BE
18 USED TO PREDICT ABSOLUTE HUMAN
19 RISKS."

20 CORRECT?

21 A NO, I SEE THAT SENTENCE -- YOU ARE -- YOU
22 READ IT CORRECTLY, YES.

23 Q ONE OF THE CONCERNS THAT AMES HAD WAS THAT
24 EXTRAPOLATION FROM THE RESULTS OF RODENT CANCER TESTS
25 DONE AT HIGH DOSES TO EFFECTS ON HUMANS EXPOSED TO LOW
26 DOSES IS ROUTINELY ATTEMPTED BY REGULATORY AGENCIES WHEN
27 FORMULATING POLICIES ATTEMPTING TO PREVENT FUTURE CANCER;
28 CORRECT?

1 A I AM NOT CERTAIN WHEN YOU ASK ME IF IT IS
2 CORRECT OR NOT. IS IT CORRECT HOW YOU READ IT OR IS IT
3 CORRECT HOW IT IS STATED?

4 Q IS THAT WHAT THE AUTHOR OF THE ARTICLE UPON
5 WHICH YOU ARE RELYING STATED WITH RESPECT TO HIS CONCERNS
6 ABOUT EXTRAPOLATION OF ANIMAL DATA TO ASSESS HUMAN CANCER
7 POTENTIAL?

8 A THAT IS WHAT HE SAID, YES.

9 Q OKAY. AND --

10 A MAY I RESPOND TO THAT RECITATION OF THAT
11 SENTENCE?

12 Q I AM SURE MR. METZGER WOULD BE PLEASED TO
13 GIVE YOU THAT OPPORTUNITY ON REDIRECT.

14 A SORRY.

15 THE COURT: NEXT QUESTION.

16 Q BY MR. SCHURZ: SO DIRECTING YOUR ATTENTION
17 TO PAGE 2 OF EXHIBIT 2517, AND I DIRECT YOUR ATTENTION TO
18 THE LEFT-HAND SIDE OF THE SECOND FULL PARAGRAPH WHERE THE
19 AUTHORS OBSERVE:

20 "IT WOULD BE A MISTAKE TO USE
21 OUR HERP INDEX AS A DIRECT ESTIMATE
22 OF HUMAN HAZARD. FIRST, AT LOW DOSE
23 RATES, HUMAN SUSCEPTIBILITY MAY
24 DIFFER SYSTEMATICALLY FROM RODENT
25 SUSCEPTIBILITY."

26 DO YOU SEE THAT?

27 A YES, I DO.

28 Q AND AMES AND HIS CO-AUTHORS WERE CONCERNED,

1 WERE THEY NOT, WITH RESPECT TO THE EXTRAPOLATION FROM
2 ANIMAL DATA IN HUMAN RISK ASSESSMENT; WERE THEY NOT?

3 A FROM THE SENTENCES THAT YOU HAVE READ, I
4 WOULD HAVE TO CONSIDER THAT CORRECT.

5 Q OKAY. AND THE AUTHORS GO ON AT SOME LENGTH
6 IN THIS TO DISCUSS VARIOUS FOODS AND THEIR CANCER HERP
7 INDEX, AND OBSERVE THAT, FOR EXAMPLE, A SANDWICH HAS A
8 HERP INDEX OF 0.4., WHICH IS ABOUT FOUR TIMES THE HERP
9 INDEX THAT YOU HAVE INDICATED HERE; IS THAT CORRECT?

10 A I DON'T KNOW WHERE YOU ARE READING FROM, BUT
11 IF WHAT YOU ARE READING IS CORRECT AND ACCURATE, THEN
12 CORRECT, FOUR TIMES.

13 Q WELL, DON'T TAKE MY WORD FOR IT. LET'S GO
14 TO PAGE 4 OF EXHIBIT 2517.

15 YOU SEE HERE, AND IT IS DISPLAYED ON THE
16 MONITOR AS WELL, DR. HUFF, THAT THEY DISCUSS HERE THAT A
17 SANDWICH HAS A HERP INDEX WITH TWO SLICES OF BREAD OF .4,
18 OR STATED DIFFERENTLY, FOUR TIMES THAT ASSOCIATED WITH
19 COFFEE; CORRECT?

20 A YES, I SEE THAT.

21 Q AND COLA HAS A HERP INDEX OF 2.7, OR 27
22 TIMES THAT THAT THEY CITE FOR COFFEE; CORRECT?

23 A I SEE THAT.

24 Q FINALLY, DIRECTING YOUR ATTENTION TO THE
25 LAST PAGE OF 2517, THE AUTHORS OFFER SOME IMPLICATIONS
26 WITH RESPECT TO DECISION MAKING.

27 I DIRECT YOUR ATTENTION TO THE SECOND FULL
28 PARAGRAPH.

1 A THE LAST PAGE, PAGE 10?

2 Q IT IS THE LAST PAGE OF EXHIBIT 2517.

3 A SORRY, THE LAST PAGE I HAVE FOR 02517 IS 10.

4 Q DO YOU SEE "IMPLICATIONS FOR DECISION
5 MAKING"?

6 A NO, I AM SORRY, I DON'T SEE THAT.

7 Q PAGE 7.

8 I APOLOGIZE, PAGE 7. I MISLED YOU.

9 A YES, I SEE PAGE 7.

10 Q HERE, THE AUTHORS OBSERVE:

11 "THUS, IT IS NOT SCIENTIFICALLY
12 CREDIBLE TO USE THE RESULTS FROM
13 RODENT TESTS DONE AT THE MAXIMUM
14 TOLERABLE DOSE TO DIRECTLY ESTIMATE
15 HUMAN RISKS AT LOW DOSES."

16 CORRECT?

17 A THAT IS WHAT IT SAYS, YES.

18 Q I UNDERSTAND THAT YOU TAKE ISSUE WITH
19 DR. AMES'S ASSESSMENTS OR USE OF ANIMAL DATA IN HUMAN
20 RISK ASSESSMENTS; IS THAT CORRECT?

21 A YES.

22 MR. SCHURZ: THANK YOU, YOUR HONOR. I HAVE NOTHING
23 FURTHER.

24 THE COURT: MR. METZGER, ANY REDIRECT?

25 MR. METZGER: I THINK JUST ONE QUESTION.

26 THE COURT: OKAY, ONE QUESTION.

27

28

1 REDIRECT EXAMINATION

2
3 BY MR. METZGER:4 Q WITH RESPECT TO ACRYLAMIDE, TO ASSESS THE
5 HUMAN RISK OF CANCER FROM ACRYLAMIDE BASED ON THE ANIMAL
6 DATA, MUST ONE EXTRAPOLATE SOLELY FROM THE MAXIMUM
7 TOLERATED DOSE?

8 A NO.

9 Q WHY NOT?

10 A BECAUSE AS SHOWN WITH THE ACRYLAMIDE DATA,
11 THAT IS NOT THE ONLY RESPONSE THAT WE SAW WITH THOSE
12 STUDIES. WE SAW CARCINOGENIC RESPONSES AT THE TWO LOWER
13 DOSES AS WELL.14 Q INCLUDING THE DOSES AS LOW AS -- WAS IT
15 WHAT?

16 A SIX PARTS PER MILLION.

17 Q OKAY, AND SIX PARTS PER MILLION I THINK YOU
18 INDICATED WAS THE EQUIVALENT OF -- WOULD THAT BE SIX
19 PEOPLE IN TEN ROSE BOWLS OR SOMETHING?

20 A YES.

21 MR. METZGER: OKAY. I HAVE NO FURTHER QUESTIONS.

22 THANK YOU, YOUR HONOR.

23 THE COURT: MAY THE WITNESS BE EXCUSED?

24 MR. METZGER: YES.

25 MR. SCHURZ: YES, YOUR HONOR.

26 THE COURT: DR. HUFF, YOU MAY BE EXCUSED.

27 THE WITNESS: THANK YOU, YOUR HONOR.

28 THE COURT: MR. METZGER, ANY FURTHER WITNESSES?

1 MR. METZGER: WE DO HAVE ONE ADDITIONAL WITNESS.
2 THAT IS DR. STEVEN BAYARD. HE WILL BE ARRIVING IN COURT
3 HERE FOR MONDAY. HE IS THE QUANTITATIVE CANCER RISK
4 EXPERT.

5 THE COURT: HE IS THE LAST WITNESS FOR THE
6 PLAINTIFF?

7 MR. METZGER: HE IS THE LAST WITNESS FOR THE
8 PLAINTIFF. WE DO HAVE OTHER MATTERS TO GET INTO EVIDENCE
9 THOUGH.

10 IN PARTICULAR, I WOULD LIKE TO BRING TO THE
11 COURT'S ATTENTION THAT WE HAVE FILED AN OBJECTION, IT IS
12 REALLY A MOTION, TO TOTALLY EXCLUDE THE TESTIMONY BY
13 JULIE GOODMAN, WHO IS THEIR REBUTTAL WITNESS. I WOULD
14 LIKE -- WE APPARENTLY HAVE PLENTY OF TIME BEFORE SHE IS
15 COMING OUT HERE. I WOULD LIKE THE COURT TO RULE ON THAT
16 BECAUSE IT GOES TO THE ENTIRETY OF HER TESTIMONY. I
17 THINK IT SHOULD BE DONE IN ADVANCE, RATHER THAN AS A
18 MOTION TO STRIKE AFTER THE TESTIMONY HAS BEEN RECEIVED.

19 THE COURT: FIRST OF ALL, DR. BAYARD IS GOING TO BE
20 HERE ON MONDAY AT 9:00 A.M.?

21 MR. METZGER: YES, YOUR HONOR.

22 THE COURT: HOW LONG IS HIS TESTIMONY GOING TO
23 TAKE?

24 MR. METZGER: I EXPECT THAT HIS TESTIMONY WILL
25 PROBABLY TAKE TWO DAYS BETWEEN THE TWO OF US. HE IS
26 DOING TWO QUANTITATIVE CANCER RISK ASSESSMENTS, SO IT IS
27 A LOT OF GROUND TO COVER.

28 THE COURT: MR. SCHURZ, DO YOU AGREE WITH THAT

1 ESTIMATE OF TWO DAYS?

2 MR. SCHURZ: I HAVE NO IDEA HOW LONG MR. METZGER IS
3 GOING TO TAKE, YOUR HONOR, BUT WE HAVE COMMUNICATED TO
4 THE COURT, AND OUR ESTIMATE STILL STANDS, THAT WE BELIEVE
5 THAT THE CROSS-EXAMINATION OF DR. BAYARD WILL BE FOUR
6 HOURS.

7 MR. METZGER: SO IT MIGHT TAKE MORE THAN TWO DAYS.
8 TWO AND A HALF DAYS. WE WILL BE DONE BY WEDNESDAY, I AM
9 CERTAIN.

10 THE COURT: OKAY. NOW, AND DEFENDANTS, WHAT
11 REBUTTAL WITNESSES DO YOU HAVE, IF ANY?

12 MR. SCHURZ: YOUR HONOR, WE HAVE INDICATED THAT WE
13 ARE PROPOSING DR. JULIE GOODMAN AS A REBUTTAL EXPERT. AT
14 THIS TIME, SHE IS THE ONE THAT FOR WHICH WE ARE CERTAIN.
15 WE DON'T BELIEVE THAT IT WILL BE NECESSARY TO RECALL
16 DR. MURRAY, BUT HAVING NOT HEARD WHAT DR. BAYARD IS GOING
17 TO SAY, WE WOULD LIKE TO RESERVE THE OPPORTUNITY TO HAVE
18 HIM COME OUT AGAIN.

19 AT THE CURRENT TIME, WE ANTICIPATE CALLING
20 DR. GOODMAN.

21 THE COURT: ALL RIGHT. AND HOW LONG DO YOU THINK
22 DR. GOODMAN'S TESTIMONY IS GOING TO TAKE?

23 MR. SCHURZ: A DAY. ONE DAY.

24 THE COURT: AND IS SHE COMING FROM OUT OF TOWN?

25 MR. SCHURZ: YES, SHE IS COMING FROM BOSTON.

26 THE COURT: ALL RIGHT. AND HAVE YOU FILED ANY
27 OPPOSITION TO PLAINTIFF'S MOTION?

28 MR. SCHURZ: NO, WE HAVE NOT, YOUR HONOR. AND WE

1 EXPECT TO.

2 THE COURT: WHEN DO YOU EXPECT TO HAVE THAT
3 OPPOSITION?

4 MS. CORASH: MONDAY.

5 MR. SCHURZ: WE WOULD ASK TO PROVIDE THAT TO THE
6 COURT ON MONDAY.

7 THE COURT: THAT IS KIND OF LATE. TODAY IS -- HOW
8 ABOUT FRIDAY SO THEN WE COULD DISCUSS IT MONDAY?

9 HAVE A HEARING ON MONDAY AND MS. GOODMAN
10 DOES NOT HAVE TO GET ON A PLANE UNTIL AS EARLY AS TUESDAY
11 ANYWAY.

12 MR. SCHURZ: WE WILL HAVE IT TO YOU FRIDAY.

13 THE COURT: SO DEFENDANTS TO FILE THEIR OPPOSITION
14 FRIDAY, AND THEN WE WILL HAVE THE HEARING ON THE MOTION
15 MONDAY MORNING.

16 MR. SCHURZ: VERY GOOD, YOUR HONOR. THANK YOU.

17 MR. METZGER: THERE ARE A FEW --

18 THE COURT: ANY OTHER ISSUES YOU WISH TO ADDRESS?

19 MR. METZGER: THERE ARE STILL SOME OUTSTANDING
20 REQUESTS FOR JUDICIAL NOTICE THAT WE NEED YOUR HONOR TO
21 RULE ON, AND ALSO THE P.M.K. TESTIMONY THAT NEEDS TO BE
22 ADDRESSED. SO WE CAN MAKE A LIST OF THE SPECIFIC
23 DOCUMENTS THAT YOU HAVE NOT YET RULED ON AS TO JUDICIAL
24 NOTICE. THESE WERE SUBMITTED EARLY ON, BUT YOU HAVE NOT
25 RULED ON.

26 THE COURT: ALL RIGHT. PLEASE, COUNSEL, SUBMIT A
27 JOINT SUBMISSION WITH REGARD TO OPEN MATTERS. I WOULD
28 ASK THAT YOU DO THAT BY FRIDAY AT NOON SO THEN I WILL

1 HAVE READING MATERIAL TO TAKE HOME OVER THE WEEKEND.

2 MR. SCHURZ: THANK YOU, YOUR HONOR.

3 MR. METZGER: THANK YOU, YOUR HONOR.

4 THE COURT: ALL RIGHT. HAVE A GOOD WEEKEND. WE
5 WILL SEE YOU MONDAY MORNING.

6 MR. METZGER: VERY GOOD.

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8 (THE MATTER WAS ADJOURNED AT 3:54 P.M.)

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SUPERIOR COURT OF THE STATE OF CALIFORNIA

FOR THE COUNTY OF LOS ANGELES

DEPARTMENT 323

HON. ELIHU M. BERLE, JUDGE

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

PLAINTIFF,)

VS.)

CASE NO.
BC435759

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

DEFENDANTS.)

AND CONSOLIDATED ACTION.)

_____)

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 192 COMPRISE
A FULL, TRUE AND CORRECT TRANSCRIPT OF THE TESTIMONY AND
PROCEEDINGS HELD IN THE ABOVE-ENTITLED MATTER ON
WEDNESDAY, OCTOBER 22, 2014.

DATED THIS 22ND DAY OF OCTOBER, 2014.

KAREN VILICICH, CSR NO. 7634
OFFICIAL REPORTER PRO TEMPORE

EXHIBIT “E”

JAMES HUFF BIOGRAPHY

Dr. Huff earned his Doctor of Philosophy Degree in Bionucleonics, with emphasis on pharmacologic mechanisms, in 1968 from Purdue University, West Lafayette, Indiana. His Ph.D. thesis: "A tracer investigation of the metabolism, distribution, and excretion of amobarbital in stressed and non-stressed rats."

After an 18 month post-doctoral appointment at the Federation of American Societies for Experimental Biology [FASEB] in Bethesda, Maryland, Dr. Huff joined Dr. Harold Hodge on the faculty of the University of Rochester Medical School, Rochester, New York as an assistant professor in the Department of Pharmacology and Toxicology. He was involved in the clinical toxicology of commercial products, effects of high altitudes on erythropoietin activity, and the persistence of cholinesterase effects of carbamate pesticides. In 1973 he began work in the Biology Division, Oak Ridge National Laboratory, Oak Ridge, Tennessee. He developed and was Chief of four units involved in the assessment of risks associated with the broad scope of toxicologic activities of chemicals and other environmental and occupational exposures. During that time he was involved with Dr. Henry Kissman at the National Library of Medicine in building the online TOXLINE data files.

In 1977 Dr. Huff became Chief of the IARC Monographs Program on the Evaluation of Carcinogenic Risks to Humans of the International Agency for Research on Cancer, Lyon, France. While at IARC, working with Dr Lorenzo Tomatis, he helped to establish the initial categories used within the IARC Monographs Program to classify exposures according to the epidemiologic and experimental evidence of carcinogenicity. Further, Dr. Huff was instrumental in strengthening and expanding the scientific content and carcinogenesis evaluations of the Monographs Program.

Returning to the United States in 1980 at the behest of Dr. David Rall, he joined the National Institute of Environmental Health Sciences and the National Toxicology Program. Dr. Huff was the first person hired by Dr. Rall, primarily to help establish the newly created NTP into a world-renowned scientific and public health-oriented program. He was a lead person for the transition of the bioassay program from the National Cancer Institute to the NTP. Dr. Huff introduced and established the NTP levels of evidence of carcinogenicity for the experimental chemical carcinogenesis bioassays for evaluating results, and as still utilized in the bioassay technical reports. While with the NTP he wrote or led the preparation and evaluation of more than 200 carcinogenesis bioassay technical reports. Along with Dr Hans Falk and later joined by Dr Vladimer Vouk, Dr Huff helped establish the Congressionally mandated NTP Report on Carcinogens, now in its 10th edition, into a science-based and globally accepted collection of chemicals and exposure circumstances considered as either known to be as carcinogenic to humans or as reasonably anticipated to be carcinogenic to humans.

Dr. Huff currently resides in the Office of the Director, NIEHS. His major research interests center on chemical carcinogenesis and their possible impact on environmental, occupational, and general public health. These activities include: conducting and evaluating long-term chemical carcinogenesis bioassays; exploring mechanisms of carcinogenic activity; identifying potential human carcinogens; conducting, evaluating, and refining in vitro and in vivo systems to improve cancer hazard predictions and risk assessments; pursuing environmental, occupational, and lifestyle causes of cancer; and examining issues and controversies in the quest to improved public health. He continues these activities by continuing to commit and dedicate his expertise, energy, and experience to reducing the cancer burden from chemicals and other environmental carcinogenic exposure circumstances.

Dr. Huff is an elected member of the Collegium Ramazzini, an international community of 180 scholars in honor of Bernardino Ramazzini, to advance the study of occupational and environmental health issues around the world. Ramazzini Days are held annually in his birthplace Carpi, Italy. Dr. Huff has been an invited speaker, chairperson, or organizer at numerous national and international workshops, symposia, and conferences, and has authored or coauthored upwards of 300 scientific publications. In addition to these, and the nearly 350 corporate toxicology documents, IARC Monographs, and NTP Technical Reports, he initiated and was the lead editor on a book on hormonal carcinogenesis: Huff J, Boyd J, Barrett JC [Editors]. Cellular and molecular mechanisms of hormonal carcinogenesis: environmental influences. *Prog Clin Biol Res* 1996;394:i-xix; 1-479.

For 2002, Dr Huff has been selected by the 55,000-member American Public Health Association to receive the David P Rall Award for Advocacy in Public Health for his long-term and consistent efforts to raise awareness about the reduction and prevention of environmentally associated diseases, especially exposure to environmental carcinogens. According to the APHA, the David Rall Award "recognizes individuals who have made outstanding contributions to public health through science-based advocacy."

Selected Publications

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EXHIBIT “F”

James Huff - Bibliography by Year Published

Numbers of Publications by Year -- Latest First

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- 2011 - [6]	- 1999 - [8]	- 1987 - [8]	- 1975 - [8]
- 2010 - [4]	- 1998 - [7]	- 1986 - [15]	- 1974 - [8]
- 2009 - [4]	- 1997 - [7]	- 1985 - [10]	- 1973 - [1]
- 2008 - [9]	- 1996 - [10]	- 1984 - [14]	- 1972 - [1]
- 2007 - [8]	- 1995 - [8]	- 1983 - [12]	- 1971 - [1]
- 2006 - [5]	- 1994 - [15]	- 1982 - [29]	- 1970 - [3]
- 2005 - [7]	- 1993 - [17]	- 1981 - [2]	- 1969 - [7]
- 2004 - [4]	- 1992 - [15]	- 1980 - [16]	- 1968 - [4]
- 2003 - [10]	- 1991 - [19]	- 1979 - [10]	- 1965 - [1]
- 2002 - [9]	- 1990 - [20]	- 1978 - [14]	
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