

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

Published Name:

CERT's Submission No. 10 regarding the Opinions of Dr. Ronald L. Melnick regarding Technologies for Reducing Acrylamide in Coffee.

Email:

nvidal@toxic torts.com

Post date:

08/15/2018 - 3:45pm

# Metzger Law Group

Practice Concentrated in Toxic  
Tort & Environmental Litigation

401 E Ocean Blvd., Ste. 800  
Long Beach, CA 90802  
phone: 562.437.4499  
fax: 562.436.1561

www.toxic torts.com

Raphael Metzger  
Brian Foster  
Abraham Pariser  
Robyn Mallon  
Monica Frye  
Scott Brust

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. 10**

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. Ronald L. Melnick regarding Technologies for Reducing Acrylamide in Coffee.

1. Exhibit A - Declaration of Dr. Ronald L. Melnick in Support of Plaintiff's motion for Summary Adjudication of Defendants' Alternative Significant Risk Level ("ASRL") Defense (May 16, 2016).
2. Exhibit B - Critique of Dr. William Risternpart's Report and Testimony (2017).
3. Exhibit C - Testimony of Ronald L. Melnick in *CERT v. Starbucks* trial, October 2, 2017 a.m.
4. Exhibit D - Testimony of Ronald L. Melnick in *CERT v. Starbucks* trial, October 2, 2017 p.m.
5. Exhibit E - Testimony of Ronald L. Melnick in *CERT v. Starbucks* trial, October 3, 2017 a.m.

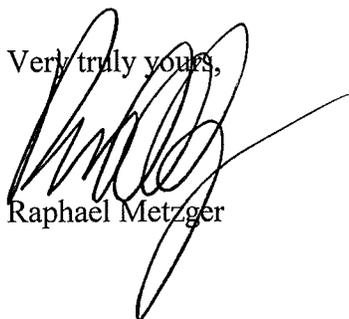
Monet Vela  
OEHHA  
August 15, 2018  
Page 2

---

6. Exhibit F - Curriculum Vitae of Ronald L. Melnick, Ph.D.

Kindly include these materials of Dr. Ronald L. Melnick 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”**



TELEPHONE (562) 437-4499  
TOLL-FREE (877) TOX-TORT  
TELECOPIER (562) 436-1561  
WWW.TOXICTORTS.COM

1  
2  
3  
4  
5  
6  
7  
8  
9  
10  
11  
12  
13  
14  
15  
16  
17  
18  
19  
20  
21  
22  
23  
24  
25  
26  
27  
28

METZGER LAW GROUP  
A PROFESSIONAL LAW CORPORATION  
RAPHAEL METZGER, ESQ., SBN 116020  
KATHRYN A. SALDANA, ESQ., SBN 251364  
401 E. OCEAN BLVD., SUITE 800  
LONG BEACH, CA 90802-4966  
TELEPHONE: (562) 437-4499  
TELECOPIER: (562) 436-1561  
WEBSITE: www.toxictorts.com

Attorneys for Plaintiff,  
Council for Education and  
Research on Toxics ("CERT")

SUPERIOR COURT OF THE STATE OF CALIFORNIA  
FOR THE COUNTY OF LOS ANGELES, CENTRAL CIVIL WEST

LAW OFFICES OF  
RAPHAEL METZGER  
A PROFESSIONAL LAW CORPORATION  
401 EAST OCEAN BOULEVARD, SUITE 800  
LONG BEACH, CALIFORNIA 90802-4966

COUNCIL FOR EDUCATION AND )  
RESEARCH ON TOXICS, a California )  
corporation, acting as a private )  
attorney general in the public )  
interest; )  
 )  
Plaintiff, )  
 )  
vs. )  
 )  
STARBUCKS CORPORATION, a )  
Washington corporation; et al., )  
 )  
Defendants. )

CASE NO. BC435759  
Consolidated with Case No.  
BC461182  
*Assigned to the Honorable Elihu  
Berle, Dept. 323*

DECLARATION OF DR. RONALD L.  
MELNICK IN SUPPORT OF  
PLAINTIFF'S MOTION FOR SUMMARY  
ADJUDICATION OF DEFENDANTS'  
ALTERNATIVE SIGNIFICANT RISK  
LEVEL ("ASRL") DEFENSE

[Filed concurrently with Notice  
of Motion; Memorandum of Points  
and Authorities; Separate  
Statement of Undisputed Facts;  
Requests for Judicial Notice;  
Declaration of Raphael Metzger;  
and Proposed Order]

DATE: August 5, 2016  
TIME: 1:30 p.m.  
DEPT: 323

PRACTICE CONCENTRATED IN TOXIC  
TORT & ENVIRONMENTAL LITIGATION  
OCCUPATIONAL & ENVIRONMENTAL LUNG  
DISEASE, CANCER, AND TOXIC INJURIES

DECLARATION OF DR. RONALD L. MELNICK IN SUPPORT OF PLAINTIFF'S  
MOTION FOR SUMMARY ADJUDICATION OF DEFENDANTS' ALTERNATIVE  
SIGNIFICANT RISK LEVEL ("ASRL") DEFENSE

TELEPHONE (562) 437-4499  
TOLL-FREE (877) TOX-TORT  
TELECOPIER (562) 436-1561  
WWW.TOXICTORTS.COM

LAW OFFICES OF  
**RAPHAEL METZGER**  
A PROFESSIONAL LAW CORPORATION  
401 EAST OCEAN BOULEVARD, SUITE 800  
LONG BEACH, CALIFORNIA 90802-4966

PRACTICE CONCENTRATED IN TOXIC  
TORT & ENVIRONMENTAL LITIGATION  
OCCUPATIONAL & ENVIRONMENTAL LUNG  
DISEASE, CANCER, AND TOXIC INJURIES

1  
2  
3  
4  
5  
6  
7  
8  
9  
10  
11  
12  
13  
14  
15  
16  
17  
18  
19  
20  
21  
22  
23  
24  
25  
26  
27  
28

DECLARATION OF DR. RONALD L. MELNICK

I, Dr. Ronald L. Melnick, declare as follows:

1. I am a food scientist, toxicologist, and environmental risk assessor.

2. I have personal knowledge of the matters set forth hereinafter and, if called as a witness, I would competently testify thereto.

3. I have been requested by Raphael Metzger, counsel for Plaintiff, Council for Education and Research on Toxics (CERT), to inform the court whether companies can reduce acrylamide in coffee to levels that would result in exposures that would not exceed the No Significant Risk Level, without negatively impacting the sensorial properties of coffee, i.e., without rendering coffee "unpalatable."

4. To undertake this task, I undertook a thorough review of the published, peer-reviewed literature regarding the formation of acrylamide in coffee, available technologies and methods to reduce acrylamide levels in coffee (with special attention to issues of palatability and feasibility), and information as to how the potato industry has addressed the problem of acrylamide in potato products.

5. As explained in detail below, numerous technologies are available to reduce the acrylamide content of coffee, many of which have been shown to substantially reduce acrylamide levels in coffee, i.e., to reduce acrylamide levels in coffee as much as 90%. Additional reductions of acrylamide levels in coffee could likely be achieved by combining methods that are effective in preventing acrylamide formation and in removing acrylamide from roasted and ground coffee beans. Further, several of the technologies have

TELEPHONE (562) 437-4499  
TOLL-FREE (877) TOX-TORT  
TELECOPIER (562) 436-1561  
WWW.TOXICTORTS.COM

LAW OFFICES OF  
**RAPHAEL METZGER**  
A PROFESSIONAL LAW CORPORATION  
401 EAST OCEAN BOULEVARD, SUITE 800  
LONG BEACH, CALIFORNIA 90802-4966

PRACTICE CONCENTRATED IN TOXIC  
TORT & ENVIRONMENTAL LITIGATION  
OCCUPATIONAL & ENVIRONMENTAL LUNG  
DISEASE, CANCER, AND TOXIC INJURIES

1  
2  
3  
4  
5  
6  
7  
8  
9  
10  
11  
12  
13  
14  
15  
16  
17  
18  
19  
20  
21  
22  
23  
24  
25  
26  
27  
28

either been shown not to negatively impact the sensorial properties of coffee or, due to their nature, would unlikely render coffee unpalatable.

**QUALIFICATIONS**

6. I received a B.S. in Food Science from Rutgers University in 1965 and a M.S. and Ph.D. in Food Science/Biochemistry from the University of Massachusetts in 1967 and 1970, respectively.

7. Upon completing my doctorate, I did a postdoctoral fellowship in the Department of Physiology-Anatomy at the University of California at Berkeley, and served as an Assistant Professor of Life Sciences at the Polytechnic Institute of New York.

8. Between September 1980 and January 2009, I was a senior toxicologist in the National Toxicology Program (NTP) at the National Institute of Environmental Health Sciences (NIEHS) in Research Triangle Park, North Carolina. NIEHS is one of the twenty-five institutes and centers that comprise the National Institutes of Health (NIH), which is a component of the U.S. Department of Health and Human Services. The mission of the NIEHS is to reduce the burden of human illness and disability by understanding how environment factors influence the development and progression of human disease. Toward this end, the NTP and NIEHS have developed research programs to characterize health effects of environmental agents and investigate mechanisms of environmental associated diseases.

9. During my more than 28 years at NTP/NIEHS, I was responsible for the design and interpretation of numerous toxicity and carcinogenicity studies conducted by the NTP and I was the

TELEPHONE (562) 437-4499  
TOLL-FREE (877) TOX-TORT  
TELECOPIER (562) 436-1561  
WWW.TOXICTORTS.COM

LAW OFFICES OF  
**RAPHAEL METZGER**  
A PROFESSIONAL LAW CORPORATION  
401 EAST OCEAN BOULEVARD, SUITE 800  
LONG BEACH, CALIFORNIA 90802-4966

PRACTICE CONCENTRATED IN TOXIC  
TORT & ENVIRONMENTAL LITIGATION  
OCCUPATIONAL & ENVIRONMENTAL LUNG  
DISEASE, CANCER, AND TOXIC INJURIES

1 principal author of technical reports of NTP toxicity/carcinogenicity  
2 studies as well as of manuscripts of studies published in scientific  
3 journals. I also served on numerous in-house and external  
4 committees.

5 10. One activity worth noting is my role as chair of the  
6 NIEHS review group for the NTP's Report on Carcinogens (RoC). This  
7 report, which is mandated by Congress, provides a compilation of  
8 health effects and exposure information on environmental agents  
9 judged to be known or likely human carcinogens. The NIEHS group that  
10 I chaired reviewed and evaluated all of the published human cancer  
11 data, animal data, and mechanistic data on nominated compounds and  
12 made recommendations for listing each agent as "known human  
13 carcinogen" or "reasonably anticipated to be a human carcinogen," or  
14 for not listing in the RoC. All nominations for listing or delisting  
15 in the Report were also reviewed by an interagency Federal scientific  
16 review group and by an external peer review committee.

17 11. I also spent one year at the White House Office of  
18 Science and Technology Policy (1995-1996) where I interacted with top  
19 officials at regulatory agencies on risk assessment issues.

20 12. I have also served on several scientific working  
21 groups and advisory committees for national and international  
22 agencies that have evaluated human health effects associated with  
23 exposure to toxic or carcinogenic agents and identified research  
24 needs to better characterize human health risks.

25 13. Since my retirement from NIEHS in January 2009, I have  
26 continued to review scientific articles submitted for journal  
27 publication and I have served as an expert reviewer and advisor of  
28 cancer risk assessments and research programs for the International

TELEPHONE (562) 437-4499  
TOLL-FREE (877) TOX-TORT  
TELECOPIER (562) 436-1561  
WWW.TOXICTORTS.COM

LAW OFFICES OF  
**RAPHAEL METZGER**  
A PROFESSIONAL LAW CORPORATION  
401 EAST OCEAN BOULEVARD, SUITE 800  
LONG BEACH, CALIFORNIA 90802-4966

PRACTICE CONCENTRATED IN TOXIC  
TORT & ENVIRONMENTAL LITIGATION  
OCCUPATIONAL & ENVIRONMENTAL LUNG  
DISEASE, CANCER, AND TOXIC INJURIES

1  
2  
3  
4  
5  
6  
7  
8  
9  
10  
11  
12  
13  
14  
15  
16  
17  
18  
19  
20  
21  
22  
23  
24  
25  
26  
27  
28

Agency for Research on Cancer, the US Environmental Protection Agency, and the European Commission.

14. My qualifications and experience are fully described in my Curriculum Vitae, a copy of which is attached as Exhibit "A".

**ACRYLAMIDE**

15. Acrylamide is a well-known neurotoxin in humans and experimental animals; it has also been identified as a reproductive toxicant in animal studies. Acrylamide and its primary oxidative metabolite, glycidamide - a DNA reactive epoxide - are genotoxic in most in vitro and in vivo systems, causing gene mutations and chromosomal aberrations.

16. Experimental studies in laboratory animals have consistently demonstrated that acrylamide is a multi-organ site carcinogen in rats and mice. Consequently, IARC, USEPA, and the NTP have classified acrylamide as a "reasonably anticipated" or "probable human carcinogen."

**ACRYLAMIDE IN FOOD**

17. The discovery that acrylamide is formed in various cooked foods has led to national and international concerns of cancer risk, as well as a burst in scientific investigations on acrylamide formation in heated foods and the mechanisms involved in its carcinogenicity. The highest levels of acrylamide were measured in carbohydrate rich foods, such as potatoes, that are heated at high temperatures (Tareke et al., 2002).



TELEPHONE (562) 437-4499  
TOLL-FREE (877) TOX-TORT  
TELECOPIER (562) 436-1561  
WWW.TOXICTORTS.COM

LAW OFFICES OF  
**RAPHAEL METZGER**  
A PROFESSIONAL LAW CORPORATION  
401 EAST OCEAN BOULEVARD, SUITE 800  
LONG BEACH, CALIFORNIA 90802-4966

PRACTICE CONCENTRATED IN TOXIC  
TORT & ENVIRONMENTAL LITIGATION  
OCCUPATIONAL & ENVIRONMENTAL LUNG  
DISEASE, CANCER, AND TOXIC INJURIES

1 provide any known health benefit or any organoleptic necessity or  
2 value.

3 22. Regrettably, levels of acrylamide in coffee appear to  
4 be increasing in most countries. An increase of the acrylamide  
5 levels in coffee (including instant coffee and coffee substitutes)  
6 was observed in the European acrylamide database from 2007 to 2010  
7 (EFSA, 2012). In a recent study in Belgium, acrylamide levels in  
8 potato crisps and gingerbread had decreased significantly, while the  
9 average acrylamide measured in coffee between 2008 and 2013 was  
10 almost twice as high as the average measured between 2002 and 2007  
11 (Claeys et al., 2016). However, "the acrylamide levels measured in  
12 coffee and coffee substitutes on the British market, with an average  
13 level of 430 ug/kg (maximum of 1056 ug/kg; 40 samples taken in 2012 -  
14 2013), were far below the levels measured in the Belgian study or  
15 reported in the compiled EFSA database, indicating that lower levels  
16 could somehow be achievable for coffee products." (Claeys et al.,  
17 2016).

#### 18 HUMAN EXPOSURE TO ACRYLAMIDE FROM COFFEE CONSUMPTION

19  
20  
21 23. Coffee is the most widely consumed beverage in the  
22 world, the U.S., and in California. According to the National Coffee  
23 Association, the average coffee consumer drinks more than 3 cups of  
24 coffee per day. As Dr. Stephen Bayard's quantitative risk assessment  
25 presented during the 2014 trial showed, exposure to acrylamide from  
26 coffee substantially exceeds California's No Significant Risk Level.

27 24. There is a crucial public health need to reduce human  
28 exposure to acrylamide from coffee. Numerous publications in the

TELEPHONE (562) 437-4499  
TOLL-FREE (877) TOX-TORT  
TELECOPIER (562) 436-1561  
WWW.TOXICTORTS.COM

LAW OFFICES OF  
**RAPHAEL METZGER**  
A PROFESSIONAL LAW CORPORATION  
401 EAST OCEAN BOULEVARD, SUITE 800  
LONG BEACH, CALIFORNIA 90802-4966

PRACTICE CONCENTRATED IN TOXIC  
TORT & ENVIRONMENTAL LITIGATION  
OCCUPATIONAL & ENVIRONMENTAL LUNG  
DISEASE, CANCER, AND TOXIC INJURIES

1 scientific literature, as well as company patents, describe methods  
2 that substantially reduce acrylamide levels in coffee. Of course,  
3 effective methods should not negatively affect the flavor and aroma  
4 of this popular and widely consumed beverage.

5 25. As shown below, many treatments that are effective in  
6 reducing acrylamide levels only minimally affect the organoleptic  
7 properties of coffee.

#### 8 9 **APPROACHES TO REDUCE ACRYLAMIDE LEVELS IN COFFEE**

10  
11 26. Mitigation strategies to reduce acrylamide levels in  
12 foods generally include selecting plant varieties with low levels of  
13 acrylamide precursors, genetic modification of plant varieties to  
14 prevent or reduce the formation of acrylamide precursors, removing  
15 or reducing precursors before processing, using asparaginase to  
16 catalyze the hydrolysis of asparagine, selecting process conditions  
17 that minimize acrylamide formation while maintaining desirable  
18 nutritional and sensory properties, adding food-compatible compounds  
19 that inhibit acrylamide formation during processing or that react  
20 with acrylamide, and removing acrylamide after it has formed  
21 (Friedman and Levin, 2008; Friedman, 2015). This section describes  
22 methods that have been evaluated for their effectiveness in reducing  
23 acrylamide levels in coffee in the published literature and company  
24 patents.

#### 25 26 **Selection of plant varieties**

27 27. The two main species of coffee plants commercially  
28 cultivated are *Coffea arabica* (the most highly regarded species) and

TELEPHONE (562) 437-4499  
TOLL-FREE (877) TOX-TORT  
TELECOPIER (562) 436-1561  
WWW.TOXICORTS.COM

LAW OFFICES OF  
**RAPHAEL METZGER**  
A PROFESSIONAL LAW CORPORATION  
401 EAST OCEAN BOULEVARD, SUITE 800  
LONG BEACH, CALIFORNIA 90802-4966

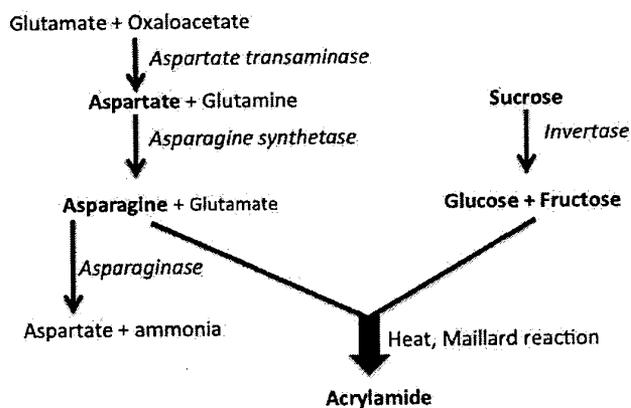
PRACTICE CONCENTRATED IN TOXIC  
TORT & ENVIRONMENTAL LITIGATION  
OCCUPATIONAL & ENVIRONMENTAL LUNG  
DISEASE, CANCER, AND TOXIC INJURIES

1 *Coffea canephora* (predominantly a form known as "robusta"). *Robusta*  
2 brewed coffee tends to be bitter and have less flavor than *arabica*.  
3 *Robusta* strains contain about 40-50% more caffeine than *arabica*.  
4 Consequently, *robusta* is used as an inexpensive substitute for  
5 *arabica* in many commercial coffee blends. Not only is *robusta* of  
6 inferior quality to *arabica*; it contains about twice the asparagine  
7 levels as *arabica*. As a result, coffee brewed from *robusta* beans  
8 that had been roasted for 7.5 minutes at 240°C was found to contain  
9 approximately double that amount of acrylamide (mean = 708 ng/g)  
10 compared to coffee brewed from *arabica* beans (mean = 374 ng/g).  
11 (Bagdonaite et al., 2008). Thus, by exclusively using higher quality  
12 *arabica* beans instead of *robusta* or *arabica-robusta* blends,  
13 acrylamide levels in brewed coffee can be reduced by almost 50%.  
14

#### Altered gene expression

15  
16 28. Recent approaches to reduce acrylamide in processed  
17 foods have focused on reducing or eliminating acrylamide precursors  
18 by enzyme treatments or altering the genetic properties of the plant.  
19

#### Formation of Acrylamide and its Precursors





TELEPHONE (562) 437-4499  
TOLL-FREE (877) TOX-TORT  
TELECOPIER (562) 436-1561  
WWW.TOXICTORTS.COM

LAW OFFICES OF  
**RAPHAEL METZGER**  
A PROFESSIONAL LAW CORPORATION  
401 EAST OCEAN BOULEVARD, SUITE 800  
LONG BEACH, CALIFORNIA 90802-4966

PRACTICE CONCENTRATED IN TOXIC  
TORT & ENVIRONMENTAL LITIGATION  
OCCUPATIONAL & ENVIRONMENTAL LUNG  
DISEASE, CANCER, AND TOXIC INJURIES

1 acrylamide (Dias et al., 2015). While rejecting unripe coffee beans  
2 would yield the greatest reduction of asparagine (and hence of  
3 acrylamide) in coffee, substantial reductions of acrylamide can also  
4 be achieved by optimally processing unripe beans. The pulping of  
5 immature beans contributes to decreased asparagine levels, and  
6 consequently acrylamide levels, for both medium and dark roast  
7 coffee. Whatever the processing type, acrylamide content is lower  
8 after dark roasting when immature beans are processed on the same day  
9 or when the fruits are stored piled in a box for 12 hours. The  
10 reduction of acrylamide from dry pulping unripe beans ranges from 20-  
11 30%. (Dias et al., 2015).

#### Change in roasting process

12  
13  
14  
15 32. The major pathway for the formation of acrylamide  
16 during the roasting of green coffee beans is the Maillard reaction  
17 in which free asparagine and the carbonyl group of reducing sugars  
18 react to form a Schiff base that undergoes decarboxylation.  
19 Acrylamide formation starts at temperatures above 120°C. During the  
20 roasting process, typically in the range of 220-250°C, acrylamide  
21 levels reach peak values within 2-5 min and then decline rapidly to  
22 5-30% of the maximum amount with continued roasting (Lantz et al.,  
23 2006; Kocadagli et al., 2012; Stadler and Theurillat, 2012). This  
24 decline is due to depletion of free asparagine, evaporation, and  
25 covalent binding by Michael addition to compounds in coffee (e.g.,  
26 melanoidins) (Pastoriza et al., 2012). With continued roasting,  
27 acrylamide levels decrease when the rate of disappearance exceeds the  
28 rate of formation (Gökmen and Senyuva, 2006). Because acrylamide is

TELEPHONE (562) 437-4499  
TOLL-FREE (877) TOX-TORT  
TELECOPIER (562) 436-1561  
WWW.TOXICORTS.COM

LAW OFFICES OF  
RAPHAEL METZGER  
A PROFESSIONAL LAW CORPORATION  
401 EAST OCEAN BOULEVARD, SUITE 800  
LONG BEACH, CALIFORNIA 90802-4966

PRACTICE CONCENTRATED IN TOXIC  
TORT & ENVIRONMENTAL LITIGATION  
OCCUPATIONAL & ENVIRONMENTAL LUNG  
DISEASE, CANCER, AND TOXIC INJURIES

1 formed at the beginning of roasting and then decreases, levels of  
2 this compound are higher in lightly roasted coffee than in dark  
3 roasted espresso coffee (Summa et al., 2007; Alves et al., 2010).  
4 Over roasting coffee beans to remove acrylamide can adversely affect  
5 coffee flavor and aroma. Robusta coffee beans contain higher levels  
6 of free asparagine and produce higher levels of acrylamide than  
7 Arabica coffee beans during roasting (Pedreschi et al., 2013).

8 33. Several processing changes have been employed to  
9 reduce acrylamide formation in heat-processed foods. These include  
10 lowering the pH (~4.0) to protonate the  $\alpha$ -NH<sub>2</sub> group of asparagine and  
11 decrease its reactivity with carbonyl compounds, adding amino acids  
12 that compete with asparagine for reaction with reducing sugars, and  
13 optimizing the time and temperature of roasting (Pastoriza et al.,  
14 2012; Pedreschi et al., 2013; Madihah et al., 2013).

15 34. A study investigating different roasting conditions  
16 on the acrylamide content of *Robusta* coffee concluded the optimal  
17 roasting temperature was 203°C, provided low velocity and dry  
18 roasting air were used. "Under these conditions, roasted beans were  
19 characterized by relatively low levels of acrylamide with moderate  
20 degradation of polyphenols and antioxidant properties deterioration,  
21 while showing a pleasant, full flavor." (Budryn et al., 2015).

22 35. A recent study of three traditional coffee roasting  
23 programs that differ in their temperature and times of roasting  
24 produced acrylamide concentrations of 193 ng/g, 136 ng/g, and 117  
25 ng/g -- a 40% difference between the most and least optimal roasting  
26 programs. The lowest acrylamide content was produced by reducing  
27 heating time during the first two stages and increasing heating time  
28 during the later stages of the roasting process. The authors

TELEPHONE (562) 437-4499  
TOLL-FREE (877) TOX-TORT  
TELECOPIER (562) 436-1561  
WWW.TOXICTORTS.COM

LAW OFFICES OF  
**RAPHAEL METZGER**  
A PROFESSIONAL LAW CORPORATION  
401 EAST OCEAN BOULEVARD, SUITE 800  
LONG BEACH, CALIFORNIA 90802-4966

PRACTICE CONCENTRATED IN TOXIC  
TORT & ENVIRONMENTAL LITIGATION  
OCCUPATIONAL & ENVIRONMENTAL LUNG  
DISEASE, CANCER, AND TOXIC INJURIES

1 observed that "it is very important to control the roast time of the  
2 last stage for reducing the acrylamide content in roast coffee" and  
3 concluded that "prolonging the roast time may reduce the content of  
4 acrylamide in roast coffee." (Xu et al., 2016).

#### 5 6 Vacuum roasting

7  
8 36. Vacuum roasting of coffee beans (containing ~7.5%  
9 moisture) for 15-20 minutes at 200°C and 0.15 kPa (~0.02 psi or  
10 0.0015 atm) resulted in 50% less acrylamide than conventional  
11 roasting at atmospheric pressure (Anese et al., 2014). Though there  
12 was concern that the low pressure might remove desirable compounds  
13 in addition to acrylamide, sensory analyses showed that "the medium-  
14 roasted coffee samplers obtained by means of the conventional and  
15 vacuum processes and having different acrylamide levels, were not  
16 perceived as different by the assessors." "The vacuum dark-roasted  
17 coffee was judged to present a slightly but significantly lower odour  
18 intensity" however "no significant differences [in odour intensity]  
19 were found between the dark-roasted coffees subjected to the  
20 conventional and combined conventional-vacuum processes." The  
21 combined process involves conventional roasting followed by vacuum  
22 treatment. Thus, vacuum treatment provides an effective method to  
23 reduce acrylamide in coffee without affecting organoleptic properties  
24 of the roasted product.  
25  
26  
27  
28

### Supercritical CO<sub>2</sub> extraction

1  
2  
3  
4  
5  
6  
7  
8  
9  
10  
11  
12  
13  
14  
15  
16  
17  
18  
19  
20  
21  
22  
23  
24  
25  
26  
27  
28

37. Supercritical CO<sub>2</sub> extraction has been used to remove caffeine from coffee beans. This technique is also effective in reducing levels of acrylamide by approximately 80% from roasted coffee beans (Banchero et al., 2013). Process parameters including temperature, pressure, extraction time, and use of a polar solvent can affect the CO<sub>2</sub> extraction yield as well as the possible loss of any desirable flavor and aroma compounds. The reported optimal conditions for acrylamide extraction were 100°C, 200 bar (2900 psi), and 9.5% ethanol (to increase the polarity of the supercritical fluid). Selective extractions are possible by this method because the solubilities of extracted compounds in supercritical CO<sub>2</sub> vary with pressure. In the Banchero study, green coffee beans were roasted at ~100°C to 150°C to maximize acrylamide formation in the samples and then treated with the supercritical solvent. The lower temperature used in the pre-roasting treatment compared to conventional roasting was considered by the authors to be "quite advantageous from an organoleptic point of view" because "the majority of aroma compounds has still to be formed and cannot be removed by the supercritical treatment. This was confirmed by exploratory degustation tests, which were conducted by coffee testing experts on the coffee brews prepared with some samples of the coffee beans that had been previously subjected to the supercritical treatment." The authors concluded "the supercritical acrylamide-mitigation strategy is expected to only slightly modify the sensory properties of coffee. Future research will be conducted to optimize the supercritical treatment in order to match an efficient removal of acrylamide with the aroma and taste standards suitable to Lavazza coffee consumers."

TELEPHONE (562) 437-4499  
TOLL-FREE (877) TOX-TORT  
TELECOPIER (562) 436-1561  
WWW.TOXICTORTS.COM

LAW OFFICES OF  
**RAPHAEL METZGER**  
A PROFESSIONAL LAW CORPORATION  
401 EAST OCEAN BOULEVARD, SUITE 800  
LONG BEACH, CALIFORNIA 90802-4966

PRACTICE CONCENTRATED IN TOXIC  
TORT & ENVIRONMENTAL LITIGATION  
OCCUPATIONAL & ENVIRONMENTAL LUNG  
DISEASE, CANCER, AND TOXIC INJURIES

1 This "process offers a clean, efficient, and environmentally  
2 acceptable method of removing acrylamide from coffee." (Friedman,  
3 2015).

#### 4 Enzyme treatments

##### 5 *Asparaginase*

6  
7  
8 38. The largest effort to reduce acrylamide formation in  
9 coffee has involved pretreatment of green coffee beans with  
10 asparaginase to decrease the availability of free asparagine for  
11 reaction with reducing sugars during roasting. Several patents on  
12 the use of asparaginase in foods have been filed. A major issue for  
13 the use of asparaginase in unroasted coffee is the delivery of the  
14 enzyme to its substrate asparagine; some recommended options are pre-  
15 drying the beans to facilitate uptake of an aqueous solution, steam  
16 treatment or other wetting process to open pores of the beans and  
17 enable direct contact between the enzyme and its substrate, and  
18 chopping the beans prior to treatment (Guenther et al., 2007; Stadler  
19 and Theurillat, 2012). Stadler (2013) reported that steaming coffee  
20 beans (100°C for 45 min) followed by soaking (50% water, 60°C) with  
21 asparaginase and aspartase reduced acrylamide formation by ~70%  
22 during roasting "with no significant impact on organoleptic  
23 properties."

24 39. Recognizing that acrylamide is a mutagenic,  
25 carcinogenic and neurotoxic chemical, coffee producers have initiated  
26 research and development activities to reduce levels of acrylamide  
27 in roasted coffee beans. Patents have been filed that describe pre-  
28 roasting treatments that enable interaction between asparaginase and

TELEPHONE (562) 437-4499  
TOLL-FREE (877) TOX-TORT  
TELECOPIER (562) 436-1561  
WWW.TOXICTORTS.COM

LAW OFFICES OF  
**RAPHAEL METZGER**  
A PROFESSIONAL LAW CORPORATION  
401 EAST OCEAN BOULEVARD, SUITE 800  
LONG BEACH, CALIFORNIA 90802-4966

PRACTICE CONCENTRATED IN TOXIC  
TORT & ENVIRONMENTAL LITIGATION  
OCCUPATIONAL & ENVIRONMENTAL LUNG  
DISEASE, CANCER, AND TOXIC INJURIES

1 asparagine in coffee beans. The Proctor & Gamble Company obtained  
2 patents for pre-treating coffee beans to reduce levels of free  
3 asparagine in the unroasted beans and levels of acrylamide in roasted  
4 beans by facilitating the degradation of asparagine by asparaginase  
5 (Dria et al., 2004; Dria et al., 2007). The interaction between  
6 asparaginase and asparagine is achieved by drying or steaming green  
7 coffee beans to open pores and then soaking the beans in an aqueous  
8 solution containing asparaginase. Conditions that are optimized  
9 include pH (~7.5-8.5), temperature (38°C), and time for enzymatic  
10 activity (45-60 min). During the incubation with asparaginase,  
11 water-soluble compounds that are extracted from the beans establish  
12 an equilibrium between the beans and the water bath. The conversion  
13 of asparagine to aspartic acid by asparaginase drives additional  
14 asparagine out of the beans. The beans are then dried (to ~ 7%  
15 moisture) before roasting. This pretreatment was reported to result  
16 in green coffee beans that can be labeled "low in asparagine" and  
17 yield a roasted coffee product that can be labeled "acrylamide  
18 reduced by over 90%."

19 40. Illy Caffé obtained a patent in which an aqueous  
20 extract is obtained by heating green coffee beans in water, cooling  
21 the extract, incubating the extract with asparaginase (plus aspartase  
22 to degrade aspartic acid to fumaric acid), concentrating the extract,  
23 drying the extracted green beans, and incubating the concentrated  
24 extract with the dried beans to allow reincorporation of the  
25 constituents that provide the organoleptic properties of  
26 conventionally roasted coffee; the reconstituted beans are then dried  
27 (Navarini et al., 2013). This method resulted in an 80% reduction  
28 in acrylamide levels in the roasted coffee. The authors specify that

TELEPHONE (562) 437-4499  
TOLL-FREE (877) TOX-TORT  
TELECOPIER (562) 436-1561  
WWW.TOXICTORTS.COM

LAW OFFICES OF  
**RAPHAEL METZGER**  
A PROFESSIONAL LAW CORPORATION  
401 EAST OCEAN BOULEVARD, SUITE 800  
LONG BEACH, CALIFORNIA 90802-4966

PRACTICE CONCENTRATED IN TOXIC  
TORT & ENVIRONMENTAL LITIGATION  
OCCUPATIONAL & ENVIRONMENTAL LUNG  
DISEASE, CANCER, AND TOXIC INJURIES

1 "this green coffee can thus be roasted, using known roasting methods,  
2 and the roasted coffee thus obtained has reduced concentration values  
3 of acrylamide." Furthermore, "the method according to the invention  
4 enables a roasted coffee to be obtained that has a reduced acrylamide  
5 content, in which *the desired organoleptic properties remain*  
6 *unaltered* [emphasis added] and can be appreciated by the consumer."

7 41. In 2014 Novozymes submitted data to the US food and  
8 Drug Administration regarding its Acrylaway® products, which it  
9 described as "asparaginase enzyme preparations for food applications  
10 that effectively reduce acrylamide in a broad range of potato-based  
11 foods, cereal-based foods and coffee." Novozymes reported:  
12 "Reduction of acrylamide formation in coffee beans has been confirmed  
13 for Robusta and for Arabica beans at both laboratory/pilot scale and  
14 at industrial scale." Regarding Robusta beans, Novozymes reported:  
15 "At pilot scale level 47% reduction in final acrylamide levels (340  
16 µg/kg to 180 µg/kg) were obtained in asparaginase treated green  
17 Robusta beans. The Robusta beans were initially steamed and  
18 afterwards soaked in a water bath for 2h at 60°C with and without  
19 asparaginase." Regarding Arabica beans, Novozymes reported: "The  
20 results from testing application of asparaginase at various dosages  
21 in green Arabica coffee beans and the effect on asparagine content  
22 and subsequent acrylamide formation after roasting is shown .... A  
23 clear effect of increasing enzyme amount was observed with a dosage  
24 dependent reduction in acrylamide from 785 µg/kg bean to 335-220  
25 µg/kg. Maximum effect was a reduction in acrylamide formation of 72%  
26 at 6000 ASNU/kg beans." ASNU is the amount of asparagine that  
27 produces 1 µmole of ammonia under specific conditions. Regarding  
28 coffee blends, Novozymes reported: "At industrial scale, reduction

TELEPHONE (562) 437-4499  
TOLL-FREE (877) TOX-TORT  
TELECOPIER (562) 436-1561  
WWW.TOXICTORTS.COM

LAW OFFICES OF  
RAPHAEL METZGER  
A PROFESSIONAL LAW CORPORATION  
401 EAST OCEAN BOULEVARD, SUITE 800  
LONG BEACH, CALIFORNIA 90802-4966

PRACTICE CONCENTRATED IN TOXIC  
TORT & ENVIRONMENTAL LITIGATION  
OCCUPATIONAL & ENVIRONMENTAL LUNG  
DISEASE, CANCER, AND TOXIC INJURIES

1 of acrylamide formation has been confirmed in Arabica, Robusta and  
2 blends of Robusta and Arabica. The reductions of acrylamide  
3 formation are: 50% for Arabica-Robusta blend, 43% for Vietnam Robusta  
4 and 63% for Arabica beans, respectively" (Novozymes 2014).  
5

#### 6 *Acrylamidase*

7  
8 42. The recent cloning of a heat stable acrylamidase from  
9 a thermophilic bacterium, *Geobacillus thermoglucosidasius* (Cha and  
10 Chambliss, 2013), provides an approach to enzymatically degrade  
11 acrylamide in roasted coffee. Cha (2013) showed that a cell free  
12 extract of this bacterium is capable of converting acrylamide to  
13 acrylic acid in brewed coffee at 70°C. "Therefore, the enzyme can be  
14 added right after brewing from a coffee maker or it can be applied  
15 to coffee filters because the temperature of coffee right after  
16 brewing is 70-75°C." Cell free extracts of the acrylamide-degrading  
17 strain *Ralstonia eutropha* reduced acrylamide levels in Folgers and  
18 Tasters Choice coffees. Immobilized mycelia from self-cloned  
19 *Aspergillus oryzae*, a filamentous fungus that produces an amidase,  
20 were also effective in degrading acrylamide in brewed coffee (Iwai  
21 et al., 2012). While these enzymatic approaches were shown to be  
22 effective in reducing acrylamide in coffee drinks, the use of  
23 amidases to reduce acrylamide in roasted coffee beans has not yet  
24 been reported. Treatment of brewed coffee with acrylamidase should  
25 not affect the organoleptic properties of the product because the  
26 activity of this enzyme is specific for acrylamide.  
27  
28



### Gamma Radiation

1  
2  
3 44. One study of gamma-irradiation of green coffee beans  
4 observed a decrease in acrylamide levels by approximately 80%,  
5 however, this treatment substantially increased acrylamide levels in  
6 roasted coffee beans (Alkhalifah et al., 2013). The use of gamma-  
7 irradiation to reduce acrylamide in roasted coffee needs to be  
8 further evaluated.

### Extended Storage Time

9  
10  
11 45. Acrylamide levels in roasted ground coffee decrease  
12 during storage depending on time and temperature. The decline in  
13 acrylamide during storage has been attributed largely to covalent  
14 binding to nucleophilic groups (e.g., Michael addition with -SH and  
15 -NH<sub>2</sub> groups of other compounds in coffee).  
16

17 46. *Roasted Coffee.* After 3 months of storage in the dark  
18 at 10-12°C, the acrylamide concentration of vacuum-packed roasted  
19 coffee beans and ground coffee were reduced about 30% (Hoenicke and  
20 Gatermann, 2005); these decreases were suggested to be due to  
21 reaction of acrylamide with sulfhydryl (HS) groups of other coffee  
22 constituents. After 6 months of storage at room temperature in  
23 sealed containers, the acrylamide content of two coffees was reduced  
24 40%, while the acrylamide content of a third coffee was reduced 65%  
25 (Andrzejewski et al., 2004). In another study, after 7 months of  
26 storage in the dark in tightly sealed containers at room temperature,  
27 the acrylamide content of roasted coffee decreased by about 30%  
28 (Delatour et al., 2004). An industry-sponsored study yielded even

TELEPHONE (562) 437-4499  
TOLL-FREE (877) TOX-TORT  
TELECOPIER (562) 436-1561  
WWW.TOXICTORTS.COM

LAW OFFICES OF  
**RAPHAEL METZGER**  
A PROFESSIONAL LAW CORPORATION  
401 EAST OCEAN BOULEVARD, SUITE 800  
LONG BEACH, CALIFORNIA 90802-4966

PRACTICE CONCENTRATED IN TOXIC  
TORT & ENVIRONMENTAL LITIGATION  
OCCUPATIONAL & ENVIRONMENTAL LUNG  
DISEASE, CANCER, AND TOXIC INJURIES

1 greater reductions of acrylamide in vacuum-packed roast and ground  
2 coffee with increasing storage time. When stored at room  
3 temperature, acrylamide levels were reduced about 35% after 2 months,  
4 about 45% after 4 months, about 55% after 6 months, and about 75%  
5 after 12 months (Lantz et al., 2006). When stored at 37°C,  
6 acrylamide levels were reduced about 70% after 2 months, about 85%  
7 after 4 months, and about 90% after 6 months (Lantz et al., 2006).

8 47. *Instant (Soluble) Coffee.* The acrylamide content of  
9 instant coffee was reduced by about 20% when stored at 25°C in the  
10 dark in closed commercial packaging for 6 months and by about 33%  
11 when stored for 12 months (Michalak et al., 2016). Storage at 4°C  
12 resulted in lesser acrylamide reductions: at 6 months acrylamide  
13 content was reduced 12%; at 12 months acrylamide content was reduced  
14 18% (Michalak et al., 2016). At room temperature, the acrylamide  
15 content of soluble coffee powder reduced 67% after 12 months of  
16 storage in the dark (Delatour et al., 2004).

17 48. *Brewed Coffee.* The acrylamide concentration of brewed  
18 coffee decreases with storage time. The acrylamide content of coffee  
19 brewed from roasted coffee stored at room temperature was reduced  
20 about 10-15% after storage for 4 weeks, about 15-20% after storage  
21 for 8 weeks, about 20-25% after storage for 12 weeks, and about 30%  
22 after storage for 16 weeks (Baum et al., 2008). Acrylamide  
23 reductions were greater when roasted coffee was stored at 37°C: about  
24 10-15% after storage for 2 weeks, about 20-30% after storage for 4  
25 weeks, and about 50% after storage for 16 weeks (Baum et al., 2008).

26 49. *Coffee Substitutes.* Storing coffee substitute at 25°C  
27 in the dark in commercial packaging also decreased acrylamide  
28 concentrations over time: at 6 months acrylamide content was reduced

TELEPHONE (562) 437-4499  
TOLL-FREE (877) TOX-TORT  
TELECOPIER (562) 436-1561  
WWW.TOXICTORTS.COM

LAW OFFICES OF  
**RAPHAEL METZGER**  
A PROFESSIONAL LAW CORPORATION  
401 EAST OCEAN BOULEVARD, SUITE 800  
LONG BEACH, CALIFORNIA 90802-4966

PRACTICE CONCENTRATED IN TOXIC  
TORT & ENVIRONMENTAL LITIGATION  
OCCUPATIONAL & ENVIRONMENTAL LUNG  
DISEASE, CANCER, AND TOXIC INJURIES

1  
2  
3  
4  
5  
6  
7  
8  
9  
10  
11  
12  
13  
14  
15  
16  
17  
18  
19  
20  
21  
22  
23  
24  
25  
26  
27  
28

15%; at 12 months acrylamide content was reduced 28% (Michalak et al., 2016). Storage at 4°C resulted in lesser acrylamide reductions: at 6 months acrylamide content was reduced 7%; at 12 months acrylamide content was reduced 12% (Michalak et al., 2016).

50. *Roasted Coffee Shelf Life.* The shelf-life of vacuum-packed roasted coffee beans is about 9 months at room temperature. "Sensory evaluation demonstrated adverse effects on the quality of coffee beverages . . . after 9 months storage of roasted coffee beans." (Kreuml et al., 2013). Arabica beans had a higher intensity of positive sensory attributes compared to Robusta coffee after 9 months of storage. Extending storage time for roasted ground coffee is thus a viable method of reducing acrylamide in coffee. Based on the data provided by Lantz et al. 2006, storing vacuum-packed roasted and ground coffee for 6 months should reduce the acrylamide content of roasted and ground coffee by approximately 55% without compromising sensorial qualities of coffee.

**ADDITION OF FOOD-COMPATIBLE COMPOUNDS**

51. The additives of cysteine to canned coffee drinks heated to 121°C for 6 minutes produced a 95% decrease in acrylamide content (Narita and Inouye, 2014). This decrease was attributed to reaction of the SH groups of cysteine with the double bond of acrylamide to form a biologically inactive compound.

**RESPONSE OF THE POTATO INDUSTRY TO ACRYLAMIDE CONCERNS**

1  
2  
3           52. Acrylamide in coffee is a public health concern  
4 because coffee is the most widely consumed beverage in the world and  
5 is the greatest source of acrylamide intake in the adult population.  
6 However, brewed coffee does not have the highest levels of acrylamide  
7 in food. Although consumed less widely and less frequently, French  
8 fries and potato chips have higher levels of acrylamide than brewed  
9 coffee. Due to the high levels of acrylamide in fried potato  
10 products, the potato industry was the focus of attention when  
11 acrylamide was discovered in food. The first Proposition 65 case  
12 regarding acrylamide in food was filed in 2002 by the Council for  
13 Education and Research on Toxics (CERT); it concerned acrylamide in  
14 French fries sold by fast-food restaurants. Three years later, in  
15 2005, the California Attorney General sued potato chip manufacturers  
16 regarding acrylamide in potato chips. It was not until 2010 that  
17 CERT filed the first Proposition 65 case regarding acrylamide in  
18 ready-to-drink coffee and until 2011 that CERT filed suit against  
19 coffee roasters regarding packaged coffee. Perhaps because the  
20 potato industry was the first industry sued regarding acrylamide in  
21 food, it has taken a more proactive and urgent approach in addressing  
22 the acrylamide problem (Bhaskar et al., 2010) than the coffee  
23 industry has taken to date.

24           53. While the coffee industry has apparently not  
25 implemented any technologies to reduce levels of acrylamide in  
26 coffee, the potato industry has implemented both agronomic and  
27 processing technologies to reduce the acrylamide content of potato  
28 chips and French fries.

TELEPHONE (562) 437-4499  
TOLL-FREE (877) TOX-TORT  
TELECOPIER (562) 436-1561  
WWW.TOXICTORTS.COM

LAW OFFICES OF  
**RAPHAEL METZGER**  
A PROFESSIONAL LAW CORPORATION  
401 EAST OCEAN BOULEVARD, SUITE 800  
LONG BEACH, CALIFORNIA 90802-4966

PRACTICE CONCENTRATED IN TOXIC  
TORT & ENVIRONMENTAL LITIGATION  
OCCUPATIONAL & ENVIRONMENTAL LUNG  
DISEASE, CANCER, AND TOXIC INJURIES

1                   54. Potato chip manufacturers were the first companies to  
2 use the enzyme, *asparaginase*, as an acrylamide mitigation technique.  
3 Potato chip manufacturers used this enzyme in potato dough to reduce  
4 the formation of acrylamide in potato chips. I am personally  
5 familiar with the resolution of the Attorney General's action against  
6 the potato chip manufacturers, because I served as an expert for the  
7 California Attorney General in that lawsuit. The case resolved when  
8 the potato chip companies agreed to reduce the acrylamide content in  
9 potato chips (through use of enzymes or other process methods), in  
10 lieu of giving cancer hazard warnings.

11                   55. Potato producers also made substantial efforts to grow  
12 potatoes with lower levels of acrylamide precursors. Varieties of  
13 potatoes that have low acrylamide precursor levels have been  
14 cultivated and introduced into agriculture (Zhu et al., 2010; Halford  
15 et al., 2012a, 2012b; Novy et al., 2013; Bethke et al., 2015; Brandt  
16 et al., 2015; Thompson et al., 2015; Wang et al., 2015).

17                   56. The measures undertaken by the potato industry to  
18 reduce levels of acrylamide in fried potato products in the decade  
19 since the discovery of acrylamide in food have proven successful.  
20 A study of acrylamide levels in 40,455 samples of potato crisps  
21 (French fries) from 20 European countries for the years 2002 to 2011  
22 - the largest dataset ever compiled relating to acrylamide levels in  
23 potato crisps - showed that the proportion of samples containing  
24 acrylamide at a level above the indicative value of 1000 ng/g<sup>-1</sup> (1000  
25 µg/kg) established by the European Commission in 2011 fell from 23.8%  
26 in 2002 to 3.2% in 2011. (Powers et al., 2013).

27                   57. RNA interference constructs have been developed to  
28 produce plants in which genes that code for enzymes that synthesize

TELEPHONE (562) 437-4499  
TOLL-FREE (877) TOX-TORT  
TELECOPIER (562) 436-1561  
WWW.TOXICTORTS.COM

LAW OFFICES OF  
**RAPHAEL METZGER**  
A PROFESSIONAL LAW CORPORATION  
401 EAST OCEAN BOULEVARD, SUITE 800  
LONG BEACH, CALIFORNIA 90802-4966

PRACTICE CONCENTRATED IN TOXIC  
TORT & ENVIRONMENTAL LITIGATION  
OCCUPATIONAL & ENVIRONMENTAL LUNG  
DISEASE, CANCER, AND TOXIC INJURIES

1 acrylamide precursor compounds have been silenced (i.e., down-  
2 regulated or reduced expression), but retain normal growth  
3 characteristics (Rommens et al., 2008; Bhaskar et al., 2010; Zhu, et  
4 al. 2016). Small RNA molecules produced from these constructs bind  
5 to the specific messenger RNA (mRNA) that is synthesized from the  
6 targeted gene preventing the mRNA from producing the protein product  
7 (i.e., the enzyme). Specificity for a particular gene is maintained  
8 because the construct is produced from the mRNA that is synthesized  
9 from that gene, and silencing typically occurs due to cleavage of the  
10 double-stranded RNA molecule by cellular enzymes.

11 58. Rommens et al. (2009) described methods to down  
12 regulate or silence genes involved in the synthesis of aspartate (for  
13 example, aspartate transaminase - see Figure above title {Formation  
14 of Acrylamide and its Precursors"}) or overexpress genes that  
15 metabolize aspartate by inserting specific gene coding polynucleotide  
16 sequences into the host plant. Asparagine is produced from  
17 aspartate. Lowering this asparagine precursor by altering the  
18 expression of genes that control aspartate metabolism was reported  
19 to reduce levels of acrylamide by about 66% in ground, roasted  
20 coffee.

21 59. An alternative method described by Rommens (2012)  
22 involves isolating a gene from a selected plant that regulates the  
23 expression of a particular trait, modifying that gene (e.g., via  
24 mutation, deletion, alteration of its expression), and then  
25 reinserting the modified gene back into the genome of the plant. The  
26 advantage of this approach is that no foreign DNA is integrated into  
27 the genome of the host plant. Silencing genes associated with  
28 negative traits or overexpressing genes that prevent negative traits

TELEPHONE (562) 437-4499  
TOLL-FREE (877) TOX-TORT  
TELECOPIER (562) 436-1561  
WWW.TOXICTORTS.COM

LAW OFFICES OF  
**RAPHAEL METZGER**  
A PROFESSIONAL LAW CORPORATION  
401 EAST OCEAN BOULEVARD, SUITE 800  
LONG BEACH, CALIFORNIA 90802-4966

PRACTICE CONCENTRATED IN TOXIC  
TORT & ENVIRONMENTAL LITIGATION  
OCCUPATIONAL & ENVIRONMENTAL LUNG  
DISEASE, CANCER, AND TOXIC INJURIES

1 can produce plants with desired characteristics. For example,  
2 overexpressing the invertase inhibitor gene (the gene product  
3 inhibits the hydrolysis of sucrose to glucose and fructose) in  
4 potatoes led to a 5-10 fold reduction in acrylamide levels in  
5 processed fries. Silencing of two tuber-specific asparagine  
6 synthetase genes reduced levels of free asparagine by nearly 20-fold  
7 and lowered the accumulation of acrylamide in French fries and potato  
8 chips by 90-95%. (Rommens et al., 2008; Chawla et al., 2012).  
9 Sensory evaluations by professionally trained experts demonstrated  
10 that "heat-processed products derived from low asparagine tubers were  
11 also indistinguishable from their untransformed counterparts." A  
12 similar approach could likely be applied to coffee plants to lower  
13 acrylamide formation in roasted coffee.

14 60. Suppression or silencing of other genes that code for  
15 enzymes that produce acrylamide synthetase has also been utilized to  
16 achieve impressive reductions in concentrations of acrylamide  
17 precursors in potato plants, with comparable reductions in acrylamide  
18 content of French fries. Suppression of the expression of the  
19 vacuolar invertase gene in potato plants not only prevents cold-  
20 induced sweetening in potatoes (Bhaskar et al., 2010), but has been  
21 shown to significantly reduce the acrylamide content by 89-90% in  
22 fried potato products (Bhaskar et al., 2010; Zhu et al., 2014; 2016)  
23 and improve French fry and potato chip quality. (Zhu et al., 2014;  
24 Rasmussen et al., 2015). Cold-induced sweetening is caused by the  
25 hydrolysis of sucrose to glucose and fructose by vacuolar invertase  
26 in potatoes that are stored at cold temperatures to prevent  
27 sprouting. Silencing of the asparagine synthetase-1 gene has been  
28 shown to reduce the acrylamide-forming potential of potatoes grown

TELEPHONE (562) 437-4499  
TOLL-FREE (877) TOX-TORT  
TELECOPIER (562) 436-1561  
WWW.TOXICTORTS.COM

LAW OFFICES OF  
**RAPHAEL METZGER**  
A PROFESSIONAL LAW CORPORATION  
401 EAST OCEAN BOULEVARD, SUITE 800  
LONG BEACH, CALIFORNIA 90802-4966

PRACTICE CONCENTRATED IN TOXIC  
TORT & ENVIRONMENTAL LITIGATION  
OCCUPATIONAL & ENVIRONMENTAL LUNG  
DISEASE, CANCER, AND TOXIC INJURIES

1 in the field without affecting tuber shape and yield (Chawla et al.,  
2 2012).

3 61. Clasen et al. (2016) recently reported the creation  
4 of Ranger Russet potato plants with undetectable levels of reducing  
5 sugars in tubers due to the induction of inactivating mutations in  
6 the vacuolar invertase gene. Acrylamide levels were reduced by 73%  
7 in potato chips prepared from tubers of the mutant plants. The  
8 method of targeted gene mutation involved the design and use of  
9 sequence specific endonucleases that cut DNA in the protein coding  
10 region of the vacuolar invertase gene.

11 62. The J.R. Simplot Company has developed a potato using  
12 native-gene modification, that decreases the formation of asparagine  
13 and greatly reduces acrylamide in French fries (Simplot 2007). The  
14 company's Innate potato was approved for use in agriculture by the  
15 Department of Agriculture (USDA 2014; 2015) and the US Food and Drug  
16 Administration recently completed its food and feed safety  
17 assessment, concluding that Simplot's Russet Burbank Generation 2  
18 potatoes are not materially different in composition, safety,  
19 nutrition, and other relevant parameters, from any other potato or  
20 potato-derived food or feed currently on the market. When the  
21 company receives approval from the EPA, it will begin selling Innate  
22 potatoes in the marketplace. A study has shown consumer willingness  
23 to pay more for low-acrylamide potato products (Lacy et al., 2016).

24 63. Thus, the potato industry has been successful in  
25 developing potatoes with low levels of acrylamide precursors and has  
26 made significant processing changes to reduce acrylamide in fried  
27 potato products. Further reductions of acrylamide in fried potatoes  
28 are expected as new potato varieties are increasingly used.

TELEPHONE (562) 437-4499  
TOLL-FREE (877) TOX-TORT  
TELECOPIER (562) 436-1561  
WWW.TOXICTORTS.COM

LAW OFFICES OF  
**RAPHAEL METZGER**  
A PROFESSIONAL LAW CORPORATION  
401 EAST OCEAN BOULEVARD, SUITE 800  
LONG BEACH, CALIFORNIA 90802-4966

PRACTICE CONCENTRATED IN TOXIC  
TORT & ENVIRONMENTAL LITIGATION  
OCCUPATIONAL & ENVIRONMENTAL LUNG  
DISEASE, CANCER, AND TOXIC INJURIES

## CONCLUSION

1  
2  
3  
4  
5  
6  
7  
8  
9  
10  
11  
12  
13  
14  
15  
16  
17  
18  
19  
20  
21  
22  
23  
24  
25  
26  
27  
28

64. Numerous publications in the scientific literature and company patents provide descriptions of methods that substantially reduce acrylamide levels in roasted coffee beans; in most cases there was no effect or a minimal change in the organoleptic properties of this popular beverage. The simplest and most economical means of reducing acrylamide in coffee are using arabica beans exclusively, using only ripe beans, increasing roasting degree for light and medium roasts, vacuum roasting, and increasing storage time. The two most effective "high-tech" approaches are pre-treatment with asparaginase (plus aspartase) and supercritical carbon dioxide extraction. Pre-treatment with asparaginase (plus aspartase) is effective in decreasing the concentration of the acrylamide precursor asparagine in green coffee beans without affecting organoleptic properties of roasted coffee. Supercritical CO<sub>2</sub> extraction is effective in removing acrylamide from roasted coffee beans. Though potential effects of the latter process on organoleptic properties of roasted coffee have not been fully evaluated, it is expected that optimizing process parameters would minimize any loss of desirable flavor and aroma compounds. The most effective approach to reduce acrylamide levels in coffee could be a combination of independent mitigation treatments such as asparaginase pre-treatment and vacuum roasting followed by supercritical CO<sub>2</sub> extraction of roasted beans and extended storage time. While this combination approach could be implemented in the near future, a long-term approach to reduce acrylamide formation in roasted coffee beans would use advances in alterations of plant genetic properties, specifically the expression of genes that control the synthesis or degradation of acrylamide

precursors.

65. The potato industry has used methodologies of modern biomolecular technology to lower acrylamide precursors in plants and produce fried potato products with substantially reduced levels of acrylamide; the advances made by the potato industry could serve as a model for the coffee industry to produce roasted coffee that also has markedly reduced levels of acrylamide.

I declare under penalty of perjury under the laws of the State of California that the foregoing is true and correct.

Executed on May 12, 2016, at  
North Logan, Utah.

*Ronald L. Melnick*  
Ronald L. Melnick, Ph.D.



TELEPHONE (562) 437-4499  
TOLL-FREE (877) TOX-TORT  
TELECOPIER (562) 436-1561  
WWW.TOXICTORTS.COM

LAW OFFICES OF  
**RAPHAEL METZGER**  
A PROFESSIONAL LAW CORPORATION  
401 EAST OCEAN BOULEVARD, SUITE 800  
LONG BEACH, CALIFORNIA 90802-4966

PRACTICE CONCENTRATED IN TOXIC  
TORT & ENVIRONMENTAL LITIGATION  
OCCUPATIONAL & ENVIRONMENTAL LUNG  
DISEASE, CANCER, AND TOXIC INJURIES

- 1 Budryn G, Nebesny E, Oracz J. (2015). Correlation Between the  
2 Stability of Chlorogenic Acids, Antioxidant Activity and  
3 Acrylamide Content in Coffee Beans Roasted in Different  
4 Conditions. *Int J Food Properties* 18:290-302
- 5 Cha M. (2013). Enzymatic control of the acrylamide level in  
6 coffee. *Eur Food Res Technol* 236:567-571.
- 7 Cha M, Chambliss GH. (2013). Cloning and sequence analysis of the  
8 heat-stable acrylamidase from a newly isolated thermophilic  
9 bacterium, *Geobacillus thermoglucosidasius* AUT-01.  
10 *Biodegradation*. 24:57-67.
- 11 Chawla R, Shakya R, Rommens CM. (2012). Tuber-specific silencing  
12 of asparagine synthetase-1 reduces the acrylamide-forming  
13 potential of potatoes grown in the field without affecting  
14 tuber shape and yield. *Plant Biotechnol J*. 10:913-924.
- 15 Claeys W, De Meulenaer B, Huygh, Scippo ML, Hoet P, Matthys C.  
16 (2016). Reassessment of the Acrylamide Risk: Belgium as a  
17 Case-Study. *Food Control* 59:628-635.
- 18 Clasen BM, Sgtoddard TJ, Luo S, Demorest ZL, et al. (2016).  
19 Improving cold storage and processing traits in potato through  
20 targeted gene knockout. *Plant Biotechnol J*. 14:1169-176.
- 21 Delatour T, Perisset A, Goldmann T, Riediker S, Stadler R. (2004).  
22 Improved sample preparation to determine acrylamide in  
23 difficult matrixes such as chocolate powder, cocoa, and coffee  
24 by liquid chromatography tandem mass spectroscopy. *J Agric Food  
25 Chem*. 52:4625-4631.
- 26 Dias EC, Borém FM, Pereira RGFA, Soares C, Fernandes JO. (2015).  
27 Influence of unripe coffee fruit processing on acrylamide  
28 formation after roasting. Chap. 3: Acrylamide in Coffee:  
Influence of Coffee Variety and Processing, pp. 127-142, in  
Soares CMD, Assessment of the dietary intake of acrylamide in  
Portugal. Development and evaluation of strategies for  
reduction of acrylamide formation in thermally processed foods.  
Doctoral Dissertation, Universidad de Porto (February 2015).
- Dria GJ, Zyzak DV, Gutwein RW, Villagran FV, et al. (2004). Method  
for reduction of acrylamide in roasted coffee beans, roasted  
coffee beans roasted coffee beans having reduced levels of  
acrylamide, and article of commerce. US 2004/0081724 A1. The  
Proctor & Gamble Company.
- Dria GJ, Zyzak DV, Gutwein RW, Villagran FV, et al. (2007). Method  
for reduction of acrylamide in roasted coffee beans, roasted  
coffee beans roasted coffee beans having reduced levels of  
acrylamide, and article of commerce. US 7,220,440 B2. The  
Proctor & Gamble Company.
- European Food Safety Authority. (2012). Update on Acrylamide  
Levels in Food from Monitoring Years 2007 to 2010. *EFSA J*.

TELEPHONE (562) 437-4499  
TOLL-FREE (877) TOX-TORT  
TELECOPIER (562) 436-1561  
WWW.TOXICTORTS.COM

LAW OFFICES OF  
RAPHAEL METZGER  
A PROFESSIONAL LAW CORPORATION  
401 EAST OCEAN BOULEVARD, SUITE 800  
LONG BEACH, CALIFORNIA 90802-4966

PRACTICE CONCENTRATED IN TOXIC  
TORT & ENVIRONMENTAL LITIGATION  
OCCUPATIONAL & ENVIRONMENTAL LUNG  
DISEASE, CANCER, AND TOXIC INJURIES

1 10(10):38, 2928, available online at  
2 <http://www.efsa.europa.eu/en/efsajournal/doc/2938.pdf>.

3 Freedman M. (2015). Acrylamide: inhibition of formation in  
4 processed food and mitigation of toxicity in cells, animals,  
5 and humans. *Food Funct.* 6(6):1752-72.

6 Friedman M, Levin CE. (2008). Review of methods for the reduction  
7 of dietary content and toxicity of acrylamide. *J Agric Food*  
8 *Chem.* 56:6113-6140.

9 Gökmen V and Senyuva HZ. (2006). Study of colour and acrylamide  
10 formation in coffee, wheat flour and potato chips during  
11 heating. *Food Chem* 99:238-243.

12 Guenther H, Anklam E, Wenzl T, and Stadler RH. (2007). Acrylamide  
13 in coffee: Review of progress in analysis, formation, and level  
14 reduction. *Food Add Contam, Suppl 1*: 60-70.

15 Halford NG, Curtis TY, Muttucumaru N, Postles J, Elmore JS,  
16 Mottram DS. (2012a). The acrylamide problem: a plant and  
17 agronomic science issue. *J Exp Bot.* 63(8):2841-51.

18 Halford NG, Muttucumaru N, Powers SJ, Gillatt PN, Hartley L,  
19 Elmore JS, Mottram DS. (2012b). Concentrations of free amino  
20 acids and sugars in nine potato varieties: effects of storage  
21 and relationship with acrylamide formation. *J Agric Food Chem.*  
22 60(48):12044-55.

23 Hoenicke K, Gatermann R. (2005). Studies on the stability of  
24 acrylamide in food during storage. *J AOAC Int* 88(1):268-273.

25 Iwai K, Fukunaga T, Narita Y, Nakagiri O, et al. (2012).  
26 Development of acrylamide-free "ready-to-drink" coffee by  
27 *Aspergillus oryzae*. 24<sup>th</sup> International Conference on Coffee  
28 Science - Coffee consumption and human physiology. Association  
for Science and Information on Coffee 64-71.

Kocadağlı T, Göncüoğlu N, Hamzalıoğlu A, Gökmen V. (2012). In  
depth study of acrylamide formation in coffee during roasting:  
role of sucrose decomposition and lipid oxidation. *Food Funct*  
3: 970-975.

Kreuml MTL, Majchrzak D, Ploederl B, Koenig J. (2013). Changes in  
Sensory quality characteristics of coffee during storage. *Food*  
*Sci. Nutr.* 1(4):267-272.

Lacy K, Huffman WE. (2016) Consumer Demand for Potato Products and  
Willingness-to-Pay for Low-Acrylamide, Sulfite-Free Fresh  
Potatoes and Dices: Evidence from Lab Auctions. *J Agric*  
*Resource Econ.* 41(1):116-37.

Lantz I, Ternité R, Wilkens J, Hoenicke K, Guenther H, van der  
Stegen GH. (2006). Studies on acrylamide levels in roasting,  
storage and brewing of coffee. *Mol Nutr Food Res.* 50:1039-1046.

TELEPHONE (562) 437-4499  
TOLL-FREE (877) TOX-TORT  
TELECOPIER (562) 436-1561  
WWW.TOXICTORTS.COM

LAW OFFICES OF  
**RAPHAEL METZGER**  
A PROFESSIONAL LAW CORPORATION  
401 EAST OCEAN BOULEVARD, SUITE 800  
LONG BEACH, CALIFORNIA 90802-4966

PRACTICE CONCENTRATED IN TOXIC  
TORT & ENVIRONMENTAL LITIGATION  
OCCUPATIONAL & ENVIRONMENTAL LUNG  
DISEASE, CANCER, AND TOXIC INJURIES

- 1 Mazzafera P. (1998). Chemical Composition of Defective Coffee  
2 Beans. Food Chem. 64(4):547-554. Michalak J, Gujska E,  
3 Czarnowska M, Klepacka J, Nowak F. (2016). Effect of Storage on  
4 Acrylamide and 5-hydroxymethylfurfural Contents in Selected  
5 Processed Plant Products with Long Shelf-Life *Plant Foods Hum*  
6 *Nutr.* [Epub ahead of print]
- 7 Mucci LA, Adami HO, Wolk A. (2006). Prospective study of dietary  
8 acrylamide and risk of colorectal cancer among women. *Int J*  
9 *Cancer.* 118:169-173.
- 10 Narita Y and Inouye K. (2014). Decrease in the acrylamide content  
11 in canned coffee by heat treatment with the addition of  
12 cysteine. *J Agric Food Chem.* 62:12218-12222.
- 13 Navarini L, Del Terra L, Colomban S, Lonzarich V, Suggi Liverani  
14 F. (2013). Method for reducing the content of acrylamide in a  
15 roasted coffee. WO 2013/005145 A1. Illy Café' SPA.
- 16 Novy, R. Collaboration Among Industry and Researchers in the  
17 Identification of Low Acrylamide, Fry Processing Potato  
18 Varieties 2013 UWEX WPVGA Poster Abstract No. 20.
- 19 Novozymes, Letter to Food and Drug Administration re: Draft  
20 Guidance ofr Inudstry on Acrylamide in Foods; Docket ID FDA-  
21 2013-D-0715, with Additional Information on acrylamide  
22 mitigation using asparaginase (January 14, 2014).
- 23 Pastoriza S, Rufián-Henares JA and Morales FJ. (2012).  
24 Reactivity of acrylamide with coffee melanoidins in model  
25 systems. *Food Sci Tech* 45:198-203.
- 26 Pedreschi F, Mariotti MS, Granby K (2014). Current issues in  
27 dietary acrylamide: formation, mitigation and risk assessment.  
28 *J Sci Food Agric.* 94:9-20.
- Porto, ACV, Freitas-Silva O, da Rosa JS, Gottschalk LMF (2015).  
Estimated Acrylamide Intake from Coffee Consumption in Latin  
America. *Am J Agric Biol Sci* 10(2):91-98.
- Powers SJ, Mottram DS, Curtis A, Halford NG. (2013). Acrylamide  
concentrations in potato crisps in Europe from 2002 to 2011.  
*Food Addit Contam. Part A Chem Anal Control Expo Risk Assess.*  
30(9):1493-500.
- Privat I, McCarthy, JG, Petlard, V, Tanksley SD, Lin C. (2010).  
Nucleic acids and proteins associated with sucrose degradation  
in coffee. US 2010/01154074 A1. NESTEC S.A.
- Rasmussen JR. (2015). Invertase Silencing Improves Fry and Chip  
Quality. 99<sup>th</sup> Annual Meeting of the Potato Association of  
America Abstract No. G41.

TELEPHONE (562) 437-4499  
TOLL-FREE (877) TOX-TORT  
TELECOPIER (562) 436-1561  
WWW.TOXICTORTS.COM

LAW OFFICES OF  
**RAPHAEL METZGER**  
A PROFESSIONAL LAW CORPORATION  
401 EAST OCEAN BOULEVARD, SUITE 800  
LONG BEACH, CALIFORNIA 90802-4966

PRACTICE CONCENTRATED IN TOXIC  
TORT & ENVIRONMENTAL LITIGATION  
OCCUPATIONAL & ENVIRONMENTAL LUNG  
DISEASE, CANCER, AND TOXIC INJURIES

- 1 Rommens CM, Yan H, Swords K, Richael C, Ye J. (2008). Low-  
2 acrylamide French fries and potato chips. *Plant Biotech J.*  
3 6:843-853.
- 4 Rommens C, Yan, H, Jingsong Y. (2009). Reduced acrylamide plants  
5 and foods. US 2009/01123626 A1. JR Simplot Company.
- 6 Rommens C. (2012). Precise breeding - low acrylamide foods. US  
7 8,252,974 B2. JR Simplot Company.
- 8 Seal CJ, de Mul A, Eisenbrand G, Haverkort AJ, et al. (2008).  
9 Risk-benefit considerations of mitigation measures on  
10 acrylamide content of foods - a case study on potatoes, cereals  
11 and coffee. *Br J Nutrition* 99 Suppl 2: S1-S46.
- 12 Simplot. (2007). Intragenic Fry Potato: Deliver Superior  
13 Quality, Reduced Cold-Sweetening, Low Bruise, Low Acrylamide  
14 Fries [Powerpoint].
- 15 Stadler R. (2013). Food process contaminants: Industry  
16 perspectives and update on mitigation. *Euro Food Chem XVII,*  
17 *Istanbul, Turkey.*
- 18 Stadler R and Theurillat V. (2012). Acrylamide in coffee. In  
19 *Coffee: Emerging Health Effects and Disease Prevention* (Chu Y-  
20 F, editor). John Wiley & Sons, Inc. pp 259-273.
- 21 Summa CA, de la Calle B, Brohee M, Stadler RH, Anklam E. (2007).  
22 Impact of the roasting degree of coffee on the in vitro radical  
23 scavenging capacity and content of acrylamide. *LWT Food Sci*  
24 *Technol* 40:1849-1854.
- 25 Thompson AL, \*\*\*\*\* (2015). Successes in Traditional Breeding  
26 Program. 99<sup>th</sup> Annual Meeting of the Potato Association of  
27 America Abstract S03.
- 28 U.S. Department of Agriculture. (2014). J.R. Simplot Co.;  
Availability of Plant Pest Risk Assessment and Environmental  
Assessment for Determination of Nonregulated Status of Potato  
Genetically Engineered for Low Acrylamide Potential and Reduced  
Black Spot Bruise. *Fed. Reg.* 79(104): 31080-2.
- U.S. Department of Agriculture. (2015). J.R. Simplot Co.;  
Determination of Nonregulated Status of Potato Genetically  
Engineered for Late Blight Resistance, Low Acrylamide  
Potential, Reduced Black Spot Bruising, and Lowered Reducing  
Sugars. *Fed. Reg.* 80(170):53101-2.
- Wang Y. (2015). A National Effort to Identify Fry Processing  
Clones with Low Acrylamide-Forming Potential. 99<sup>th</sup> Annual  
Meeting of the Potato Association of America Abstract No. G61.
- Xu T, Yang C, Zeng S, Wang M. (2016). Content and Formation of  
Acrylamide in Traditional Coffee Roast Programmes 2nd  
*International Conference on Machinery, Materials Engineering,*

TELEPHONE (562) 437-4499  
TOLL-FREE (877) TOX-TORT  
TELECOPIER (562) 436-1561  
WWW.TOXICTORTS.COM

LAW OFFICES OF  
**RAPHAEL METZGER**  
A PROFESSIONAL LAW CORPORATION  
401 EAST OCEAN BOULEVARD, SUITE 800  
LONG BEACH, CALIFORNIA 90802-4966

PRACTICE CONCENTRATED IN TOXIC  
TORT & ENVIRONMENTAL LITIGATION  
OCCUPATIONAL & ENVIRONMENTAL LUNG  
DISEASE, CANCER, AND TOXIC INJURIES

1  
2  
3  
4  
5  
6  
7  
8  
9  
10  
11  
12  
13  
14  
15  
16  
17  
18  
19  
20  
21  
22  
23  
24  
25  
26  
27  
28

*Chemical Engineering and Biotechnology (MMECEB 2015)*, pp. 884-887.

Zeracryl AS. (2013). Report of trial at two French fries manufacturers and one restaurant. Letter from K.O Tvedt to US Food and Drug Administration.

Zhu F, Cai YZ, Ke J, Corke H. (2010). Compositions of phenolic compounds, amino acids and reducing sugars in commercial potato varieties and their effects on acrylamide formation. *J Sci Food Agric.* 90(13):2254-2262.

Zhu X, Richael C, Chamberlain P, Busse JS, Bussan AJ, Jiang J, Bethke PC. (2014). Vacuolar invertase gene silencing in potato (*Solanum tuberosum* L.) Improves processing quality by decreasing the frequency of sugar-end defects. *PLoS One* 9(4):e93381.

Zhu X, Gong H, He Q, Zeng Z, Busse JS, Jin W, Bethke PC, Jiang J. (2016). Silencing of vacuolar invertase and asparagine synthetase genes and its impact on acrylamide formation of fried potato products. *Plant Biotechnol J.* 14:709-718.

TELEPHONE (562) 437-4499  
TOLL-FREE (877) TOX-TORT  
TELECOPIER (562) 436-1561  
WWW.TOXICTORTS.COM

LAW OFFICES OF  
**RAPHAEL METZGER**  
A PROFESSIONAL LAW CORPORATION  
401 EAST OCEAN BOULEVARD, SUITE 800  
LONG BEACH, CALIFORNIA 90802-4966

PRACTICE CONCENTRATED IN TOXIC  
TORT & ENVIRONMENTAL LITIGATION  
OCCUPATIONAL & ENVIRONMENTAL LUNG  
DISEASE, CANCER, AND TOXIC INJURIES

**ELECTRONIC PROOF OF SERVICE**

STATE OF CALIFORNIA, COUNTY OF LOS ANGELES)

I am employed in the County of Los Angeles, State of California. I am over the age of 18 years and am not a party to the within action. My business address is 401 E. Ocean Blvd., 8<sup>th</sup> Floor, Long Beach, CA 90802.

On May 16, 2016, I served the foregoing document, described as: **DECLARATION OF DR. RONALD L. MELNICK IN SUPPORT OF PLAINTIFF'S MOTION FOR SUMMARY ADJUDICATION OF DEFENDANTS' ALTERNATIVE SIGNIFICANT RISK LEVEL ("ASRL") DEFENSE** on the interested parties to this action by submitting an electronic version of the document via FTP upload to LexisNexis/FileAndServe - File & ServeXpress pursuant to the Court's Order.

I declare under penalty of perjury under the laws of the State of California that the above is true and correct.

Executed on May 16, 2016, at Long Beach, California.



Nina S. Vidal, Declarant

TELEPHONE (562) 437-4499  
TOLL-FREE (877) TOX-TORT  
TELECOPIER (562) 436-1561  
WWW.TOXICTORTS.COM

LAW OFFICES OF  
**RAPHAEL METZGER**  
A PROFESSIONAL LAW CORPORATION  
401 EAST OCEAN BOULEVARD, SUITE 800  
LONG BEACH, CALIFORNIA 90802-4966

PRACTICE CONCENTRATED IN TOXIC  
TORT & ENVIRONMENTAL LITIGATION  
OCCUPATIONAL & ENVIRONMENTAL LUNG  
DISEASE, CANCER, AND TOXIC INJURIES

1  
2  
3  
4  
5  
6  
7  
8  
9  
10  
11  
12  
13  
14  
15  
16  
17  
18  
19  
20  
21  
22  
23  
24  
25  
26  
27  
28

SERVICE LIST  
(CERT vs. Starbucks, Case No. BC435759)

-o0o-

Trenton H. Norris, Esq.  
Rachel L. Chanin, Esq.  
Arnold & Porter  
Three Embarcadero Center, 7<sup>th</sup> Floor  
San Francisco, CA 94111  
(7-Eleven, Inc., BP West Coast Products  
LLC, Winchell's Franchising, LLC, Yum Yum  
Donut Shops, Inc., Albertson's, LLC)  
*Settlement pending with Winchell's*

Michele B. Corash, Esq.  
Robin S. Stafford, Esq.  
Morrison & Foerster  
425 Market Street  
San Francisco, CA 94105-2482  
(Starbucks Corporation, Starbucks Holding  
Company, Seattle Coffee Company, Peet's  
Operating Company, Inc. (incorrectly sued  
herein as Peet's Coffee and Tea, Inc.);  
International Coffee & Tea, LLC)

(Updated 04/26/16 nsv)

TELEPHONE (562) 437-4499  
TOLL-FREE (877) TOX-TORT  
TELECOPIER (562) 436-1566  
WWW.TOXICTORTS.COM

LAW OFFICES OF  
**RAPHAEL METZGER**  
A PROFESSIONAL LAW CORPORATION  
401 EAST OCEAN BOULEVARD, SUITE 800  
LONG BEACH, CALIFORNIA 90802-4966

PRACTICE CONCENTRATED IN TOXIC  
TORT & ENVIRONMENTAL LITIGATION  
OCCUPATIONAL & ENVIRONMENTAL LUNG  
DISEASE, CANCER, AND TOXIC INJURIES

1  
2  
3  
4  
5  
6  
7  
8  
9  
10  
11  
12  
13  
14  
15  
16  
17  
18  
19  
20  
21  
22  
23  
24  
25  
26  
27  
28

SERVICE LIST  
(CERT v. Brad Berry, Case No. BC435759)

-o0o-

<p>Gregory P. O'Hara, Esq. Rhys W. Cheung, Esq. Nixon Peabody 2 Palo Alto Square 3000 El Camino Real, Suite 500 Palo Alto, CA 94306 (The Kroger Co., Ralphs Grocery Company)</p> <p>Jeffrey B. Margulies, Esq. Fulbright &amp; Jaworski 555 S. Flower St., 41<sup>st</sup> Floor Los Angeles, CA 90071 (Target Corporation, Safeway, Inc. Sprouts Farmers Markets, LLC; Reily Foods Company; H.N. Fernandez, Inc.)</p> <p>Renee D. Wasserman, Esq. Alecia E. Cotton, Esq. Rogers Joseph O'Donnell 311 California Street San Francisco, CA 94104 (Bristol Farms, Costco Wholesale, Inc.)</p> <p>Jeffrey B. Margulies, Esq. Jade Jurdi, Esq. Norton Rose Fulbright US LLP 555 So. Flower St., 41<sup>st</sup> Fl. Los Angeles, CA 90071 (Albertson's LLC)</p> <p>Daniel J. Faria, Esq. Kate Ides, Esq. O'Melveny &amp; Myers 400 S. Hope Street Los Angeles, CA 90071-2899 (Trader Joe's Company; Mountanos Brothers Coffee Company)</p> <p>Michael D. Abraham, Esq. Robert H. Bunzel, Esq. Kerry L. Duffy, Esq. Bartko, Zankel, Bunzel, &amp; Miller 900 Front St., Suite 300 San Francisco, CA 94111 (Wal-Mart Stores, Inc. and Sam's West, Inc.)</p>	<p>Michele B. Corash, Esq. Robert Falk, Esq. Robin Stafford, Esq. Travis Brandon, Esq. Morrison &amp; Foerster 425 Market Street San Francisco, CA 94105-2482 (Brad Barry Company, Ltd., Caribou Coffee Company, Inc., F. Gavina &amp; Sons, Inc., Green Mountain Coffee Roasters, Inc., Illy Caffe North America, Inc., International Coffee &amp; Tea, Llc, the J.M. Smucker Company, Kraft Foods Inc., Massimo Zanetti Beverage USA, Inc., Melitta U.S.A., Inc., Nestle USA, Inc., Peet's Coffee &amp; Tea, Inc., Rowland Coffee Roasters, Inc., Sara Lee Corporation, Seattle's Best Coffee Llc, Smucker Foodservice, Inc., Starbucks Corporation, TC Global, Inc., Vilore Foods Company, Inc., DD IP Holder Llc, The Folgers Coffee Company, Godiva Chocolatier, Inc., Starbucks Holding Company; Kraft Foods Global, Inc.; Apffels Coffee, Inc., Coffee Bean International, Inc., Dona Mireya, Inc., dba Jones Coffee Roasters; Equator Coffee &amp; Teas; Boyer Coffee Company; Caffe Ibis, Inc.; The Coca- Cola Company; Community Coffee Company, Inc.; Copper Moon Coffee, LLC; JBR, Inc., dba Rogers Family Company; Lavazza Premium Coffees Corp.; Cascade Coffee, Inc.; Coffee Roasters of Arizona, Inc.; Gold Medal Products Co.; Millstone Coffee, Inc.; Mother Parkers Tea &amp; Coffee, Inc.; Southern Wine and Spirits of America, Inc.; Central Coast Coffee Roasting Co., Inc.; James c. Cannell Coffees, Inc. Db a Jim's Organic Coffee; Paradise Beverages, Inc. dba Hawaii Coffee Company; Regal Commodities; Steep &amp; Brew, Inc.; Victor Allen's Coffee, LLC; Napa Valley Coffee Roasting Company; Kauai Coffee Company LLC; Peerless Coffee Co., Inc., dba Adam's Organic Coffees; Montana Coffee Traders, Inc.; Falcon Trading Company, Inc.; Intelligentsia Coffee &amp; Tea, Inc.; Mayorga Coffee, LLC; Hometown Coffee Co.; New England Tea and Coffee Co., Inc.; Zavida</p>
---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------	--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------

**DECLARATION OF DR. RONALD L. MELNICK IN SUPPORT OF PLAINTIFF'S  
MOTION FOR SUMMARY ADJUDICATION OF DEFENDANTS' ALTERNATIVE  
SIGNIFICANT RISK LEVEL ("ASRL") DEFENSE**

TELEPHONE (562) 437-4499  
TOLL-FREE (877) TOX-TORT  
TELECOPIER (562) 436-1561  
WWW.TOXICTORTS.COM

LAW OFFICES OF  
**RAPHAEL METZGER**  
A PROFESSIONAL LAW CORPORATION  
401 EAST OCEAN BOULEVARD, SUITE 800  
LONG BEACH, CALIFORNIA 90802-4966

PRACTICE CONCENTRATED IN TOXIC  
TORT & ENVIRONMENTAL LITIGATION  
OCCUPATIONAL & ENVIRONMENTAL LUNG  
DISEASE, CANCER, AND TOXIC INJURIES

1  
2  
3  
4  
5  
6  
7  
8  
9  
10  
11  
12  
13  
14  
15  
16  
17  
18  
19  
20  
21  
22  
23  
24  
25  
26  
27  
28

Coffee Company, Inc.; Quartermaine Coffee Roasters; S & D Coffee, Inc.; Verve Pacific Avenue Café, LLC; Eight O'Clock Coffee Company)

Brendan W. Brandt, Esq.  
Andrew Ross  
Varner & Brandt  
3750 University Ave., Suite 610  
Riverside, CA 92501  
(Stater Bros. Markets)

J.T. Wells Blaxter, Esq.  
Brian R. Blackman, Esq.  
Erin W. Keefe, Esq.  
Blaxter | Blackman LLP  
One Bush St., Suite 650  
San Francisco, CA 94104  
(Whole Foods Market California, Inc.; Allegro Coffee Company)

Lawrence Y. Wong, Esq.  
Darryl J. Horowitz, Esq.  
Coleman & Horowitz, LLP  
1880 Century Park East, Suite 404  
Los Angeles, CA 90067  
(Luberski, Inc., dba Hidden Villa Ranch)

Megan Irwin, Esq.  
Bryan Cave LLP  
3161 Michelson Dr., Suite 1500  
Irvine, CA 92612-4414  
(Kerry Inc., dba Kerry Ingredients, Inc.)

Megan E. Irwin, Esq.  
Bryan Cave LLP  
Two N. Central Avenue, Suite 2200  
Phoenix, AZ 85004  
(Co-counsel for Kerry Inc., dba Kerry Ingredients, Inc.)

Ian K. Boyd, Esq.  
Matthew A. Stratton, Esq.  
Harvey Siskind LLP  
Four Embarcadero Center, 39<sup>th</sup> Floor  
San Francisco, CA 94111  
(Rockstar, Inc.)

Tara Sky Woodward, Esq.  
Bradley Arant Boulton Cummings LLP  
1615 L Street, N.W., Suite 1350

Washington, DC 20036  
(*Specially Appearing* for S&D Coffee, Inc.)

Charles F. Gorla, Esq.  
Gorla, Weber & Jarvis  
1011 Camino del Rio South, Suite 210  
San Diego, CA 92108  
(Café Calabria Coffee Roasting Company)

Lawrence E. Skidmore, Esq.  
Kathleen C. Lyon, Esq.  
Erin J. Tognetti, Esq.  
Aronowitz Skidmore Lyon  
200 Auburn Folsom Road, Suite 305  
Auburn, CA 95603  
(L. Paul Phillips dba Safari Morning Coffee)

Gary M. Roberts, Esq.  
Melanie A. Tory, Esq.  
SNR Denton US LLP  
601 S. Figueroa Street, Suite 2500  
Los Angeles, CA 90017  
(Churchill Coffee Company, LLC)  
*Settlement pending*

Updated 02/12/16 nsv

TELEPHONE (562) 437-4499  
TOLL-FREE (877) TOX-TORT  
TELECOPIER (562) 436-1561  
WWW.TOXICTORTS.COM

LAW OFFICES OF  
**RAPHAEL METZGER**  
A PROFESSIONAL LAW CORPORATION  
401 EAST OCEAN BOULEVARD, SUITE 800  
LONG BEACH, CALIFORNIA 90802-4966

PRACTICE CONCENTRATED IN TOXIC  
TORT & ENVIRONMENTAL LITIGATION  
OCCUPATIONAL & ENVIRONMENTAL LUNG  
DISEASE, CANCER, AND TOXIC INJURIES

**PROOF OF SERVICE**

1  
2  
3  
4  
5  
6  
7  
8  
9  
10  
11  
12  
13  
14  
15  
16  
17  
18  
19  
20  
21  
22  
23  
24  
25  
26  
27  
28

STATE OF CALIFORNIA, COUNTY OF LOS ANGELES )

I am employed in the County of Los Angeles, State of California. I am over the age of 18 years and am not a party to the within action. My business address is 401 East Ocean Blvd., #800, Long Beach, CA 90802.

On May 16, 2016, I served the foregoing document, described as: **DECLARATION OF DR. RONALD L. MELNICK IN SUPPORT OF PLAINTIFF'S MOTION FOR SUMMARY ADJUDICATION OF DEFENDANTS' ALTERNATIVE SIGNIFICANT RISK LEVEL ("ASRL") DEFENSE** on the parties to this action as follows:

    X     (BY MAIL) I caused copies of such document, enclosed in sealed envelopes, to be deposited in the mail at Long Beach, California with postage thereon fully prepaid to:

Comfort Foods, Inc.  
25 Commerce Way, Suite 5  
North Andover, MA 01845-1002  
(Comfort Foods, Inc.)

I am "readily familiar" with the firm's practice of collecting and processing correspondence for mailing. It is deposited with U.S. Postal Service on that same day in the ordinary course of business. I am aware that on motion of any party served, service is presumed invalid if the postal cancellation date or postage meter date is more than one day after the date of deposit for mailing set forth in this affidavit.

         (BY FACSIMILE) I served the foregoing document by faxing true copies thereof from facsimile number (562) 436-1561, to the facsimile numbers indicated on the attached list. Said document was transmitted by facsimile transmission, which was reported complete and without error.

         (BY OVERNIGHT MAIL) I caused such document to be delivered to the firms indicated on the attached list by Express Mail or by another express service carrier, by placing the document in an envelope designated by the carrier and addressed as indicated on the attached list, with the delivery fees provided for, and depositing same in a box or facility regularly maintained by that carrier or by delivering same to an authorized courier or driver authorized by the carrier to receive documents.

I declare under penalty of perjury under the laws of the State of California that the above is true and correct.

Executed on May 16, 2016, at Long Beach, California.

  
\_\_\_\_\_  
Nina S. Vidal, Declarant

# **EXHIBIT “B”**

## **Critique of Dr. William Ristenpart's Report and Testimony**

In his brief listing of opinions regarding the formation and mitigation of acrylamide in roasted coffee, Dr. Ristenpart wrote (page 4, opinion 3) that “no viable commercial measures for reducing acrylamide in coffee are currently available.” This inaccurate portrayal of the current status on acrylamide mitigation is addressed in my comments provided below.

### **1) Dr. Ristenpart is apparently not aware of the extensive work that has been done to reduce levels of acrylamide in coffee**

In his report (3a), Dr. Ristenpart acknowledges that there are numerous patents and articles available demonstrating reduction of acrylamide in roasted coffee by treatments of coffee beans pre-roasting, during roasting, or post-roasting; however, he then defers to a document he titled USDA [actually FDA] Guidance for Industry Acrylamide in Foods, which “contains nonbinding recommendations” and that claimed “a viable commercial process is not yet available.” The latter claim was based on a 2013 document prepared by FoodDrinkEurope, “Acrylamide Toolbox 2013.” In his deposition, he also refers to an article by Seal et al., (Risk-Benefit Considerations of Mitigation Measures on Acrylamide Content of Foods -- a Case Study on Potatoes, Cereals and Coffee, Br J Nutrition 99 Suppl 2: S1-S46, 2008) that claimed “no significant further mitigation [of acrylamide in coffee] appears in sight,” which was based on a 2005 article by Grob (reference 83). The 2008 article by Seal (which was Commissioned by ILSI Europe) and the 2005 reference of Grob are certainly outdated and do not reflect the numerous advances that have been made to reduce acrylamide levels in roasted coffee.

For some unknown reason, the Acrylamide Toolbox 2013 failed to mention the successes in reducing acrylamide levels in roasted coffee using asparaginase that had been described in patents filed by Proctor & Gamble Company (Dria et al., Method for Reduction of Acrylamide in Roasted Coffee Beans, Roasted Coffee Beans Having Reduced Levels of Acrylamide, and Article of Commerce. US 2004/0081724 A1, The Proctor & Gamble Company, 2004; and Dria et al., Method for Reduction of Acrylamide in Roasted Coffee Beans, Roasted Coffee Beans Having Reduced Levels of Acrylamide, and Article of Commerce. US 7,220,440 B2, The Proctor & Gamble Company, 2007) and by Illy Caffé (Navarini et al., Method for Reducing the Content of Acrylamide in a Roasted Coffee, WO 2013/005145 A1, Illy Café SPA, 2013). The latter patent describes their product “in which the desired organoleptic properties remain unaltered.”

Dr. Ristenpart wrote that there are several patents and articles in the scientific literature that describe techniques for reducing acrylamide levels in roasted coffee and at his deposition he stated that he “read the articles cited by Dr. Melnick in his declaration.” He also stated that “many of the articles reference here [in the Seal 2008 paper] are probably also referenced in Dr. Melnick's declaration;” that statement is incorrect as a large majority of the articles that I cited were published after 2008. Evidently, Dr. Ristenpart did not believe the claims by Proctor & Gamble Company and by Illy Caffé in their filed patents or he may not have read those patents very carefully (at his deposition, he acknowledged [page 30] that he did not review the 2004 P&G patent application). It is not clear if the patent applications from Proctor & Gamble Company and from Illy Caffé were provided to Dr. Ristenpart before his deposition. At his

deposition, Dr. Ristenpart stated “I wasn’t aware that there was an active research thrust [to reduce acrylamide levels in coffee] by any particular company (page 143).

**2) Dr. Ristenpart is apparently not aware of the fact that the production of roasted coffee beans with reduced levels of acrylamide (from asparaginase treatment of green coffee beans) has been scaled up to commercial production levels.**

In section 3d of his report, Dr. Ristenpart wrote “in my review of the patent and scientific literature by Dr. Melnick, and in my own external review of the current literature, I find no evidence of acrylamide remediation or reduction techniques for coffee that have been successfully scaled up to commercial production levels.” Though Dr. Risenpart may not have found such evidence, I became aware of industry reports that were produced in this litigation pursuant to a protective order that contradict Dr. Ristenpart’s opinion on this point.

In September 2011, Helmut Guenther, Food Scientist at Kraft Foods in Germany, prepared an “Update on Acrylamide and Using Asparaginases to Reduce Levels in Coffee” in which he updated the European coffee industry regarding a collaborative effort between Novozymes and Hermsen/CR3, a German coffee roaster, writing: “Novozymes is aware of the current coffee industry position that using enzymes is not seen as an option to reduce - for efficiency, quality, cost and food safety reasons (as e.g. detailed in the FoodDrinkEurope Acrylamide Toolbox) and is addressing this by showing data with achieved reductions of up to 70% (instead of our industry findings of a 10 - max 45% reduction). This is together with mentioning that coffee has been processed at industrial scale already. According to the presentation to Sara Lee, **more than 200 tons of coffee have been processed on industrial scale and sold to the market.** Additionally they are referring to the opportunity to combine the enzyme process with other green coffee treatments (steaming)...” (KRAFT-00025779 to 00025780).

It is not clear if Dr. Risenpart had this information and decided to ignore it or whether the defendants in this case withheld this important information from their expert witness.

**3) Dr. Risenpart is apparently not aware that evaluations of roasted coffee with reduced levels of acrylamide have been found to have no or only minimal impact on palatability.**

In section 3e of his report, Dr. Ristenpart wrote “many of the proposed techniques [to reduce acrylamide levels in roasted coffee] have clear drawbacks in regard to adverse impacts on palatability...” However, the fact that 200 tons of coffee were produced using Novozyme’s asparaginase technique and sold to market is proof of general consumer acceptance of Novozymes’ asparaginase treatment of coffee for the reduction of acrylamide. In addition,

a) in the vacuum roasting study by Anese et al. (Effect of Vacuum Roasting on Acrylamide Formation and Reduction in Coffee Beans. Food Chem. 145:168-172, 2014), the authors noted that sensory analyses showed that “the medium-roasted coffee samplers obtained by means of the conventional and vacuum processes and having different acrylamide levels, were not perceived as different by the assessors.” [Note: at his deposition, Dr.

Ristenpart claimed (pages 190-191) that the reduced level of acrylamide was observed in this study “when the beans were still yellow” ...”and that is not commercially viable because nobody wants yellow coffee.” On this issue, Anese et al. stated that vacuum roasting was effective in reducing acrylamide in **medium roast beans**, which can provide commercial opportunities since medium-roasted coffee consumption is relatively high for American and North European markets].

b) in the supercritical CO<sub>2</sub> extraction study by Banchemo et al. (Supercritical Fluid Extraction as a Potential Mitigation Strategy for the Reduction of Acrylamide Level in Coffee. *J Food Engineering* 115:292-297, 2013), the authors conclude that the mitigation strategy had only a slight effect on sensory properties of coffee as judged by coffee-testing experts on coffee brews prepared with samples of the coffee beans that had been previously subjected to the supercritical treatment.

c) in the patent filed by Illy Caffé concerning the reduction of acrylamide in roasted coffee by treatment of green coffee beans with asparaginase, the authors note “the method according to the invention enables a roasted coffee to be obtained that has a reduced acrylamide content, in which the desired organoleptic properties remain unaltered and can be appreciated by the consumer.”

d) Richard Stadler, the Head of Nestlé’s Quality Management Department, reported (*Food Process Contaminants: Industry Perspectives and Update on Mitigation. Euro Food Chem XVII, Istanbul, Turkey, 2013*) that steaming coffee beans (100 °C for 45 min) followed by soaking (50% water, 60°C) with asparaginase and aspartase reduced acrylamide formation by ~70% during roasting “with no significant impact on organoleptic properties.”

e) In a more recent study with asparaginase treatment of green coffee beans (Xu et al., *Effect of Asparaginase on Flavour Formation in Roasted Coffee*, in *Flavour Science: Proceedings of the XIV Weurman Flavour Research Symposium*, Sept. 15-19, 2014, Queen’s College Cambridge, pp. S63-S66, 2015), the investigators reported “up to 84% reduction of acrylamide was achieved with only minor changes in the composition of the most concentrated aroma compounds formed when the coffee was roasted.”

f) in 2005, Kraft Scientists reported that Informal tasting of cured coffees, which achieved a 74% reduction of acrylamide, demonstrated the effectiveness of the nitrogen atmosphere in preventing the formation of off flavors (KRAFT-00025891 to 00025896).

In my view, the opinion expressed by Dr. Ristenpart concerning effects of acrylamide reduction on palatability lacks foundation.

#### **4. Dr. Ristenpart Misunderstood the Benefit of Extended Storage Time on Reduction of Acrylamide Levels in Roasted Coffee Beans or Ground Coffee.**

In dismissing the value of extended storage time to reduce acrylamide levels in roasted coffee at his deposition, Dr. Ristenpart refers to an article by Perez-Martinez et al. (Changes in Volatile Compounds and Overall Aroma Profile during **Storage of Coffee Brews** at 4 and 25 °C, J. Agric. Food Chem. 56:3145-3154, 2008) in which the authors reported a decreases in fresh aroma with an increase rancid aroma within 3 and 7 days of storage (at 4 and 25 °C, respectively) of hermetically sealed brewed Arabica coffee. This article is irrelevant to the effects of storage on roasted coffee beans or ground coffee because acrylamide reduction during storage is due to its covalent binding to insoluble components of the coffee bean, which are not present in coffee brews, and the development of a rancid aroma within 3-7 days of storage is prevented when ground or whole coffee beans are stored under vacuum or with nitrogen gas under pressure. To further support his opinion, Dr. Ristenpart refers to an article by Ross et al. (Effect of Storage Conditions on the Sensory Quality of Ground Arabica Coffee, J. Food Quality 29:596-606, 2006). In this study, roasted Arabica coffee was found to be significantly more bitter when ground coffee beans were stored in tin-tied mylar-gusseted bags for 1, 2, or 3 weeks at room temperature or at -23 °C. This study also does not adequately capture the benefits of extended storage for vacuum-packed roasted coffee beans and ground coffee. For example, Starbucks specifies a shelf of up to 60 weeks for ground and whole bean roasted coffee (STARBUCKS-00011632 to 00011656), while Illy claims that due to their inert gas pressurization packaging technology, “the flavor and freshness of the unopened Illy coffee can be fully preserved for a long period of 2 years” while opened Illy coffee remains fresh for 7 days at room temperature ([https://shop.illy.com/online/store/termsview\\_E-SPOT-Footer-Column01-Row02\\_ec](https://shop.illy.com/online/store/termsview_E-SPOT-Footer-Column01-Row02_ec)). The Illy packaging technique is claimed to improve aroma over time by causing volatile aroma compounds to bind to oils contained in the coffee beans. Thus, the method used to package coffee beans or ground coffee is critical for evaluating the effect of storage time on coffee flavor and aroma. In contrast to volatile aroma compounds, acrylamide levels decrease with extended storage time due to its covalent binding to compounds in coffee grounds (Baum et al., Fate of <sup>14</sup>C-Acrylamide in Roasted and Ground Coffee During Storage. Mol. Nutr. Food Res. 52:600-608, 2008).

#### **5. Dr. Ristenpart fails to recognize that the coffee industry needs to pursue scale-up of methods that are effective in reducing acrylamide levels in roasted coffee.**

While numerous techniques have been developed to reduce acrylamide levels in roasted coffee, in many cases, the authors of these studies do not have the resources or expertise to scale up their method to commercial production levels or to fully evaluate the impact of their method on organoleptic properties of their product. However, the findings from these investigators provide the coffee industry with potentially useful approaches to produce a healthier product. In my view, it is incumbent on the coffee industry to be proactive in pursuing the further development of promising methods. However, it seems that from the limited actions and non-actions that the coffee industry managers have taken recently to reduce the levels of the mutagenic carcinogen acrylamide from their product, they anticipate greater success from litigation rather than from mitigation of acrylamide in coffee.

**EXHIBIT “C”**

1 SUPERIOR COURT OF THE STATE OF CALIFORNIA

2 FOR THE COUNTY OF LOS ANGELES

3 DEPARTMENT 323

HON. ELIHU M. BERLE, JUDGE

4

5 CERT, )  
6 )  
7 ) Plaintiff, )  
8 ) vs. ) SUPERIOR COURT  
9 ) CASE NO. BC 435759  
10 ) BC 461182  
11 )  
12 ) STARBUCKS CORP, ET AL., )  
13 )  
14 ) Defendants. )  
15 )  
16 )

---

17 REPORTER'S TRANSCRIPT OF PROCEEDINGS

18 Monday, October 2, 2017

19 (A.M. Session)

20 APPEARANCES OF COUNSEL:

21 FOR THE PLAINTIFFS: METZGER LAW GROUP  
22 BY: RAPHAEL METZGER, ESQ.  
23 ABRAHAM I. PARISER, ESQ.  
24 401 East Ocean Boulevard  
25 Suite 800  
26 Long Beach, California 90802  
27 (562) 437-4499  
28 sbrust@toxictorts.com  
rmetzger@toxictorts.com  
apariser@toxictorts.com

21 FOR THE ROASTER AND DOE DEFENDANTS:  
22 MORRISON/FOERSTER  
23 BY: JAMES M. SCHURZ, ESQ.  
24 425 Market Street  
25 San Francisco, California 94105-2482  
26 (415) 268-7124  
27 jschurz@mofo.com

28 (Appearances continued on next page.)

DAVID A. SALYER, CSR, RMR, CRR  
Official Pro Tem Court Reporter  
License No. 4410

1 APPEARANCES OF COUNSEL: (CONTINUED)

2 FOR KEURIG: SKADDEN, ARPS, SLATE, MEAGHER  
& FLOM, LLP  
3 BY: RAOUL D. KENNEDY, ESQ.  
4 525 University Avenue  
Palo Alto, California 94301  
(650)470-4550  
5 rkennedy@skadden.com

6 FOR HN FERNANDEZ, ET AL.:

7 NORTON ROSE FULBRIGHT, LLP  
8 BY: JEFFREY B. MARGULIES, ESQ.  
555 South Flower Street  
41st Floor  
9 Los Angeles, California 90071  
(213)892-9286  
10 jmargulies@nortonrosefulbright.com

11  
12  
13  
14  
15  
16  
17  
18  
19  
20  
21  
22  
23  
24  
25  
26  
27  
28



1 CASE NUMBER: BC 411192/BC435759  
2 CASE NAME: CERT CASES  
3 LOS ANGELES, CALIFORNIA MONDAY, OCTOBER 2, 2017  
4 DEPARTMENT 323 ELIHU M. BERLE, JUDGE  
5 REPORTER: DAVID A. SALYER, CSR 4410  
6 TIME: 9:15 a.m.

7 -o0o-

8 THE COURT: All right. In CERT versus Starbucks,  
9 counsel ready to proceed?

10 MR. METZGER: Yes, your Honor.

11 THE COURT: All right. I'm not in the comfort zone.  
12 I was worried I didn't get any new briefs on this case this  
13 morning. We don't want to have a morning without briefing.

14 MR. MARGULIES: It's here.

15 THE COURT: Okay. Mr. Metzger, are you ready to call  
16 your next witness?

17 MR. METZGER: Yes.

18 The plaintiff calls Dr. Ronald Melnick.

19 THE COURT: Just one second. I want to set up the  
20 LiveNote.

21 Just one moment, please.

22 Okay. All right.

23 I'll ask the clerk to swear the witness.

24

25 RONALD MELNICK,  
26 having been called as a witness and sworn testified as  
27 follows:

28 THE WITNESS: I do.

1 THE CLERK: And would you please state and spell your  
2 name for the record.

3 THE WITNESS: Ronald Melnick, R-O-N-A-L-D,  
4 M-E-L-N-I-C-K.

5 THE CLERK: Thank you.

6 THE COURT: Good morning, Dr. Melnick.

7 Mr. Schurz, you were standing for some reason?

8 MR. SCHURZ: I was, your Honor.

9 I thought before we proceeded with Dr. Melnick, there  
10 was one issue of housekeeping that your Honor had asked us to  
11 address, and I believe we have done so.

12 We've been directed to do this on the record. It  
13 relates to the exchange of a document that's been identified,  
14 Exhibit 73540.

15 Counsel have met and conferred and agreed that the only  
16 change here is the branding of this exhibit with page numbers.  
17 And we would, at this time, ask permission to exchange  
18 Exhibit 73540 with the one that has been branded with page  
19 numbers.

20 THE COURT: Any objection?

21 MR. METZGER: No, your Honor.

22 THE COURT: The Court will order the substitution.

23 Thank you.

24 MR. SCHURZ: Thank you.

25 THE COURT: Mr. Metzger, you may proceed.

26 ///

27 ///

28 ///

**DIRECT EXAMINATION**

1  
2 BY MR. MELNICK:

3 Q. Good morning, Dr. Melnick.

4 A. Good morning.

5 Q. I think it's been about three years since you  
6 sat in that chair.

7 A. Yes, it's been three years almost exactly.

8 Q. Well, welcome back.

9 So let's see. In the first phase trial, you testified  
10 about risk assessment and some other subjects. And now you've  
11 done more work on the case; is that correct?

12 A. Quite a bit of work.

13 Q. Okay.

14 A. A lot more.

15 Q. All right. First, a few housekeeping things.  
16 Would you confirm that what I'm handing you,  
17 Exhibit 60076, is your current curriculum vitae?

18 A. It's close to current.

19 Q. Okay.

20 A. I attended another meeting of the International  
21 Agency for Research on Cancer in June of this year, and  
22 typically when I participate in those I add those to my CV.

23 So I believe this doesn't include that.

24 Q. All right. And in attending that meeting, were  
25 you an actual member of the IARC Working Group?

26 A. Yes. I was invited by IARC to participate in  
27 that meeting as a member of the Working Group.

28 Q. All right. And what was that meeting regarding?

1           A.       There were approximately seven different agents,  
2 most of them which are somehow found in foods.

3           And the report, the initial report hasn't come out, the  
4 monograph, but a publication was put out in the Lancet  
5 Oncology, and in that the title of it is described as  
6 chemicals that cause urinary tract tumors.

7           Q.       All right. Regarding your education, would you  
8 refresh the Court as to what your degrees are in.

9           A.       Okay. I have a BS and MS and Ph.D in food  
10 science.

11           And in my graduate studies, and this was at the  
12 University of Massachusetts at Amherst, I also was providing  
13 an emphasis in biochemistry.

14           Q.       Okay. You spent many years at the National  
15 Toxicology Program involved with animal cancer bioassays; is  
16 that correct?

17           A.       Yes. I joined the National Toxicology Program  
18 in 1980, and I retired from that program in January of 2009.

19           So it's approximately -- almost 29 years.

20           Q.       Okay. And you also worked at the National  
21 Institute for Environmental Health Sciences; is that correct?

22           A.       Yes.

23           The National Toxicology Program is composed of several  
24 components within the Department of Health and Human Services.

25           The major component is located at the National  
26 Institute of Environmental Health Sciences in North Carolina.  
27 This is the one institute of the National Institutes of Health  
28 which is not located in Bethesda.

1 Q. Okay. I see that you won a National Institute  
2 of Health Plain Language Award for developing a brochure  
3 entitled Cancer and the Environment. What You Need to Know.  
4 What You Can Do; is that correct?

5 A. Yes. I remember that.

6 Q. Okay. Approximately how many working groups  
7 have you been invited by the International Agency for Research  
8 on Cancer to attend and participate as a Working Group member?

9 A. Well, I've been invited to IARC, if I can use  
10 that acronym, 13 times.

11 Ten of those times were related to monograph meetings  
12 which evaluate the carcinogenicity data for a large number of  
13 chemicals.

14 The other times related to mechanisms.

15 In fact, there will be a book coming out fairly shortly  
16 from some of that work which relates to after having conducted  
17 100 volumes of IARC monographs, a number of chemicals were  
18 identified as human carcinogens. So it was what have we  
19 learned during that course of time with respect to the  
20 mechanisms of carcinogenesis as well as site concordance  
21 between animals and humans.

22 Q. Would you inform Judge Berle of some of your  
23 publications that you considered to be relevant to this phase  
24 of the trial.

25 A. Okay. Well, I've spent a lot of effort related  
26 to chemicals which metabolize to epoxide intermediates. One  
27 that I've now published numerous studies on is 1,3-Butadiene.  
28 This is a chemical used in the synthetic rubber industry

1 making styrene butadiene rubber, for example.

2 And as a consequence of working with butadiene, I  
3 nominated to the NTP that we should also study chloroprene and  
4 isoprene.

5 The work on butadiene was a combination of studies.

6 For example, the animal bioassay determining what are  
7 the sites of cancer induction as well as redesigning the study  
8 after the first to better characterize the dose-response  
9 relationships. So the larger study up to that time was my  
10 butadiene study with five exposure levels.

11 In addition to that, we also developed oncogenetic  
12 models on butadiene to try to characterize the dosimetry,  
13 which I'll probably explain later, of the epoxide  
14 intermediates that are formed from butadiene. And those are  
15 the ones that are considered to be involved in the  
16 carcinogenicity of that chemical.

17 Q. What is the relationship or import of your work  
18 regarding epoxides and mechanisms of carcinogenesis for  
19 epoxides with respect to acrylamide?

20 A. Okay. Well, acrylamide is metabolized the same  
21 way as 1,3-butadiene, the same way as vinyl chloride to an  
22 epoxide intermediate chlorpropamide. The epoxide intermediate  
23 is glycidamide. And it reacts with DNA, similarly to the  
24 oxide intermediates of butadiene and the epoxide intermediate  
25 of vinyl chloride.

26 Ethylene oxide is an epoxide as purchased and it also  
27 behaves similarly.

28 Q. Okay. I believe that one of the topics that

1 you're going to talk about today is primary prevention of  
2 cancer; is that correct?

3 A. Yes, I would.

4 Q. And I see that you have published in the  
5 peer-reviewed literature some articles regarding that one  
6 entitled "Primary Prevention of Cancer," published in The  
7 Scientist, 2002.

8 Do you remember that?

9 Look at the bottom of page 20 of your curriculum vitae.

10 A. Let me just say that the work of the National  
11 Toxicology Program is designed to identify agents in the  
12 environment or workplace which pose a carcinogenic potential.

13 That information is intended for use by regulatory  
14 agencies to eliminate or reduce human exposure to chemicals  
15 which are hazardous to human health.

16 That is what we consider primary prevention. The  
17 prevention of the development of the disease and in this case  
18 by reducing or eliminating exposure to the agent which would  
19 induce cancer.

20 Q. Okay. I see you also have an article in  
21 Environmental Health Prospectus entitled "Declaring Chemicals  
22 Not Carcinogenic to Humans Requires Validation, Not  
23 Speculation."

24 Could you tell us generally what that's about.

25 A. I believe -- could you give me the number again?

26 Q. It's number 100.

27 A. Okay. At this time there was a number of  
28 studies that we had conducted, others had conducted, relating

1 to kidney cancer in rats caused by agents which induces a  
2 certain protein. It's called alpha-2-globulin. And there was  
3 attempts by certain people to claim that if you see  
4 alpha-2-globulin neuropathy, the disease in the kidney, that  
5 that would be sufficient evidence to claim that it is not a  
6 human carcinogen because that would be the mechanism of  
7 carcinogenesis and humans don't produce alpha-2-globulin.

8 It's a hypothesis. And what we believed was that  
9 rather than speculating that that is the case, we need  
10 scientific evidence to test a hypothesis before implementing  
11 it for public health reasons.

12 Q. Okay. And let's see.

13 I'm looking at the bottom of page 25 of your curriculum  
14 vitae.

15 Is this item number 17, Bond and Melnick,  
16 "Electrophilic Compounds in Tumor Concordance and Mechanisms  
17 of Carcinogenesis," an IARC scientific publication in press --  
18 is that the publication that you were speaking of earlier that  
19 is coming out?

20 A. Yes. That one has been in press for at least  
21 six or seven months, so it's due out anytime.

22 When I was at IARC in June, I asked that same question,  
23 and I was told it would be another month or two.

24 Q. Okay.

25 A. But I still haven't seen it.

26 Q. So I don't want to spend a lot of time on your  
27 experience and qualifications because you've already testified  
28 in the phase one trial.

1           But is there anything in particular that you would wish  
2 to share with the Court regarding your experience that you  
3 think relates, in particular, to the issues that you'll be  
4 testifying about today?

5           A.       Well, like some of the questions that you asked  
6 me, I've written papers about mechanisms of carcinogenesis for  
7 epoxide-forming chemicals. I've worked on pharmacokinetic  
8 modeling of chemicals, including those that form epoxide  
9 intermediates.

10           I've conducted -- led the efforts for numerous animal  
11 bioassays for the National Toxicology Program.

12           I have served on, like I mentioned, ten IARC Working  
13 Group evaluations of carcinogenicity.

14           I've also served on a number of review groups for EPA  
15 in their IRIS evaluations on risk assessment of chemicals.

16           I've served as a reviewer for journal articles, a  
17 reviewer for contract proposals.

18           I'm not sure how extensive you want me to go, but a lot  
19 of this is -- and I might say that much of this is still  
20 ongoing even though I've retired from NTP.

21           Q.       Okay. Turning to acrylamide, when did you first  
22 begin research regarding acrylamide?

23           A.       Well, I think I was always aware of acrylamide  
24 being a carcinogen, because it had been studied numerous years  
25 ago.

26           In 2002 it was identified as a chemical present in  
27 foods.

28           And in 2006 I was asked by the California State

1 Attorney General's Office if I would consult with them on the  
2 case that they were working on related to acrylamide and  
3 acrylamide reduction in french fries and potato chips.

4 Q. Okay.

5 A. So the history -- my history related to  
6 acrylamide was awareness of it as a chemical carcinogen back  
7 in the nineties, but the intensity of my interest increased as  
8 I served as a consultant for the California Attorney General.

9 Q. All right. And we met through your service for  
10 the California Attorney General on the prior acrylamide  
11 litigation regarding french fries and potato chips, correct?

12 A. Yeah, I was deposed several times on that, and I  
13 believe you were in the room one or two of those.

14 Q. All right.

15 MR. METZGER: Your Honor, I would offer at this time in  
16 evidence trial Exhibit 60076, Dr. Melnick's almost current  
17 curriculum vitae.

18 THE COURT: Any objection?

19 MR. KENNEDY: No objection, your Honor.

20 THE COURT: The exhibit is admitted.

21 (Exhibit 60076 received in evidence.)

22 Q. BY MR. METZGER: Let's see.

23 Dr. Melnick, is one of the things that I asked you to  
24 do regarding this case to research the published technologies  
25 regarding reduction of acrylamide, especially in coffee?

26 MR. KENNEDY: Objection, your Honor. Irrelevant that  
27 there's any duty to mitigate under the applicable statutes.

28 THE COURT: Overruled.

1 THE WITNESS: Yes. That's one of the many things you  
2 asked me to do.

3 Q. BY MR. METZGER: And did you initially prepare a  
4 declaration regarding those technologies that have been  
5 published in the peer-reviewed literature?

6 MR. KENNEDY: Same objection, your Honor. Also object,  
7 lack of foundation.

8 There's been no showing that he's an expert in  
9 acrylamide, that he's actually done any research in the area  
10 or that he's done anything other an literature search.

11 THE COURT: Overruled.

12 THE WITNESS: The question was did I prepare it?

13 Q. BY MR. METZGER: Yes.

14 A. Yes, I did.

15 Q. Okay. And then at some point did you receive  
16 confidential documents that had been produced by certain of  
17 the defendants in this case regarding acrylamide reduction?

18 MR. KENNEDY: Your Honor, object under People versus  
19 Sanchez.

20 We're now getting into case-specific hearsay. So we  
21 know the Supreme Court has ruled that doesn't qualify under  
22 801(b) unless it's been independently established by a  
23 competent witness.

24 We object to any questions along these lines unless  
25 they're either in hypothetical question form or there's  
26 specific identification of where the materials he's relying on  
27 were offered in evidence by a competent witness.

28 THE COURT: The hypothetical is assume you received

1 documents from the defendants?

2 He asked him did he receive documents.

3 MR. KENNEDY: He certainly has. Many of those contain  
4 multiple levels of hearsay.

5 THE COURT: He hasn't gotten there yet. He just asked  
6 if he received documents.

7 MR. KENNEDY: I just want to make sure -- it seems to  
8 me it's irrelevant whether he did or not unless we're talking  
9 about documents for which there is a hearsay exception and are  
10 competently admitted. Otherwise the fact he's received  
11 case-specific material is irrelevant.

12 THE COURT: Thank you.

13 Objection is overruled.

14 MR. METZGER: All right.

15 THE WITNESS: Yes, I did receive confidential  
16 documents, lots of them.

17 Q. BY MR. METZGER: Regarding --

18 A. Regarding acrylamide in coffee and means of  
19 reducing acrylamide in coffee.

20 Q. Okay. And did this include confidential studies  
21 that had been done by various coffee companies?

22 A. The documents indicated the companies that had  
23 provided this information. So much of it had been done by the  
24 coffee companies, yes.

25 Q. Okay. And did you review those documents?

26 A. Yes, I did.

27 Q. All right. And based on your review of those  
28 documents, did you expand your previous declaration to include

1 information regarding reduction of acrylamide that you had  
2 ascertained from your review of the industry confidential  
3 documents?

4 A. Yes, I did.

5 I received those in January and then added that  
6 information to my declaration from -- previously submitted.

7 Q. I'm going to show you what has been marked as  
8 Exhibit 59957 and ask you if this is the updated and expanded  
9 declaration that you prepared regarding reduction of  
10 acrylamide in coffee.

11 A. Yes, I believe it is.

12 Q. All right. I'm going to show you a few more  
13 things.

14 One is Exhibit 60076(sic), a document entitled  
15 "Opinions of Ronald Melnick."

16 I'll ask you, is this a report that you prepared  
17 setting forth some of your opinions for this second phase of  
18 the trial?

19 A. Yes, it is.

20 Q. Oh, 60077.

21 THE COURT: 60077.

22 MR. METZGER: Did I misspeak earlier?

23 Oh, I apologize.

24 Q. And as part of your work in this case, did you  
25 read the deposition as well as the trial testimony of  
26 Dr. William Ristenpart?

27 A. I also read his report. So I read his report,  
28 the transcript of his deposition and the transcript of his

1 trial testimony.

2 Q. And did you prepare a critique of  
3 Dr. Ristenpart's report in testimony?

4 A. It's of his report and deposition.  
5 I prepared a report in response to that.

6 Q. Right.

7 And is Exhibit 60081 that report critiquing Dr. William  
8 Ristenpart's report and testimony that you prepared?

9 A. Yes, it is.

10 Q. And did you also read the report and the  
11 deposition and trial testimony of Dr. David Kessler?

12 A. Yes, I have.

13 Q. And did you prepare a critique of Dr. David  
14 Kessler's report and his deposition testimony?

15 A. Yes, I did.

16 Q. And is Exhibit 60079 the report that you  
17 prepared critiquing Dr. Kessler's report and testimony?

18 A. Yes, it is.

19 Q. All right. And did you also read the deposition  
20 and the trial testimony of Dr. Lorenz Rhomberg?

21 A. Yes, I did.

22 Q. And did you prepare a report critiquing  
23 Dr. Rhomberg's report and his deposition?

24 A. Yes, I did.

25 Q. Is that Exhibit 60080?

26 A. Yes, that is it.

27 Q. Okay. You've done a lot of work on this case,  
28 and some of this is quite complex, is it not?

1 A. It could be for a number of people.

2 Yes, it is complex. There's many aspects that  
3 contribute to understanding the issues related to acrylamide,  
4 its reduction and its risk.

5 Q. And did you take it upon yourself to prepare  
6 some demonstrative aids to help with the presentation and the  
7 understanding of the testimony that you intend to give today?

8 A. Yes, I have prepared them.

9 MR. METZGER: What is the next exhibit in order?

10 MR. INFANTE: 61950.

11 Q. BY MR. METZGER: All right. Is Exhibit 61950 a  
12 printout of the slides that you prepared?

13 A. This looks like the ones.

14 Q. All right. And would you inform the Court, give  
15 us the overview of the different topics that you are prepared  
16 to talk about today.

17 A. If we could go to the next slide.

18 MR. KENNEDY: Your Honor, I want to make sure the  
19 record is protected here.

20 Bullet point 3 talks about selection of tumor sites and  
21 application of a pharmacokinetic factor.

22 In his deposition Dr. Melnick was asked the extent of  
23 his criticisms of Dr. Rhomberg and he talked about the PK  
24 factor and said absolutely nothing about tumor sites.

25 We have not been told subsequently he was planning to  
26 do that.

27 I suspect it will be more fine-tuned on the objecting  
28 when we actually get there, but I just don't want any

1 misunderstanding that we didn't object from the outset  
2 regarding any critique concerning the tumor sites.

3 THE COURT: All right. Thank you.

4 The testimony is subject to, always, cross-examination  
5 and motion to strike.

6 Mr. Metzger, you may continue.

7 MR. METZGER: All right.

8 Q. So, Dr. Melnick, what are the topics that you  
9 would like to discuss with the Court today regarding the work  
10 that you've done?

11 A. Okay. I've broken this down into six topics.

12 The first one on the principles for the determination  
13 of an NSRL are ones which have been reviewed numerous times  
14 within this Court and its involvement in performing a  
15 quantitative cancer risk assessment. But I want to just  
16 present a couple slides on that topic just to ensure that my  
17 opinions and valuations are consistent with those  
18 recommendations on how to perform a determination of an NSRL  
19 as well as the defendants.

20 Q. Incidentally, did you testify regarding tumor  
21 site selection and risk assessment in the first phase trial?

22 MR. KENNEDY: Objection. The record speaks for itself.

23 THE COURT: Overruled.

24 THE WITNESS: If I was asked the question, I'm sure I  
25 would have. But I don't recall --

26 Q. BY MR. METZGER: Three years ago.

27 A. -- whether that question came up.

28 Q. I'll join Mr. Kennedy that the record speaks for

1     itself.

2             A.       I would have to review it, the testimony.

3             Q.       I didn't mean to tax your memory.  Sorry.

4             What are the other topics that you have worked on and  
5     that you think are important to relate?

6             A.       The second is the mitigation of acrylamide in  
7     coffee to show that there are available and effective methods  
8     to substantially reduce acrylamide in coffee.

9             I want to talk about the quantitative cancer risk  
10    assessment of acrylamide in coffee that was presented in this  
11    court with emphasis on tumor sites.

12            Q.       When you say in this court, are you referring to  
13    Dr. Rhomberg?

14            A.       Yes, presented by Dr. Rhomberg.

15            Q.       Okay.

16            A.       With respect to tumor sites and application of  
17    pharmacokinetic factor.

18            The issue of tumor sites is one I've been dealing with  
19    since 1980 with respect to identifying cancer sites in animal  
20    studies and in terms of my work for IARC as well as for EPA,  
21    the sites that should be included in the cancer risk  
22    assessments.

23            The issue of quantitative benefit-risk assessment to  
24    show that there is a methodology available.

25            And a big topic is what do we mean by sound  
26    considerations of public health and how do those sound  
27    considerations of public health influence the concept of a  
28    cancer risk at 1 per 100,000 versus 1 per 10,000, which is one

1 times 10 to the minus 5.

2 Q. Let me interrupt you for just one second.

3 You mentioned a quantitative benefit-risk assessment of  
4 coffee. Is that different from a quantitative risk assessment  
5 of coffee?

6 A. Yes, it is.

7 Q. What is the difference?

8 A. In one case the quantitative cancer risk  
9 assessment is of acrylamide which is present in coffee, so  
10 what is the risk level from acrylamide in coffee.

11 The benefit-risk assessment is an evaluation of the  
12 benefits and risks of coffee with consideration of whether or  
13 not acrylamide is present.

14 Q. Okay.

15 A. And then I will present, after my last bullet,  
16 my overall conclusions on the issues of acrylamide in coffee.

17 Q. All right. So let's start, if we could, with  
18 the principles also for the determination of the NSRL,  
19 quantitative cancer risk assessment.

20 And what are the principles that you considered to be  
21 important?

22 A. If we can go -- thank you.

23 MR. KENNEDY: Objection, your Honor, to bullet points 3  
24 and 4. They're pure legal interpretations, and they aren't  
25 even accurate legal interpretations.

26 THE COURT: All right. Counsel, you can argue it  
27 later.

28 Let's move forward.

1 Q. BY MR. METZGER: Go ahead, Dr. Melnick.

2 A. Okay. So the lifetime exposure for an NSRL is  
3 one which results in not more than one excess cancer in an  
4 exposed population of 100,000.

5 This is specified as the non-significant risk level, or  
6 the NSRL.

7 Currently for acrylamide the safe harbor level is  
8 0.2 micrograms per day. If you look this up on OEHHA's sites,  
9 that's what you will find.

10 I know the presentation by Dr. Rhomberg indicated that  
11 19 micrograms was a risk that would be appropriate.

12 And I just want to point out that the ratio of  
13 19 micrograms to 0.2 micrograms is approximately a 100-fold  
14 increase over the current non-safe harbor level.

15 However, an alternative --

16 Q. Excuse me. When you said would be appropriate,  
17 are you speaking of your opinion or Dr. Rhomberg's opinion?

18 A. The opinion that he put forward.

19 Q. Okay.

20 A. However, an alternative level is one which could  
21 be considered, but it must be supported by sound  
22 considerations for public health. And they're specified as  
23 where chemicals are produced by cooking necessary to render  
24 the food palatable or to avoid microbiological contamination.

25 Those are examples that are provided. They are not  
26 necessarily all of the factors, but those are the ones that  
27 were cited.

28 THE COURT: Any numerical limitations on that?

1           Whatever the ultimate number is, somebody comes in and  
2 says, well, cooking is necessary to make food palatable and  
3 therefore we can pick any number. We would like to eat wild  
4 mushrooms found in the forest. One out of two is okay.

5           THE WITNESS: Well, in my view that might be arbitrary  
6 in terms of the selection of the number.

7           THE COURT: In other words, where do you draw the line?  
8 What's the number?

9           THE WITNESS: An appropriate risk level is actually a  
10 policy decision.

11           The policy decision I think was put forward in the  
12 Prop 65 rule. The definition of non-significant risk level is  
13 1 per 100,000.

14           To deviate from that -- I've seen in this court it's  
15 been mentioned as an alternative significant risk level, an  
16 ASRL, which I sort of object to that term because it's an  
17 alternative risk level.

18           So it is implying that the citizens of California can  
19 be exposed to a chemical without warning in which the risk is  
20 greater than 1 per 100,000.

21           THE COURT: Right. And then what's the limit?

22           THE WITNESS: This would have to be one in which people  
23 would be willing to accept.

24           So, for example, if the state wanted to put out a rule  
25 and ask the citizens would you accept a ten-fold higher cancer  
26 risk --

27           THE COURT: We're not in front of the legislature here,  
28 and we're not putting any propositions on the ballot.

1 I mean, I read stories occasionally about some exotic  
2 foods that may be dangerous to consumption, but people do eat  
3 it. They're taking a risk by preparing it properly, but  
4 nevertheless there's risk.

5 I'm just asking what is the limit when you say  
6 alternative level to make the food palatable?

7 THE WITNESS: I think the acceptance of risk is  
8 personal.

9 For example, we all assume certain risks when we leave  
10 our home and drive on highways. So what is an acceptable  
11 level is really up to either the people or the legislature to  
12 decide what is appropriate.

13 EPA and FDA have an acceptable risk of 1 per 100,000.

14 I know I'm not asking your question.

15 THE COURT: But we don't have a legislature here. We  
16 have a regulation and it says alternative risk.

17 So what is it?

18 MR. METZGER: We're going to get to that.

19 Q. Does it require a calculation, Dr. Melnick?

20 A. You can do a benefit-risk analysis and see --

21 THE COURT: Is that a policy decision or a legal  
22 decision?

23 I guess I'll let the lawyers argue that.

24 Go ahead, counsel.

25 THE WITNESS: And the last point here was from the  
26 final statement of reasons, whereas if the beneficial effects  
27 do not outweigh the risks, then the 10 to the minus 5 standard  
28 applies.

1 Q. Is that an important concept for your opinion in  
2 this case?

3 A. Yes, it is. Because in order to move away from  
4 that 10 to the minus 5 standard, beneficial effects must be  
5 demonstrated.

6 THE COURT: Is there a mathematical calculation of the  
7 benefit?

8 You say, okay, it's not 10 to the minus 5. Maybe it's  
9 10 to the minus 4, 10 to the minus 3, and that's counteracted  
10 by the beneficial effects.

11 Is that purely subjective or is there some mathematical  
12 calculation?

13 THE WITNESS: I haven't seen a calculation that would  
14 say we can fine tune it to 10 to the minus 4 or 2 times 10 to  
15 the minus 4 or 5 times 10 to the minus 5. I haven't seen any  
16 type of calculation like that.

17 Q. BY MR. METZGER: Well, Dr. Melnick, let me ask  
18 you --

19 MR. KENNEDY: Objection. The witness hasn't had a  
20 chance to finish his answer yet.

21 THE COURT: Yes.

22 Finish your answer.

23 So you haven't seen any mathematical -- is there any  
24 way to calculate human satisfaction?

25 THE WITNESS: Yes. That is possible by a method which  
26 we'll talk about a little bit later called BRAFO, which is a  
27 benefit-risk assessment for foods where you can quantify the  
28 benefits or the risks if it's necessary to go to that extent.

1 THE COURT: Go ahead, Mr. Metzger.

2 Q. BY MR. METZGER: So, first of all, from your  
3 understanding, does determination of an NSRL require a  
4 quantitative risk assessment?

5 A. Yes.

6 Q. Okay.

7 A. I'm sure it's specified.

8 Q. And what are the important aspects of that that  
9 need to go into that quantitative risk assessment?

10 A. Well, first they must be of comparable  
11 scientific validity to be evidence of standards which led to  
12 the listing of the chemical.

13 Q. And by that you mean the listing of acrylamide  
14 as a chemical known to the State to cause cancer?

15 A. Yes, exactly.

16 Q. Okay.

17 A. In determining and performing a quantitative  
18 risk assessment, it's based on the most sensitive study which  
19 is of sufficient quality, and those exist.

20 Secondly, this is not all of the principles, but I  
21 think these are the principles which impact this discussion.

22 Assume no threshold. In other words, the response, the  
23 tumor response is linear, down to zero exposure.

24 If there is insufficient human data to do a  
25 quantitative risk assessment, then the human cancer potency  
26 estimate is derived from the animal cancer potency, which is  
27 that response versus dose and applying a body weight scaling  
28 factor.

1           The potency is expressed in a certain way. It's a  
2 value of risk relative on a per milligram/per kilogram per day  
3 exposure.

4           That's how the law, the rule, Prop 65 wants the potency  
5 to be expressed.

6           In terms of body scaling, I believe it had previously  
7 been a human body weight to animal body weight to one-third  
8 the power, but now it's raised to the one-fourth power.

9           This is a method for enabling the determination of the  
10 human cancer potency from animal cancer potency.

11           However, a pharmacokinetic adjustment may be made when  
12 available evidence can be taken into account with confidence.

13           That's why this is going to be discussed in a lot more  
14 detail later on to see whether we can consider that  
15 pharmacokinetic factor with confidence.

16           That's why I have it italicized.

17           Q.       At this point would you define for us what a  
18 pharmacokinetic adjustment or a pharmacokinetic factor or  
19 model is? What is all of that?

20           A.       This is something I was going to discuss later,  
21 but I can go into it a little bit now.

22           Q.       Give us just a quick.

23           A.       The body weight scaling is intended to take into  
24 effect two factors.

25           One is pharmacokinetics. This is how the body handles  
26 a chemical that enters. And this includes factors such as the  
27 absorption of the material, how it distributes in the body,  
28 how it's metabolized and how it's eliminated.

1           This is important for providing information on  
2 dosimetry, which is a concentration in a tissue over a certain  
3 period of time. Okay?

4           So for a pharmacokinetic adjustment, rather than using  
5 the scaling factor, the pharmacokinetic factor might be used  
6 which would be a ratio of the dosimetry in a tissue in humans  
7 relative to the dosimetry of that same compound in animals.

8           So it may not be the same as what you would obtain from  
9 a scaling factor.

10          Pharmacodynamics, which isn't really addressed at this  
11 point because we don't have information, is how the cell  
12 responds to that active material when it is present.

13          So you have the dosimetry that says this is the  
14 concentration of, for example, glycidamide in a tissue over a  
15 certain amount of time. What's the response that we might  
16 expect relative to that.

17          That becomes the pharmacodynamics. Do animals and  
18 humans behave the same? We don't have enough information. So  
19 the scaling factor seems to be most appropriate, because it's  
20 scales for factors, physiological differences.

21          Q.       Thank you for explaining that.

22          So now let's start with the reduction or mitigation of  
23 acrylamide in coffee.

24          Could you tell -- before we get into the individual  
25 studies, could you give us kind of an overview of the  
26 different -- how these different technologies within this  
27 puzzle of reducing acrylamide in coffee.

28          A.       I'm sorry. How they --

1 Q. Just the overview of the different types of  
2 technologies.

3 MR. KENNEDY: Object. Narrative answer.

4 THE COURT: Overruled.

5 THE WITNESS: Some are physical removal and some are  
6 biological removal, by influencing, for example, the formation  
7 of precursors for acrylamide in the tissue of the bean or  
8 plant or whatever.

9 Q. BY MR. METZGER: All right. And have you  
10 prepared a series of slides which illustrate the different  
11 technologies and the results?

12 A. I've prepared a series of slides, yes, on that.

13 Q. Okay. And the first one here is acrylamide  
14 levels in Arabica versus Robusta roasted coffees.

15 And what did you find regarding the differences for  
16 acrylamide in Arabica versus Robusta?

17 A. There are numerous articles that have looked at  
18 these two strains of coffee, and it is rather consistent that  
19 the Arabica has a lower level than the Robusta.

20 These are two publications which show that type of  
21 difference. You can see in the Lantz paper it was 35 percent  
22 reduced in Arabica, and in the Bagdonaite paper it was  
23 47 percent.

24 The plus or minus is the standard deviation, so it  
25 gives you the sense there is a deviation within measurements.  
26 But in spite of that, these differences are significant and  
27 are reflective of the difference between the two strains, that  
28 Arabica in almost all cases that I've seen where the

1 comparisons were made roasting to the same level was lower in  
2 its acrylamide levels.

3 Q. You earlier mentioned some technologies that  
4 prevent the formation of the precursors.

5 Could you tell us what that's about?

6 A. Okay. This is shown in the next slide. It's  
7 going to require a little bit of explanation on some of this.

8 First of all, the precursors for acrylamide -- there we  
9 go -- it's asparagine reacting with reducing sugars, glucose  
10 and fructose, with the application of heat in the Maillard  
11 reaction forming acrylamide at the very bottom.

12 So the approaches that are used -- this is now in the  
13 biological sense -- is how can you lower the amount of  
14 precursor compound, as I mentioned, asparagine and glucose and  
15 fructose.

16 The use of an enzyme called asparaginase can reduce  
17 asparagine in plants, tissues substantially.

18 If you reduce asparagine, you get less acrylamide.

19 I will be showing this slide as a typical example where  
20 it shows that treatment to remove asparagine pre-roasting can  
21 reduce acrylamide levels by somewhere in the range of 70 to  
22 90 percent.

23 So one approach is treatment with enzymes to remove the  
24 precursor.

25 Now, as I mentioned in 2006 I was a consultant for the  
26 State Attorney General, and this related to reducing  
27 acrylamide in potato chips and french fries.

28 And that case settled because there was an agreement

1 made --

2 MR. KENNEDY: Objection, your Honor. Lack of  
3 foundation that he knows why a case settled.

4 THE COURT: Overruled.

5 THE WITNESS: It was settled because there was some  
6 agreement to reduce the levels. I don't know the full  
7 details, but there was agreement to reduce the levels of  
8 acrylamide in potato products.

9 MR. KENNEDY: Move to strike the answer.

10 THE COURT: Let me ask you this.

11 To your knowledge, has any coffee manufacturer  
12 attempted this process of reducing the amount of asparagine in  
13 coffee?

14 THE WITNESS: Yes.

15 THE COURT: And who that is?

16 THE WITNESS: We'll be getting into some of that very  
17 shortly.

18 THE COURT: Okay.

19 THE WITNESS: But what I want to talk about --

20 MR. KENNEDY: Your Honor, this is just a narrative at  
21 this point.

22 THE COURT: Overruled.

23 THE WITNESS: What I really want to talk about is the  
24 development that the potato industry made in terms of trying  
25 to reduce acrylamide.

26 THE COURT: No.

27 What you want to talk about is not relevant.

28 Mr. Metzger, please move on.

1 Q. BY MR. METZGER: We'll get to the potato  
2 industry in just a moment, Dr. Melnick.

3 THE COURT: We have enough trouble with coffee. I  
4 don't want to get into potatoes and potato chips.

5 THE WITNESS: Part of my reason for doing this is to  
6 show --

7 MR. KENNEDY: Objection. There is no question pending.  
8 He's trying to earn his money, I know.

9 THE COURT: Dr. Melnick, please listen to the question.  
10 Mr. Metzger is a well-experienced attorney. He knows how to  
11 ask questions.

12 MR. METZGER: Thank you, your Honor.

13 Q. All right. Regarding the formation and  
14 enzymatic mitigation of acrylamide that you have on this flow  
15 chart or whatever one calls it, is there anything else that  
16 you would like to inform the Court regarding the use of  
17 enzymes to reduce acrylamide in coffee?

18 You mentioned asparaginase. Anything else?

19 A. Okay. There are other approaches that are  
20 available, and these approaches make use of some advances in  
21 molecular biology.

22 The enzymes that are shown in red, as well as  
23 invertase, in fact all the arrows that have a word next to  
24 them are enzymes.

25 Enzymes are proteins. Enzymes catalyze reactions.  
26 They make the rate of reaction faster.

27 What has been done is to silence some of the genes that  
28 make asparagine or silence the gene invertase in potatoes that

1 makes glucose and fructose.

2 So silencing the gene -- for example the asparagine  
3 synthetase -- is a mechanism for reducing asparagine formation  
4 of which that is a precursor for the Maillard reaction that  
5 leads to acrylamide.

6 I can try to explain a little bit, if you would like,  
7 in terms of how this is being done. But you would have to ask  
8 me the question.

9 I'm used to giving a talk rather than having a  
10 presentation where someone asks me a question.

11 THE COURT: It's a different forum. You're guided by  
12 rules of procedure, doctor.

13 All right. Mr. Metzger.

14 Q. BY MR. METZGER: All right. We'll talk about  
15 gene silencing in a moment.

16 I see there is another enzyme there which I think is --  
17 is that acrylamidase?

18 A. Yes, it is.

19 Q. And will you tell us what that is.

20 A. That is an enzyme which will break down  
21 acrylamide to acrylic acid plus ammonia. So it is also  
22 another enzyme means of reducing acrylamide once it has  
23 formed.

24 Q. So asparaginase prevents the formation of  
25 acrylamide, but once acrylamide is formed, acrylamidase gets  
26 rid of it; is that it, in essence?

27 A. Correct.

28 They are acting in different ways. One prevents the

1 formation. One removes it once it has formed.

2 Q. Okay. All right.

3 So now I would like you to tell the Court how  
4 asparaginase has been used successfully to reduce acrylamide  
5 in the potato industry.

6 MR. KENNEDY: Objection, irrelevant.

7 THE WITNESS: Okay.

8 THE COURT: Overruled.

9 You may answer.

10 THE WITNESS: Actually, it was through the gene  
11 silencing technique which I didn't describe.

12 Q. All right. So tell us about the gene silencing  
13 technique, then.

14 A. The DNA molecule codes for proteins. There's --  
15 the structure of the DNA molecule was determined in the 1950's  
16 by Watson and Crick.

17 It is a double-stranded molecule which has connections,  
18 four bases that pair with each other to make the DNA molecule.  
19 These are adenine, thymine, guanine and cytosine.

20 This is the code. The code is read by an enzyme that  
21 makes RNA.

22 RNA, then, is the message and it's called -- it  
23 synthesizes a messenger RNA. Messenger RNA is single  
24 stranded. It is read in the ribosomes, where three of these  
25 bases define what amino acid can be added on to a growing  
26 chain -- it's called a polypeptide chain -- leading to the  
27 formation of a protein.

28 Now, one technique that has been used is to create

1 what's called an inhibitor, mRNA, because the strands bind  
2 complementary to each other. Like I mentioned, adenine,  
3 thymine, guanine and cytosine and guanine.

4 So now you have the same structures on a single strand  
5 of RNA.

6 They develop constructs in which a short chain RNA  
7 binds to the actual messenger RNA and that gets cleaved. So  
8 the enzyme never gets synthesized. That's one technique.

9 The other is to take the gene out, modify it, put it  
10 back in such that it is no longer active.

11 That has been done by the potato industry, and I might  
12 say it's been very successful.

13 Q. And what happens when you inactivate that  
14 invertase gene? What does that do?

15 A. It prevents -- to an extent on the conversion of  
16 sucrose to glucose and fructose, but they have also  
17 inactivated asparagine synthetase, so that prevents the  
18 formation of the asparagine which is also a precursor for  
19 acrylamide.

20 Q. Okay. Well, what has the potato industry done?  
21 What is the status of that in terms of regulation?

22 MR. KENNEDY: Objection to the demonstrative and lack  
23 of foundation.

24 We're talking about a press release from a potato  
25 company here. No showing that this is something that real  
26 scientists rely for purposes of 801(b).

27 THE COURT: Overruled.

28 THE WITNESS: The patents were developed in the 2000,

1 2009 for reducing acrylamide.

2 In February of this year, the FDA and EPA gave  
3 clearance for growing and selling potatoes with this gene  
4 alteration in the United States.

5 Part of the reason that they're also excited about it  
6 is because sensory evaluation said it was indistinguishable  
7 from heat-processed products.

8 So they have a product which, if I can read, says there  
9 is a reduction in the chemical asparagine and the reduced  
10 asparagine shows that the levels of acrylamide can be reduced  
11 up to 90 percent in potatoes that are cooked at high  
12 temperatures.

13 So there has been success by this type of approach. It  
14 does take years. It's not something that can be done  
15 overnight. But this goes back to -- I was mentioning in 2006  
16 the potato industry agreed to make a -- work on mitigation.  
17 And they developed techniques that are now enabling potatoes  
18 to be grown which will have much lower acrylamide levels than  
19 conventional potatoes.

20 MR. KENNEDY: Move to strike. No foundation that he  
21 knows what causes excitement in the potato industry or any of  
22 the details as to what they were doing.

23 THE COURT: Overruled.

24 Let's get back to coffee.

25 MR. METZGER: Yes, your Honor.

26 Q. All right. So now let's talk about asparaginase  
27 treatment for coffee which I think his Honor asked you about  
28 earlier.

1           And what has been done regarding that?

2           MR. KENNEDY: Object, narrative answer.

3           THE COURT: Overruled.

4           THE WITNESS: Okay. This is work done by Novozymes,  
5 who have produced the asparaginase.

6           And they have demonstrated that treatment of coffee  
7 pre-roasting can reduce acrylamide levels, and You can see it  
8 reduces asparagine levels.

9           Asparagine are the graph, block graphs. Acrylamide is  
10 the line values.

11           And the acrylamide, you can notice, decreases from  
12 800 micrograms per kilogram down to approximately  
13 200 micrograms per kilogram. In other words, a 75 percent  
14 reduction.

15           So this is one example. Others have reported on the  
16 use of asparaginase as a treatment. This is one example just  
17 showing what the data looked like.

18           Q.       Okay. And this is in coffee.

19           Is this roasted coffee, brewed coffee or what?

20           A.       This is in -- this is in roasted coffee. These  
21 are micrograms per kilograms, so it's in the roasted coffee,  
22 but the treatment is prior to roasting.

23           Q.       Okay. So would you explain to the judge how  
24 this works, how you get the asparagine in there?

25           A.       Well, they steam and soak the beans in the  
26 presence of an enzyme. I believe it's done at approximately  
27 60 degrees centigrade, which may seem high but the enzyme that  
28 they used is active.

1           They've determined that it was peak activity at that  
2 temperature. They've also worked out what is the optimal PH  
3 for the enzyme, so you work through these kinds of conditions.

4           It breaks down the asparagine, and then the fluids are  
5 allowed to reinfuse into the beans. They are dried and then  
6 the beans are roasted.

7           Q.       Okay.

8           THE COURT: Is this during the roasting process or the  
9 brewing process?

10          THE WITNESS: Pardon me?

11          It's done prior to roasting, and this is the level in  
12 the roasted coffee.

13          THE COURT: Has any manufacturer attempted this  
14 process?

15          THE WITNESS: Yes.

16          THE COURT: And who is that?

17          THE WITNESS: Novozymes worked with a company in  
18 Germany to produce -- I believe it was 200 tons of treated  
19 coffee that had been treated with asparaginase.

20          THE COURT: Is this the coffee that is distributed to  
21 the public?

22          THE WITNESS: It was made available, yes. It was sold  
23 to the market.

24          THE COURT: Sold to the market.

25          And what is the success rate in selling this to the  
26 market?

27          THE WITNESS: I haven't seen that type of information.

28          THE COURT: What percentage of that market in Germany

1 does this company enjoy?

2 THE WITNESS: For its total production, I don't know.  
3 It's just -- it's a demonstration of a scale-up to an  
4 industrial level as opposed to a strict laboratory  
5 demonstration that they can do it.

6 THE COURT: That's what I'm trying to find out, how  
7 they do it? How much did they do of what they did?

8 THE WITNESS: I don't know what their present  
9 production levels are.

10 THE COURT: What year did they commence this  
11 production?

12 THE WITNESS: What year?

13 I believe it was around five years ago, four years ago.  
14 But I would have to look back to the records to see. I don't  
15 have that off to top of my head.

16 THE COURT: Are they still doing that today?

17 THE WITNESS: That I don't know.

18 THE COURT: Has it been accepted by the consuming  
19 public?

20 THE WITNESS: I haven't seen information to that  
21 effect.

22 THE COURT: All right. Thank you.

23 Mr. Metzger?

24 MR. KENNEDY: Move to strike.

25 Total lack of foundation.

26 May I voir dire on this?

27 THE COURT: Let Mr. Metzger finish the examination.

28 Q. BY MR. METZGER: Dr. Melnick, have you seen any

1 information that any of those 200 tons of coffee,  
2 acrylamide-reduced coffee, that was prepared by that company  
3 in Europe, that any of that was not accepted by the public and  
4 returned?

5 A. No, I haven't seen that either.

6 THE COURT: When you say 200 tons, what percentage of  
7 the German coffee market is that?

8 THE WITNESS: I don't know what percentage. But I  
9 don't think they would --

10 THE COURT: How many tons of coffee are consumed by the  
11 American public?

12 THE WITNESS: Oh, it's probably hundreds of thousands.

13 THE COURT: Hundreds of thousands of tons?

14 THE WITNESS: Yes.

15 THE COURT: Thank you.

16 Mr. Metzger?

17 Q. BY MR. METZGER: All right. Dr. Melnick, could  
18 you explain to the Court, perhaps using the next graph that  
19 they prepared, how the acrylamide is formed and degrades in  
20 the process of roasting coffee.

21 A. Well, as I mentioned, it's formed by the  
22 reaction of asparagine with the reducing sugars, glucose or  
23 fructose.

24 And this is a typical graph showing the formation of it  
25 on a time scale. I know this type of information has been  
26 presented before.

27 Q. What is the significance to you about this?

28 A. So what you see on the first part is the rise

1 that's occurring between 50 and maybe 110 seconds under this  
2 condition. It's for a medium roast.

3 It reaches a peak, after which the rate of its  
4 degradation or loss increases such that it comes -- reduces  
5 down to approximately 20 to 30 percent remaining in the final  
6 product.

7 The reason I show this is because this, then, provides  
8 information on opportunities for removing acrylamide when you  
9 know what the apparent -- that there is a formation and a  
10 degradation reaction occurring.

11 Some of the studies subsequent show how they've made  
12 use of this type of information to reduce acrylamide levels.

13 Q. Okay. So have you read articles in the  
14 peer-reviewed literature regarding the effect of roast time on  
15 acrylamide levels in coffee?

16 A. In the next slide. This is from the  
17 confidential report.

18 Q. Oh, so this is not from the peer-reviewed  
19 literature. This is from a confidential internal report  
20 prepared by one of the coffee companies?

21 THE COURT: Can we go back to the last slide for a  
22 moment.

23 What's your understanding as to how much time coffee is  
24 processed in the general coffee market?

25 In other words, what do most manufacturers do? How  
26 long do they roast?

27 THE WITNESS: It varies substantially depending on --  
28 it's time-temperature relationships.

1           Actually in the next slide we can look at four  
2 different times of roasting times.

3           THE COURT: Is there a difference in terms of taste as  
4 far as the consumer reaction or the consumer acceptability  
5 with coffee having different roasting times?

6           THE WITNESS: Definitely. Because some people like  
7 light-roasted coffee. Some people like medium-roasted coffee.  
8 Some people like dark-roasted coffee.

9           So there is a preference. And as I'll show later,  
10 dark-roasted coffee, because you're continuing down that  
11 chart, has lower acrylamide levels than medium- or  
12 light-roasted coffee.

13           But there are preferences among coffee consumers for  
14 different degrees of roast.

15           THE COURT: All right. Thank you.

16           MR. METZGER: All right.

17           Q.       So regarding the report from Kraft in 2007 that  
18 you reviewed, what did you take away from that confidential  
19 report that Kraft did?

20           A.       So in this chart they have roasted coffee beans  
21 for different amounts of time, one and half minutes, two and a  
22 half minutes, five minutes or eight minutes. But they show  
23 with that dotted line -- the vertical dotted lines where the  
24 beans were all at the same roast color.

25           And in this what you can see, if I can find a  
26 pointer -- it may be hard to see.

27           For example, on the one-and-a-half minute, that dotted  
28 line is crossing at approximately 350 micrograms per kilogram.

1           If you can go up to where that dotted line crosses to  
2 the right -- you went too far. Just a little bit over to the  
3 right. To the right. Right there.

4           The dotted line is showing with a roast of  
5 one-and-a-half minutes achieving the same roast color, the  
6 acrylamide level is approximately 150.

7           If you then follow where that dotted line crosses the  
8 descending part of the graph, you can see that it constantly  
9 decreases.

10          In fact, by the time you get up to eight minutes or  
11 five minutes, you've reduced the amount of acrylamide by  
12 approximately 50 percent.

13          So you can see that Kraft acknowledged there was a  
14 decrease in acrylamide levels with longer roasting times even  
15 at the same color.

16          Q.       So how do you or how did Kraft get a decrease in  
17 acrylamide when roasting to the same roast color? How did  
18 they do that?

19          A.       It appears that with the increasing roasting  
20 time during the descending phase particularly causes a  
21 decrease in acrylamide levels.

22          So I showed you a typical graph, the up and down. Now  
23 they're manipulating, looking what happens to acrylamide as  
24 you start to change some of the process conditions.

25          Q.       Well, how did they get the same roast color with  
26 a longer time with lower acrylamide? What else was changed?

27          A.       The effect would be due to the heat. There's  
28 differences in temperature to enable the longer time to

1 produce the same roast color.

2 Q. So a lower temperature with a longer roast time  
3 yielded less acrylamide?

4 A. Exactly.

5 Q. Roasted to the same color?

6 A. Exactly.

7 Q. All right. Did any of the articles that you  
8 reviewed use standard roast profiles to see how they varied  
9 and the resultant acrylamide?

10 A. This is an example of that where --

11 Q. Is that Xu, 2016?

12 A. Yes, it is.

13 And what I've tried to present are two of the different  
14 programs for roasting where you can see there's temperature  
15 changes for different intervals of time between program 1 and  
16 program 2.

17 And the net effect is that by reducing the heating time  
18 of the first two stages but increasing the time of the later  
19 two stages, they were able to show a reduction in acrylamide  
20 levels in coffee, in the roasted coffee.

21 Q. So there was a 39 percent reduction of  
22 acrylamide from roast program 1 in comparison to roast program  
23 number 2, correct?

24 A. That's correct.

25 Q. Was roast program number 1 a standard roast  
26 profile that was used in industry as opposed to just some  
27 experimental program?

28 A. It's defined as a traditional coffee roast

1 program.

2 Q. All right. So what do you conclude from this  
3 study?

4 A. Well, one approach to reduce acrylamide would be  
5 to focus on the time of the later stages to reduce acrylamide.  
6 To extend the later stages does have an effect on reducing  
7 acrylamide.

8 Q. Okay. And have you reviewed studies regarding  
9 the effect of the degree of roast on the formation or the  
10 level of acrylamide in coffee?

11 A. Yes, I have.

12 I think I mentioned this to his Honor a couple minutes  
13 ago, but this a slide that, in fact, will show that type of a  
14 difference.

15 Q. All right.

16 A. Between light, medium and dark roasted coffees  
17 for both Robusta and Arabica.

18 Q. And is this data you have here from the Alves  
19 2010 study?

20 A. Yes, that's where I obtained this data.

21 Q. And would you explain what you observed from  
22 this study.

23 A. So if you focus first on just Robusta coffee,  
24 you can see the differences between light, medium and dark,  
25 that the medium is 67 percent lower in acrylamide compared to  
26 the light, and the dark is 72 percent lower in acrylamide than  
27 the light.

28 Similarly for the Arabica, the medium-roasted coffee

1 was 77 lower in acrylamide than the light and the dark was  
2 83 percent lower than the light.

3 So, in essence, the degree of roasting, as you go from  
4 light, medium to dark -- and this makes sense -- reduces the  
5 level of acrylamide. Because that's based on that first curve  
6 that I showed the formation and the destruction, elimination  
7 of acrylamide, that as you get darker the acrylamide levels  
8 decrease.

9 Now, you don't want to overcook the coffee, but this is  
10 coffee which is consumable, acceptable, palatable.

11 This is actually fairly well established in the coffee  
12 industry.

13 Q. Okay. Have you also reviewed articles regarding  
14 or for that matter industry studies regarding the effect of  
15 pressure on acrylamide formation?

16 A. Yes.

17 Did you skip a slide? Okay.

18 Q. And this study, this says in the bottom  
19 left-hand corner, "Kraft, 2006." Is this a Kraft confidential  
20 report you reviewed for you this case?

21 A. Yes. These data were obtained from a Kraft  
22 confidential report.

23 And what I have done is tried to provide the essence of  
24 their study on the steaming and pressure effects on acrylamide  
25 levels in coffee that had been roasted for 120 seconds to  
26 different color levels.

27 And what you can see here is that with steaming there  
28 is a reduction in acrylamide. This is all Robusta coffee.

1           As they increase the pressure from atmospheric 2.7, 3.7  
2 and 4.7 bar -- one bar is the equivalent of one atmospheric  
3 pressure or approximately 14 and a half pounds per square  
4 inch -- that it was effective in reducing acrylamide levels  
5 such that the steam that the high pressure provided, the  
6 percentage decreased compared Robusta was 47 percent lower.

7           The 16 would be the lighter color.

8           The others showed 36 and 30 percent reductions.

9           So it was showing approximately a 30 to 45 percent  
10 reduction by applying steam and pressure during the roasting  
11 process.

12          Q.       And has vacuum roasting been evaluated for its  
13 effect on reducing acrylamide in roasted coffee?

14          A.       Yes, it has.

15          This is a paper by -- I believe it's pronounced Anese.

16          Q.       Anese, 2014?

17          A.       Yes.

18          Q.       All right.

19          A.       And what they are comparing is conventional  
20 roast at 200 degrees centigrade. What they call combined is  
21 ten minutes of conventional roast followed by a vacuum roast,  
22 as well as then vacuum roasting at the same temperature,  
23 200 degrees, under a vacuum.

24          And what they're showing is that where you're seeing  
25 high levels of acrylamide, if you apply the vacuum early on,  
26 this will remove the acrylamide by approximately 15 percent.

27          And one thing I want to point out is that on the  
28 F minutes -- those aren't minutes. That's what's called a

1 thermal effect that they achieved within the coffee bean.

2 Under vacuum roasting, if you can read the boxes that I  
3 highlighted, at the 3.8 F minutes was actually a 15-minute  
4 roasting time. So it was -- they were roasted not for just  
5 four minutes.

6 And you can see as you increase the roasting time, they  
7 start to approach each other, the vacuum as well as the  
8 conventional or the combined.

9 And the conclusion from this is that for people who  
10 appreciate light-roasted coffee -- and this is true in  
11 Northern Europe and in many places in America -- where I just  
12 showed the information that light roasted has the higher  
13 levels of acrylamide, that it's possible to reduce the  
14 acrylamide levels for light-roasted coffee by applying a  
15 vacuum when the acrylamide levels are at their highest levels.

16 Q. And what was the percentage reduction in  
17 acrylamide that was achieved in this study using vacuum  
18 roasting?

19 A. It was approximately 50 percent.

20 Q. 5-0?

21 A. Yes.

22 Q. Oh, okay.

23 I wasn't sure if you said 15 or 50?

24 A. It was 50.

25 Q. F-I-F-T-Y. I got it.

26 Just from using vacuum roasting?

27 A. Yes.

28 Q. All right. Are there other Kraft confidential

1 studies that you reviewed that provided yet other means of  
2 reducing acrylamide in roasted coffee?

3 A. All right. So these are examples of roasting  
4 processes, now, if we consider post-roasting, the events to  
5 reduce acrylamide.

6 And in this case in the Kraft studies they reported on  
7 the reduction during heat curing of coffee at -- either under  
8 nitrogen or in an air environment at temperatures of  
9 40 degrees, 70 degrees and 100 degrees centigrade.

10 Q. What is heat curing. Can you explain that?

11 A. Well, the beans have been roasted. They're now  
12 applying another heat treatment on the beans to see if that  
13 would be effective in reducing acrylamide levels.

14 And the heat, 40 degrees centigrade, isn't particularly  
15 high. It's a little higher than body temperature, but  
16 70 degrees and 100 degrees.

17 What you can see is at 100 degrees and 70 degrees were  
18 effective in reducing acrylamide levels by applying this  
19 relatively mild heat treatment on the roasted coffee beans.

20 Q. What was the reduction of acrylamide in the  
21 roasted coffee using this post-roasting heat curing process  
22 that Kraft determined?

23 A. Well, the graph is showing that the reduction  
24 went to -- from approximately 450, 425 to maybe 100  
25 micrograms.

26 So that would indicate a decrease of approximately  
27 75 percent.

28 But they also did some taste testing. And that's

1 what's shown on the following graph, which shows where the  
2 acceptable level was found in their taste testing studies to  
3 be up to approximately a 45 percent reduction of acrylamide.

4 So if you over-reduce, obviously the tasting is  
5 decreasing in its value. But it is acceptable, from their  
6 determination, up to approximately 45, 50 percent reduction in  
7 acrylamide.

8 Q. By using this post-roast heat curing process?

9 A. Yes.

10 Q. All right. And in this process could you  
11 explain what the difference is, whether you do the heat curing  
12 in the ambient air or whether you use a nitrogen atmosphere?

13 Explain that, please.

14 A. Well, they did it under both conditions, and  
15 evidently there's not a big difference between the nitrogen  
16 atmosphere versus ambient air.

17 I imagine it's probably done under nitrogen to prevent  
18 any oxidative damage they might have anticipated. But it  
19 doesn't seem that that really has much of an influence since  
20 the roasting at 70 degrees or a 100 degrees wasn't that  
21 different between nitrogen and ambient air.

22 Q. You mentioned oxidative damage and using  
23 nitrogen in a nitrogen atmosphere to prevent that.

24 Would you explain that to the Court, what oxidative  
25 damage is, first.

26 A. Yes.

27 Within foods, coffee, there are fatty acids, lipids,  
28 which include fatty acids. Triglyceride is a lipid with three

1 fatty acid chains on them.

2 And when there is unsaturated fatty acids, this means  
3 that there's double bonds as opposed to single bonds  
4 connecting these carbon chains.

5 These carbon chains run 16, 18 carbons in length.

6 Where there are unsaturated bonds -- these would be  
7 double bonds -- these are prone to attack by oxygen.

8 And with oxidative damage you can start to form  
9 products that would be undesirable -- aldehydes, acids, et  
10 cetera -- that the nitrogen environment would prevent because  
11 it would replace the oxygen which would have allowed the  
12 oxidative damage to occur.

13 Q. And in this study, even just using an  
14 environment of air, ambient air, they were still able to  
15 achieve this acrylamide reduction through this post-roast heat  
16 curing process?

17 A. Yes.

18 Q. All right. Let's talk a little bit about  
19 decaffeination.

20 How is decaffeination done?

21 A. Well, it used to be done by adding solvents.  
22 One solvent in particular that had been used was methylene  
23 chloride.

24 However, when methylene chloride was demonstrated to be  
25 carcinogenic, the industry looked towards alternative ways of  
26 decaffeinating.

27 One that became particularly popular was use of  
28 supercriticals, carbon dioxide extraction.

1 Q. What is that?

2 A. Carbon dioxide, you know, is a gas. If you put  
3 it under pressure, it will have fluid-like properties.

4 So it can penetrate and act in a fluid-like manner to  
5 move materials in or out.

6 Supercritical CO2 is used in numerous other processes,  
7 but it has been used now in the coffee industry for removing  
8 caffeine by extracting it into this stream, this supercritical  
9 CO2.

10 It's particularly done under particular conditions  
11 which they work out which would be optimized for the compound  
12 that they are trying to extract.

13 Q. Excuse me. Is supercritical extraction used  
14 today in the coffee industry to decaffeinate coffee?

15 A. Oh, yes, it is.

16 Q. Has supercritical extraction been investigated  
17 as a means of reducing acrylamide in coffee?

18 A. Yes. That's what's shown in this graph.

19 Q. And is this the Banchemo 2013 study?

20 A. Correct.

21 I might point out that Banchemo had a co-author who was  
22 from Lavazza, a coffee manufacturing company in Italy.

23 So if we consider back to that graph in terms of where  
24 the acrylamide is formed early on in the process, what they  
25 did in this case was to optimize a condition for acrylamide  
26 formation.

27 So they pre-roasted at 151 degrees for 20 minutes and  
28 then applied the supercritical CO2 extraction.

1           And what you're looking at, then, is the percentage of  
2 acrylamide which was extracted as a function of the CO<sub>2</sub>.

3           And the reason for this at that time was to remove the  
4 acrylamide when it is at a high level prior to the formation  
5 of the majority of these aromatic and taste compounds which  
6 form at the higher temperatures.

7           So they worked out a condition in which the acrylamide  
8 could be extracted prior to the real final roasting of coffee  
9 beans.

10          Q.       And what was the effect or the percentage of  
11 reduction of acrylamide that Banchero found using  
12 supercritical carbon dioxide extraction?

13          A.       Well, in this case, as you can see, the graph  
14 goes up to 80 percent at the 100 degrees, 200 bar. That's the  
15 pressure, 200 atmospheres.

16          Q.       And the last question before the break. I can  
17 see -- is this a technology that can be implemented by  
18 companies that are already using supercritical extraction to  
19 decaffeinate their coffee?

20          MR. KENNEDY: Lack of foundation.

21          THE COURT: Overruled.

22          THE WITNESS: Definitely.

23                They have the -- places to have the facility. Because  
24 if they're extracting caffeine, they could easily apply it  
25 towards the removal of acrylamide. That's the purpose of them  
26 conducting this kind of experiment.

27          THE COURT: All right. Thank you. We'll stop at this  
28 point, and we'll have the morning recess.

1 I'll be off the record in this case for 15 minutes.

2 (Recess.)

3 THE COURT: All right. Back on the trial, CERT versus  
4 Starbucks.

5 Dr. Melnick is on the stand. Mr. Metzger was  
6 questioning him.

7 Counsel, you may proceed.

8 MR. METZGER: Thank you, your Honor.

9 Q. Dr. Melnick, I would like to go back to slide  
10 12, which was the effect of roast time on acrylamide levels,  
11 the Kraft 2007 study.

12 And did you read Dr. Ristenpart's testimony that beans  
13 that are roasted the same color have the same acrylamide  
14 level?

15 A. I did see that in his testimony, yes.

16 Q. And in your opinion is Dr. Ristenpart correct in  
17 that testimony?

18 A. Well, not according to these data. Because  
19 these data demonstrate that the acrylamide levels decrease  
20 with longer roast times when roasted to the same color.

21 Q. Okay. And would you -- I would like to go to  
22 the latest slide, number 17, the heat curing treatment.

23 Did you read Dr. Ristenpart's testimony that heat  
24 curing occurs at high temperatures, around 120 to 160 degrees  
25 centigrade, and that sensory testing of the cured coffee was  
26 invariably negative because it creates a baked flavor?

27 A. Yes, I did read that.

28 Q. And in your opinion is Dr. Ristenpart's

1 testimony on that point correct?

2 A. No. Because as shown in the slide, the curing  
3 was effective at 70 degrees as well as 100 degrees centigrade,  
4 not 120 to 160 degrees.

5 This is not a baking temperature. And therefore, I  
6 believe his statements are inaccurate, unless he has data  
7 showing that curing occurs at 150 degrees.

8 But the data that Kraft has provided shows that curing  
9 is effective at lower temperatures.

10 Q. All right. So now I would like to ask you about  
11 a chemical called cysteine.

12 Can you tell us, first, what that is.

13 A. Well, cysteine is an amino acid. So is lysine  
14 and arginine, which are shown in this slide. But cysteine is  
15 one of the amino acids involved in protein synthesis. So all  
16 of our bodies contain cysteine, and it's part of our protein.

17 Q. Did you read this study by Narita in 2014  
18 regarding the use of cysteine as a use of reducing acrylamide  
19 in coffee?

20 A. Yes, I did.

21 Q. Could you tell us what that involved.

22 A. This was a study for ready to drink coffee.

23 Now, what they did was they tried adding these three  
24 different amino acids separately to the canned coffee and  
25 examined for its effect on acrylamide levels when it was  
26 heated to 120 degrees centigrade for six minutes.

27 So this is brewed coffee.

28 Now, let me explain just quickly why this is effective.

1           Acrylamide has a carbon called a double bond next to a  
2 carbonyl group.

3           Similarly to the way acrylamide is detoxified in the  
4 body by glutathione, a compound like acrylamide which is an  
5 electrophile, meaning it wants electrons. It's looking for  
6 electrons; it likes them. That's what electrophile means.

7           Cysteine has a sulfhydryl group, an SH group, and it's  
8 a donator.

9           What happens is cysteine will react with acrylamide,  
10 form a covalent bond. And by forming that covalent bond, when  
11 the acrylamide cysteine complex gets ingested, it cannot  
12 undergo oxidation to glycidamide, the epoxide that you want to  
13 try to avoid.

14           Q.       And why are you trying to avoid glycidamide?

15           A.       Glycidamide -- we'll talk about later -- is a  
16 DNA-reactive compound which is a mutagen which causes  
17 chromosomal damage and is linked to the carcinogenicity of  
18 acrylamide.

19           Q.       In this study by Narita, what did they do?

20           A.       So they added various amounts of these three  
21 different amino acids to the canned coffee, heated it to  
22 120 degrees for six minutes, and followed the effect of the  
23 additive on acrylamide levels.

24           As you can see, they got, with cysteine, over  
25 90 percent reduction in acrylamide levels, which is quite, in  
26 my view, impressive.

27           Q.       All right. And this is in coffee, roasted  
28 ground coffee in a can?

1           A.       Yeah, it's brewed. It's ready to drink.

2           Q.       So the amino acids were put in the can of the  
3 roasted ground coffee and then it was just brewed? That's all  
4 that was done?

5           A.       Correct.

6           Q.       All right. So now let's talk about storage.

7           And the first thing I would like you to do is explain  
8 to Judge Berle the difference between storing coffee in the  
9 open air and storing coffee in sealed bags or cans.

10          A.       Well, as I mentioned earlier before the break,  
11 that coffee can undergo lipid oxidation and create all flavors  
12 as a result of that oxidation.

13                Oxidation, as the name implies, is oxygen involved in  
14 reacting with the double bonds of the lipid, causing it to  
15 undergo various breakages and form new compounds.

16                So the difference between how you store the coffee is  
17 critical for maintaining high quality. Because if you store  
18 it with access to atmospheric oxygen, it will undergo staling  
19 in relationship to the oxidation of the lipids which are  
20 present.

21          Q.       How quickly?

22          A.       Well, I would -- one week, two weeks in open  
23 air, depending temperature, room temperature, it's not  
24 something which people tend to enjoy.

25          Q.       Okay. And what about storing coffee in sealed  
26 bags or cans? How does that differ?

27          A.       Okay. Well, there are companies, Illy in  
28 particular -- I'm familiar with their cans -- that they

1 provide a pressurized inert gas with a very tight seal on  
2 them.

3 And they indicate that that coffee is stable for up to  
4 two years.

5 Q. So that coffee can sit in that can and doesn't  
6 go stale and then you can open that can and brew that coffee  
7 up to two years later?

8 A. Well, that's what their website indicates.

9 However, they also say once opened, the coffee will go  
10 bad within one or two weeks.

11 Starbucks also lists their coffee as being stable for  
12 up to 60 weeks.

13 I know from personal experience, because I've drunk  
14 Illy coffee, where we -- where I've seen the date stamp on the  
15 bottom. Never did I wait to the end of the date stamp to try  
16 the coffee, but I've tasted it personally after it's been  
17 stored for three months. This is in relationship to how I  
18 travel back and forth between two locations.

19 When we order something, we don't use it up while we're  
20 there. And it's -- in my own experience it's still as good as  
21 it was if I opened a fresh can.

22 So the companies indicate that their coffee is stable  
23 because of their specialized means of storing the can, and a  
24 critical part of that is avoiding opening to the oxygen in the  
25 air.

26 Q. All right. So I'm gathering that storing  
27 acrylamide -- I'm sorry -- that storing coffee in the open air  
28 is not a viable means of getting rid of acrylamide, letting it

1 evaporate in the open air, because you're going to have foul  
2 coffee; is that right?

3 A. That sounds pretty close to what you wouldn't  
4 want to do.

5 Q. All right. But have there been studies that  
6 have researched storing coffee, after it's been roasted, in  
7 sealed bags or other sealed containers as a means of reducing  
8 acrylamide?

9 A. I haven't seen that data, per se.

10 All I have seen is what Illy and Starbucks say about  
11 their storing of coffee and my own experience.

12 This particular slide --

13 Q. All right. Let's look at -- what is this?

14 This is the Baum 2008 study?

15 A. Yes.

16 Q. And this about storage of coffee?

17 A. Yes. This is what happens to acrylamide during  
18 storage.

19 Q. And when you say storage, is this storage in  
20 sealed situations or not?

21 A. Yes.

22 Q. Okay. All right.

23 So in 2008 -- that's almost ten years ago -- what did  
24 these investigators find regarding -- or what did they do to  
25 do the study regarding storage of coffee and its effect on  
26 acrylamide?

27 A. Okay. What they did was inject a radio-labeled  
28 form of acrylamide. That's what's indicated as C14

1 acrylamide. This is a label so you can identify the presence  
2 of that acrylamide and where it ends up as a consequence of  
3 storage.

4 Q. You label it so you can follow it?

5 A. Exactly.

6 Q. Okay.

7 A. Okay. So what they have done here is stored  
8 coffee at two different temperatures. One is room  
9 temperature, and one is at 37 degrees. This is the roasted  
10 coffee with injected radio-labeled acrylamide.

11 They then followed the course of the radio-label over  
12 time, storage time, and looked to see where the label was with  
13 respect to the brew or the filter.

14 So this would be coffee, roasted coffee brewed by  
15 filtration.

16 So when you filter, the grounds remain in the filter  
17 paper and the brew comes through. They therefore followed  
18 where the radio label was going and what it --

19 Q. The radio-labeled acrylamide?

20 A. The radio-labeled acrylamide. Correct.

21 What they found was the amount in the brew -- that's  
22 what people drink -- decreases with time of storage, both at  
23 room temperature and 37 degrees storage.

24 But in the filter paper -- now, this is going to be  
25 radio-labeled. Because what's happening is that the  
26 acrylamide is binding to the matrix material in the filter and  
27 not made available into the brew.

28 So what you can see, then, is that these curves are

1 showing a decrease in the brew and an increase in the filtered  
2 paper, because it's in that matrix which is trapped by the  
3 filter paper.

4 So what is concluded from this -- and they couldn't  
5 extract it very easily with solvents -- is that this is what  
6 happens, as I mentioned, with cytosine, what happens with  
7 acrylamide in the body.

8 There are components to which acrylamide will bind and  
9 form a stable covalent bond and hence be retained in that  
10 material, which is the filtered material, that matrix  
11 material. And that's the explanation for what's happening in  
12 acrylamide in roasted coffee during storage time.

13 Q. So how long was the coffee stored in sealed  
14 containers after being roasted in this study?

15 A. Well, in this particular study it was up to  
16 approximately 50 weeks.

17 Q. All right. And when that coffee was brewed,  
18 what did that show regarding the acrylamide that ended up in  
19 the brewed coffee?

20 A. Well, in this case it was reduced by  
21 approximately 45 percent during that storage period.

22 Q. So --

23 A. And you can see the curve, so that you have  
24 different time intervals where you can follow the decrease in  
25 acrylamide in a brew.

26 What this also indicates is that acrylamide will wind  
27 up in the brew in filtered coffee unless it has bound to that  
28 matrix material.

1 Q. Okay. So is simply storing coffee for a period  
2 of time -- say 50 weeks -- in sealed containers in your  
3 opinion a viable means of reducing acrylamide in coffee?

4 A. Yes, it is a viable means. But it depends,  
5 again, on to what extent you want to see the reduction in  
6 acrylamide.

7 So, for example, even at 37 degrees at approximately  
8 15, 16 weeks, you're approaching 50 percent reduction.

9 So that it may not be necessary to even wait the full  
10 one year. This provides the information in terms of the  
11 effectiveness of removing the acrylamide in relationship to  
12 two different temperatures.

13 Q. Okay.

14 A. And it's not going to remove it all. In this  
15 study it didn't remove it all, but it does show its  
16 effectiveness in removing acrylamide from the eventual brew.

17 Q. So after about 15 weeks of storage, the  
18 resultant acrylamide concentration was reduced by about  
19 45 percent?

20 A. Let's see. Let me see if I can show you which  
21 point I'm looking at here.

22 Do you see that right in there?

23 Q. Right.

24 A. That's the data I'm looking at. That's  
25 approximately 15, 16 weeks.

26 And this is 60 percent. It's less than 60 percent.

27 So it is effective.

28 Q. All right. To do this, would industry need to

1 build new coffee roasting plants?

2 A. Not new roasting plants but maybe storage  
3 plants.

4 Q. A storage room?

5 A. Yes.

6 Q. Okay. That's it?

7 A. Yeah, that's it.

8 Q. Low tech?

9 A. Compared to what we know now about technology,  
10 that would be about as low as you can go.

11 Q. You just put it in a storage room for 16 weeks  
12 and you get rid of about 45 percent of the acrylamide?

13 A. Yeah. It is a function of the temperature, too.

14 Q. All right. Incidentally, Dr. Ristenpart  
15 testified that after roasting coffee stales quickly unless  
16 used within a week.

17 Is that testimony of his correct?

18 A. That's definitely not correct because -- I  
19 believe he got that statement from an individual, an author  
20 who writes several books. It was in the introduction to a  
21 book about coffee history which gave no information in terms  
22 of the conditions in which the coffee was stored.

23 And if it staled within a week, it was certainly not  
24 stored under an inert gas or nitrogen during that period.

25 Q. The way Illy does it?

26 A. The way Illy does it, yes.

27 Q. Okay. So now let's look at the end of the  
28 process where one actually brews coffee using a filter.

1           Have researchers investigated the use of that enzyme  
2 that you mentioned earlier, acrylamidase, to get rid of the  
3 acrylamide once it's formed?

4           A.       Right. So the early studies are preventing  
5 acrylamide formation. At this stage the acrylamide is  
6 present. So now we're considering the possibilities of  
7 removing the formed acrylamide.

8           And in this case data reported by Smucker's  
9 demonstrated that in Folgers roast coffee, by incorporating  
10 acrylamidase into the filter paper was effective in reducing  
11 acrylamide to below the detection limit.

12          And I might point out that the enzyme that they used  
13 was from bacteria Bacillus, a species.

14          And it's obviously bad bacteria, but Bacillus are --  
15 there's a number of bacteria that cause lactic acid  
16 fermentation or strains of Bacillus that conduct that. But  
17 this is the enzyme was incorporated, not the bacteria,  
18 incorporated into the paper and was effective in reducing the  
19 acrylamide levels as a consequence of its breakdown of  
20 acrylamide.

21          Q.       So incorporated into the filter paper was an  
22 enzyme, not a fungus?

23          A.       Not a fungus, no. Just an enzyme from bacteria,  
24 but it was an enzyme, yes.

25          Q.       So when Dr. Ristenpart testified that people  
26 don't want fungus in their coffee from this method, did that  
27 make any sense to you?

28          A.       It makes sense that you wouldn't want fungus,

1 but this isn't fungus. This is an enzyme from bacteria in  
2 filter paper. So in that sense it doesn't make sense.

3 Now, I should point out something which I find  
4 interesting, is that --

5 MR. KENNEDY: Objection, your Honor. He's now  
6 volunteering an answer to an unasked question.

7 THE COURT: Next question.

8 Q. BY MR. METZGER: What else did you find of  
9 interest regarding the Smucker 2015 study, doctor?

10 A. So coffee when it's filtered is warm, very hot.

11 This study was done at 80 degrees. And you might think  
12 that why would the enzyme degrade, but there are places where  
13 bacteria can grow at very high temperatures.

14 In fact, a person won a Nobel Prize for identifying an  
15 enzyme that was used in DNA identification and sequencing,  
16 isolated this enzyme from Yellowstone Park, where there was  
17 bacteria growing at very high temperatures.

18 So proteins will degrade -- undergo denaturation at  
19 typical high temperatures, but there are some that are stable.  
20 And this was obviously a stable enzyme such that it could be  
21 used when hot water is poured over the ground coffee.

22 Q. Okay. And the acrylamide reduction in this  
23 study was what?

24 A. Well, depending the amount of enzyme that was  
25 used, the fourth bar shows a 54 percent reduction. And if  
26 it's below the limit of detection, it's approaching  
27 100 percent.

28 Q. All right. And have attempts been made to

1 reduce acrylamide in instant coffee?

2 A. Yes, it has.

3 Q. And what study is that?

4 A. This is the study by Cha of 2013.

5 Q. And what did Cha do?

6 A. Okay. It wasn't the pure enzyme, but it was a  
7 cell-free extract.

8 That means they lysed the bacteria.

9 Q. L-Y-S-E-D?

10 A. Yes.

11 Q. What does that mean?

12 A. They burst it.

13 Q. Okay.

14 A. Okay. They burst the cell, and the material  
15 which they could obtain from within is the extract.

16 So it's no longer a cell that can divide. It's just  
17 the extract from the cells.

18 And you can precipitate down some of the cell debris  
19 and have an extract remaining, a liquid extract.

20 So what they did was, in this case, add different  
21 amounts of that cell-free extract to brewed instant coffee. I  
22 think he allowed it to work for various amounts of time and  
23 examined the effects on the concentration of acrylamide.

24 In this case, you can see they've added acrylamide to  
25 see its effectiveness. And it reduced the acrylamide almost  
26 100 percent with 80 microliters of their extract.

27 It's a demonstration of an effectiveness of the  
28 cell-free extract which contains acrylamidase.

1 Q. This is still acrylamidase?

2 A. Yes. In breaking down acrylamide even in  
3 instant coffee.

4 Q. And what was the percentage reduction of  
5 acrylamide in instant coffee for this study?

6 A. From this study -- it depends on the amount of  
7 enzyme and how long you incubate, but it's approaching  
8 100 percent.

9 Q. Okay. Now, Dr. Ristenpart testified that to  
10 remove acrylamide by acrylamidase that that would require a  
11 two- to four-hour treatment.

12 Was Dr. Ristenpart correct about that?

13 A. Well, from the data presented by Cha, 20 minutes  
14 will reduce it more than 80 percent.

15 So I would think he's a little off on his estimation of  
16 the time necessary.

17 Q. All right. Dr. Melnick, have you prepared a  
18 summary regarding the different acrylamide reduction  
19 technologies for coffee indicating the percentages of  
20 reduction of acrylamide?

21 A. Yes, I have. That's shown in the next slide.

22 Q. All right. So tell us what you conclude from  
23 this.

24 A. Okay. So we've walked through most of these --  
25 maybe all of them -- methods for reducing acrylamide.

26 I'm showing some of the data which indicates the  
27 percentage reduction that can be achieved by various  
28 techniques.

1           These go back to some of the slides that I showed  
2 earlier, such as the selection of the coffee bean, the  
3 asparaginase treatment, as you said near 90 percent.

4           I might point out that those authors, Navarini from  
5 Illy, and Dria is from Procter & Gamble. So it's based on  
6 patent work.

7           There are roasting techniques which show certain amount  
8 of effectiveness in removing acrylamide.

9           There's post-roasting techniques which are also  
10 effective. Some of these we discussed recently. Curing,  
11 supercritical carbon dioxide extraction or adding cysteine.  
12 Storage is also a means of effectively reducing acrylamide.  
13 And the post-brewing techniques of acrylamidase treatment.

14           Now, you can see that there are a number of techniques  
15 that have effectiveness. And in many examples you can combine  
16 multiple techniques for removing them.

17           So, for example, asparaginase treatment can reduce  
18 acrylamide effectively. If you add some additional storing,  
19 storage time, you can get even further reduction, so that an  
20 effort should be very easily accomplished by using techniques  
21 which already exist and combining some of those to reduce  
22 acrylamide, in my view, by at least 90 percent.

23           Q.       Okay. I want to note just one thing here.

24           On this table you also have altered gene expression.  
25 It says 90 percent in potatoes.

26           A.       Right.

27           Q.       Has anybody yet done the study altering the gene  
28 expression to reduce acrylamide in coffee the way it's been

1 done in potato plants?

2 A. I haven't seen any evidence of that. That's why  
3 I put -- the title of this slide was "Acrylamide in coffee,"  
4 but because there is a methodology that exists for potatoes,  
5 that methodology could be applied to coffee.

6 In fact, the Simplot people make that kind of  
7 statement, that it could be effective in coffee as well.

8 I don't know if they produced any data on that because  
9 they're focused on potatoes. But altered gene expression was  
10 where I was talking about silencing genes involved in  
11 producing precursors.

12 These techniques are relatively new within the past 20  
13 years. And these could be pursued -- could have been pursued,  
14 as well.

15 Q. So other than the altered gene expression is all  
16 of the -- are all of the studies and the techniques and the  
17 percentage reductions of acrylamide that you've summarized on  
18 the slide, are those all for coffee?

19 A. Yes. Just the gene expression was not for  
20 coffee, but all the others were data obtained from coffee  
21 analyses and treatments.

22 Q. Right.

23 And were there some studies that provide a means of  
24 reducing acrylamide in light roast coffee?

25 A. Yes.

26 The vacuum roast was effective in removing it from  
27 light roast. Light roast is one of the bigger concerns,  
28 because that has the highest acrylamide levels.

1 Q. And some of these studies, did they show  
2 effectiveness in reducing acrylamide in instant coffee?

3 A. Yes. Those were the studies done with  
4 acrylamidase, that, yes, once it's formed it still can be  
5 removed.

6 Q. And did some of the studies even show the  
7 ability to reduce the acrylamide levels in dark roast coffee?

8 A. In dark roast?

9 Well, the asparaginase treatment is selective for  
10 asparagine. So it's going to be effective regardless of the  
11 roast level because it's taking away a precursor.

12 So it would be effective at any level of roasting if  
13 you reduce the precursor compound which is required for  
14 forming the acrylamide.

15 Q. Okay. Well, let me ask you a little, then,  
16 about asparagine.

17 You've indicated it's an amino acid.

18 A. That's correct.

19 Q. Okay. Is asparagine essential for flavor  
20 formation in coffee?

21 MR. KENNEDY: Lack of foundation.

22 THE COURT: Overruled.

23 THE WITNESS: Well, one way to evaluate that is to run  
24 sensory tests on asparaginase-treated coffee, and that has  
25 been done.

26 Q. BY MR. METZGER: So now let's talk about that.

27 What studies did you find that it actually evaluated  
28 flavor or other sensorial properties of techniques used to

1 reduce acrylamide in coffee?

2 A. Okay. So this is a listing of eight examples  
3 where the impact of acrylamide reduction was evaluated for  
4 flavor and consumer acceptance.

5 For asparaginase, the Illy Company indicated that the  
6 organoleptic properties remained unaltered.

7 Stadler, who was at Nestle company, indicated that with  
8 70 percent reduction of acrylamide there was no significant  
9 impact on organoleptic properties.

10 The Xu paper examined a number of aroma compounds, and  
11 there were only minor changes that they observed when they  
12 reduced acrylamide by 84 percent.

13 So it does not appear -- it does not seem like removing  
14 asparagine is going to have a large impact on flavor.

15 I might point out that asparagine is one of 20 some odd  
16 amino acids which still can participate in the Maillard  
17 reaction. And, in particular, lysine is an amino acid which  
18 is very prone to undergo the Maillard reaction with reducing  
19 sugars.

20 So there are other amino acids that are available, and  
21 the taste-testing evaluations that have been done indicate  
22 that they're not seeing significant changes.

23 Q. What about the vacuum roasting technique.

24 Was there any sensory evaluation for that study?

25 A. Yes. Anese reported that there was no perceived  
26 difference by the assessors.

27 So as I mentioned, this would be a light-roasted coffee  
28 which you have to assess it for light-roasted coffee, not

1 dark-roasted coffee in case people have a preference for a  
2 different level of roasting.

3 Q. And what was observed by the Kraft scientists  
4 for the heat curing technique post-roast?

5 A. Well, again, now, the curing, as I mentioned,  
6 was done both under nitrogen or under atmospheric conditions  
7 of air being present.

8 Under the nitrogen it was effective in preventing the  
9 formation of all flavors. And they did do sensory evaluations  
10 for that.

11 Q. And what did Illy in its studies observe  
12 regarding the storage of roasted coffee in sealed containers?

13 A. Well, they advertise that the flavor and  
14 freshness are preserved for up to two years if the can is  
15 unopened.

16 Q. Okay. Dr. Ristenpart testified that asparagine  
17 is needed for the Maillard reaction that yields products  
18 crucial for flavor.

19 Is asparagine essential for coffee flavor?

20 A. The reduction of asparagine does not cause  
21 significant effects on flavor.

22 Q. How do you know that?

23 A. From these sensory reports.

24 But if a claim such as that is made, then do the  
25 experiment.

26 You know, I always believe in proposals. Hypotheses  
27 are valuable, but they need to be tested rather than just  
28 arbitrarily claim that you need asparagine for the flavor.

1 Q. Are the published peer-reviewed studies  
2 regarding reduction of acrylamide in coffee and the industrial  
3 confidential studies that you reviewed, are those hypotheses  
4 or do those result in conclusions from experiments?

5 MR. KENNEDY: Compound.

6 THE COURT: Overruled.

7 THE WITNESS: Well, most of them are based on some  
8 sensory evaluation.

9 Some are based on statements made in patents, and I  
10 didn't see the data from the patents to see how they did it.  
11 All I can infer is that they were either telling the truth or  
12 misleading in their patent application, and I can't  
13 distinguish between the two.

14 Q. BY MR. METZGER: All right. Regarding the  
15 patent applications, there were patent applications by what  
16 coffee companies?

17 A. Illy and Procter & Gamble.

18 Q. And what did you conclude from your analysis of  
19 those patent applications?

20 A. Well, that the statements, I believe, are  
21 accurate. But they didn't provide the actual data in the  
22 patent of their sensory tests, so I --

23 Q. You would like to see that?

24 A. I would like to see it. I believe in seeing  
25 data, both ways.

26 But the intention is to produce acrylamide-reduced  
27 coffee that is acceptable. And those patent applications were  
28 developed because they had enough evidence to file them.

1           Q.       Okay.  So, Dr. Melnick, would you tell the Court  
2 what is your overall conclusion regarding the feasibility of  
3 reducing acrylamide in coffee and still ending up with  
4 palatability?

5           A.       Well, that's what I state over here in this  
6 slide, that in my opinion it can be reduced selectively by at  
7 least 90 percent without significantly affecting sensorial  
8 properties of coffee.

9           As I indicated before, it may be best to explore  
10 combination techniques if -- for example, when I was showing  
11 some curves where it seemed like the acceptability was  
12 starting to deteriorate, so you work under the levels of  
13 acceptable and perhaps include a secondary process to reduce  
14 further without affecting palatability.

15          In my opinion this is very doable effort.

16          Q.       And would you tell the Court which of the  
17 techniques -- perhaps we could go back to the summary slide.

18          Which of these techniques can be implemented without  
19 having to tear down and rebuild coffee roasting plants or  
20 processing plants?

21          A.       Well, asparaginase doesn't require rebuilding  
22 coffee plants.

23          The supercritical CO2 extraction method, those are  
24 available for companies that are making decaffeinated coffee.

25          Storage wouldn't require tearing down any kind of  
26 facility.

27          And acrylamidase treatment is also -- or adding  
28 cysteine are very simple methodologies that could be

1 implemented for reducing coffee without much modification of  
2 any processing facility.

3 Q. All right.

4 MR. METZGER: Your Honor, would it be appropriate to  
5 take a lunch break now?

6 THE COURT: Okay. At this time we will be in recess  
7 until 1:30 this afternoon.

8 Have a pleasant lunch.

9 (At 12:00 noon, a recess was taken until 1:30 p.m.  
10 of the same day.)

11

12

13

14

15

16

17

18

19

20

21

22

23

24

25

26

27

28

1 SUPERIOR COURT OF THE STATE OF CALIFORNIA

2 FOR THE COUNTY OF LOS ANGELES

3 DEPARTMENT 323

HON. ELIHU M. BERLE, JUDGE

4

5 CERT, )  
6 )  
7 ) Plaintiff, )  
8 )  
9 ) vs. )  
10 )  
11 ) STARBUCKS CORP, ET AL., )  
12 )  
13 ) Defendants. )  
14 )  
15 )  
16 )  
17 )  
18 )  
19 )  
20 )  
21 )  
22 )  
23 )  
24 )  
25 )  
26 )  
27 )  
28 )

---

SUPERIOR COURT  
CASE NO. BC 435759  
BC 461182

12 I, DAVID A. SALYER, Official Pro Tem Reporter of the  
13 Superior Court of the State of California, for the County of  
14 Los Angeles, do hereby certify that the foregoing pages, 1  
15 through 72, inclusive, comprise a true and correct transcript  
16 of the proceedings taken in the above-entitled matter reported  
17 by me on October 2, 2017.

18 DATED: October 2, 2017.

22 \_\_\_\_\_  
23 DAVID A. SALYER, CSR, RMR, CRR  
24 Official Pro Tem Court Reporter  
25 CSR No. 4410

# **EXHIBIT “D”**

1  
2  
3  
4  
5  
6  
7  
8  
9  
10  
11  
12  
13  
14  
15  
16  
17  
18  
19  
20  
21  
22  
23  
24  
25  
26  
27  
28

SUPERIOR COURT OF THE STATE OF CALIFORNIA

FOR THE COUNTY OF LOS ANGELES

DEPARTMENT 323

HON. ELIHU M. BERLE, JUDGE

CERT,	)	
	)	
	)	PLAINTIFF,
	)	CASE NO. BC 435759
VS.	)	
	)	BC 461182
STARBUCKS CORP, ET AL.,	)	
	)	
	)	DEFENDANTS.
	)	

---

REPORTER'S TRANSCRIPT OF PROCEEDINGS

MONDAY, OCTOBER 2, 2017

**P.M. SESSION**

APPEARANCES OF COUNSEL:

FOR THE PLAINTIFFS: METZGER LAW GROUP  
 BY: RAPHAEL METZGER, ESQ.  
 AVI PARISER, ESQ.  
 401 East Ocean Boulevard  
 Suite 800  
 Long Beach, California 90802  
 (562)437-4499  
 sbrust@toxictorts.com  
 rmetzger@toxictorts.com  
 apariser@toxictorts.com

FOR THE ROASTER AND DOE DEFENDANTS:  
 MORRISON/FOERSTER  
 BY: JAMES M. SCHURZ, ESQ.  
 425 Market Street  
 San Francisco, California 94105-2482  
 (415)268-7124  
 jschurz@mofo.com

(Appearances continued on next page.)

MARK SCHWEITZER, CSR, CRR, RPR  
OFFICIAL PRO TEM COURT REPORTER  
LICENSE NO. 10514  
213-663-3494

1 APPEARANCES OF COUNSEL: (CONTINUED)

2 FOR KEURIG: SKADDEN, ARPS, SLATE, MEAGHER  
3 & FLOM, LLP  
4 BY: RAOUL D. KENNEDY, ESQ.  
5 525 University Avenue  
6 Palo Alto, California 94301  
7 (650)470-4550  
8 rkennedy@skadden.com

9 FOR HN FERNANDEZ, ET AL.:  
10 NORTON ROSE FULBRIGHT, LLP  
11 BY: JEFFREY B. MARGULIES, ESQ.  
12 LAUREN SHOOR, ESQ.  
13 555 South Flower Street  
14 41st Floor  
15 Los Angeles, California 90071  
16 (213)892-9286  
17 jmargin@nortonrosefulbright.com  
18 lshoor@nortonrosefulbright.com

19 FOR 7-ELEVEN, ET AL.: ARNOLD & PORTER KAYE SCHOLER  
20 BY: SEAN A. MCCORMICK, ESQ.  
21 One Embarcadero Center  
22 22nd Floor  
23 San Francisco, California 94111-3711  
24 (415)471-3303  
25 sean.mccormick@apks.com

26 FOR KERRY, INC.: BRYAN CAVE, LLP  
27 BY: MEGAN IRWIN, ESQ.  
28 (949)223-7000

FOR WHOLE FOODS: BLAXTER/BLACKMAN, LLP  
BY: J.T. WELLS BLAXTER, ESQ.  
475 Sansome Street  
Suite 1850  
San Francisco, California 94111  
(415)500-7700  
wblaxter@blaxterlaw.com

(Appearances continued on next page.)

1 APPEARANCES CONTINUED:

2 FOR WALMART STORES, ETC.:

BARTKO, ZANKEL, TARRANT & MILLER  
BY: MICHAEL D. ABRAHAM, ESQ.  
One Embarcadero Center  
Suite 800  
San Francisco, California 94111  
(415)956-1900  
mabraham@bzbm.com

7 FOR COSTCO, ETC.:

ROGERS, JOSEPH, O'DONNELL  
BY: RENEE D. WASSERMAN, ESQ.  
311 California Street  
San Francisco, California 94104  
(415)956-2828  
rwasserman@rjo.com

11 FOR THE KROGER COMPANY:

NIXON, PEABODY, LLP  
BY: LAUREN M. MICHALS, ESQ.  
GREGORY P. O'HARA, ESQ.  
One Embarcadero Center  
Suite 1800  
San Francisco, California 94111  
(415)984-8261  
lmichals@nixonpeabody.com  
gohara@nixonpeabody.com

16 FOR STATER BROS, ETC.:

VARNER & BRANDT, LLP  
BY: BRENDAN W. BRANDT, ESQ.  
3750 University Avenue  
Suite 610  
Riverside, California 92501-3323  
(951)274-7777  
brendan.brandt@varnerbrandt.com

21 FOR SARA LEE:

SHOOK, HARDY & BACON, LLP  
BY: FRANK C. ROTHROCK, ESQ.  
NAOKI S. KANEKO, ESQ.  
5 Park Plaza  
Suite 1600  
Irvine, California 92614-2546  
(949)475-1500  
frothrock@shb.com  
nkaneko@shb.com

26

27

28

1  
2  
3  
4  
5  
6  
7  
8  
9  
10  
11  
12  
13  
14  
15  
16  
17  
18  
19  
20  
21  
22  
23  
24  
25  
26  
27  
28

M A S T E R I N D E X

October 2, 2017; P.M. Session

CHRONOLOGICAL INDEX OF WITNESSES

WITNESSES:	PAGE
RONALD MELNICK, PREVIOUSLY SWORN. DIRECT EXAMINATION (CONTINUED) BY MR. METZGER:	151

ALPHABETICAL INDEX OF WITNESSES

WITNESSES:	PAGE
RONALD MELNICK, PREVIOUSLY SWORN. DIRECT EXAMINATION (CONTINUED) BY MR. METZGER:	151



1 CASE NUMBER: BC411192/BC435759  
2 CASE NAME: CERT CASES  
3 LOS ANGELES, CALIFORNIA MONDAY, OCTOBER 2, 2017  
4 DEPARTMENT 323 ELIHU M. BERLE, JUDGE  
5 REPORTER: MARK SCHWEITZER, CSR 10514  
6 TIME: 1:45 P.M.

7 -o0o-

8 THE COURT: Good afternoon, Counsel. Back on the  
9 record in CERT versus Starbucks. All counsel are present.  
10 And Dr. Melnick is on the stand.

11 You may be seated. Do you understand you are still  
12 under oath?

13 THE WITNESS: Yes.

14

15 **RONALD MELNICK, PREVIOUSLY SWORN.**

16

17 THE COURT: Mr. Metzger, you may proceed.

18 MR. METZGER: Thank you, your Honor. Before we  
19 begin, I wanted to make a quick announcement and inform the  
20 Court that there was one defendant that identified 105  
21 witnesses for the remedies phase of the trial. And to spare  
22 us all, CERT has settled with that defendant. That is  
23 7-Eleven.

24 THE COURT: All right, thank you.

25

26 **DIRECT EXAMINATION (CONTINUED)**

27 BY MR. METZGER:

28 Q. Good afternoon, Dr. Melnick.

1           Let's talk about cancer risk assessment, if we can.

2           Now, as part of your work in this case, did you  
3 review probably several years ago and as well more recently  
4 the published risk assessment regarding acrylamide of various  
5 governmental agencies and authoritative bodies?

6           A.    Yes, I have.

7           Q.    And have you prepared a summary of how they  
8 went about selecting tumors for those?

9           A.    Yes, I have.

10          MR KENNEDY:  Objection, your Honor.  We filed a  
11 short brief with the Court this morning.  As I said earlier,  
12 when Mr. -- Dr. Melnick prepared his critique of Dr. Rhomberg,  
13 it's Exhibit 6 to his deposition, he focused in entirely on  
14 pharmacokinetic factors.  In his deposition on July 28th, he  
15 testified at length about PK factors.  Page 169, we asked him  
16 does this critique contain all of your present criticisms of  
17 Dr. Rhomberg's work?

18          His answer was:  "Yes, at this point in time, that  
19 is the extent of my criticisms, critique of Dr. Rhomberg's  
20 report and deposition.  If something more is stated, for  
21 example, if he were to write back a critique about me, I would  
22 take a look at it and look at the basis of it, but at this  
23 point that's where I stand."

24          And that was the last we heard about the subject  
25 until yesterday, when we were served with the demonstratives  
26 in this case, including, I think it's either 12 or 13 dealing  
27 with the new topic of tumor selection, and we would object to  
28 it at this point.

1 THE COURT: Mr. Metzger, what's your response?

2 MR. METZGER: Well, yes, I believe that Dr. Melnick  
3 testified about this back in 2014. And in addition, there's  
4 some information here -- this is not even necessarily  
5 regarding Dr. Rhomberg but in part regarding Dr. Melnick and  
6 these tumor sites. Dr. Melnick was, of course, one of the top  
7 people at the national toxicology program that did these  
8 studies, and Dr. Rhomberg, in fact, when asked who are the  
9 experts in doing these, he mentioned Dr. Melnick.

10 THE COURT: Well, I'll let the witness testify  
11 subject to a motion to strike.

12 You may proceed.

13 MR. METZGER: Thank you, your Honor.

14 Q. So how many risk assessments did you find of  
15 acrylamide for cancer risk from your review, Dr. Melnick?

16 A. There were four assessments that I identified.

17 Q. And the earliest was what?

18 A. In 2005.

19 Q. That's the FA -- what is that, Food and  
20 Agricultural Association/World Health Association. And what  
21 type of tumors did they include in their risk assessment?

22 A. Just for a little background, if I may.

23 Q. Sure. Go ahead.

24 A. The NTP conducted studies of acrylamide in rats  
25 and mice, and it was published approximately 2012.

26 Prior to that, there were studies in rats that were  
27 conducted by Freedman and Johnson. There's two separate  
28 papers. Those were the tumor incidence data that were used in

1 the risk assessments up until the EFSA, the fourth one  
2 indicated here. So yes, in the FAO/WHO risk assessment, they  
3 looked at mammary gland tumors in female rats, and they  
4 included the fibroadenomas and adenocarcinomas and  
5 peritesticular mesothelioma, the thyroid, and the central  
6 nervous system.

7 I highlight in the red the tumors which were  
8 excluded by Dr. Rhomberg.

9 Q. Okay. And the next was OEHHA in 2005. And  
10 what tumors did OEHHA include?

11 A. OEHHA in their assessment -- this is the  
12 assessment that was never finalized. But in that assessment  
13 it was all sites and that included the mammary gland, which  
14 were the fibroadenomas and adenocarcinomas, as well as the  
15 thyroid gland and the tunica mesotheliomas. That's the same  
16 as the peritesticular mesothelioma.

17 Q. Okay. And in 2010, the U.S. EPA risk  
18 assessment.

19 A. The U.S. EPA included the mammary gland  
20 fibroadenomas and adenocarcinomas in thyroid tumors in female  
21 rats, and the tunica vaginalis mesothelioma and thyroid gland  
22 in male rats. They didn't exclude any of those particular  
23 tumors.

24 Q. Okay. So up until the time that the NTP  
25 published its studies on acrylamide in 2012, all of the  
26 earlier risk assessments included all of these different  
27 tumors?

28 A. That is correct.

1 Q. Now, the European Food Safety Association in  
2 2015, is that the most recent cancer risk assessment for  
3 acrylamide by a major agency?

4 A. That is the only one I have identified.

5 Q. Okay. And what tumors did EFSA include?

6 A. EFSA did what's called a margin of exposure.  
7 And they base that margin of exposure on the harderian gland  
8 tumors in mice.

9 Q. Okay. So the risk assessment was based just on  
10 the harderian gland tumor; is that correct?

11 A. Yes, their margin of exposure values were based  
12 on the harderian gland tumors.

13 Q. And would you explain to Judge Berle what a  
14 margin of exposure is?

15 A. The margin of exposure is the relative dose in  
16 animals that produces a certain percentage of a tumor  
17 response, such as a 10 percent response, compared to the  
18 exposure that humans experience from that same agent.

19 Q. So is it essentially looking at how far apart  
20 the dose is that produces an effect in animals compared to  
21 what humans are actually exposed to?

22 A. That is correct.

23 Q. And is the lower the margin of exposure the  
24 greater the health concern?

25 A. That is correct. And the typical standard for  
26 that, as stated by both EFSA and the Food and Agricultural  
27 Association World Health Association work, is 10,000, and that  
28 is the type of margin of exposure that they want to see

1 without concern.

2 THE COURT: What does that mean? 10,000.

3 MR. METZGER: Let me ask this this way.

4 Q. Is that the difference between where the  
5 animals show an effect and what humans are actually exposed  
6 to, a 10,000 fold difference?

7 A. Yes, it's where the animals show a 10 percent  
8 response rate compared to the human exposure. What is the  
9 dose that is associated with a 10 percent response rate  
10 compared to the exposure in humans.

11 THE COURT: All right. So you're saying that a 10  
12 percent exposure rate in an animal is equivalent to a risk  
13 rate in a human being?

14 THE WITNESS: No. I'm saying that the dose that  
15 creates the 10 percent response compared to the exposure that  
16 humans experience. So looking at a 10 percent response rate  
17 in animals, what dose causes that and what is the exposure in  
18 humans.

19 Q. BY MR. METZGER: And how far apart those two  
20 values are. Is that it?

21 A. Yeah. What is that ratio.

22 THE COURT: And where does the 10,000 play here?

23 THE WITNESS: Well, 10,000 is the typical value in  
24 which they consider it not a concern.

25 Q. BY MR. METZGER: And what is the value for  
26 acrylamide?

27 A. It was approximately 70.

28 Q. 70?

1           A.    Yes.  And therefore, they said this is a  
2 concern.

3           Q.    So is that essentially saying that adverse  
4 effects, responses in animals are seen at a dose that's just  
5 70 times what humans are exposed to?

6           A.    Yes.  The 10 percent response rate is only 70  
7 times higher than human exposure, which they consider to be,  
8 and I do as well, consider to be a health concern.

9           Q.    Okay.  Now, regarding the EFSA 2015 risk  
10 assessment, how is it that EFSA did its risk assessment based  
11 exclusively on the harderian gland, which people don't even  
12 have?

13          A.    I've written an article on this particular  
14 issue of epoxides and sites on which epoxides induce cancer  
15 and in that article -- this was back in 2002 -- I noted that  
16 harderian gland was a common target for a number of  
17 epoxide-forming chemicals.

18          Q.    Like acrylamide?

19          A.    Well, I didn't take food acrylamide in that,  
20 but there were a number of other epoxide-only chemicals that  
21 were -- induced harderian gland tumors in mice.  In fact, I  
22 sort of considered that to be like the canary in the coal mine  
23 for epoxides, where this is the warning that the harderian  
24 gland represents a site for cancer induction by  
25 epoxide-forming chemicals.

26          Q.    So what is the significance in terms of risk  
27 assessment that the harderian gland tumors in mice are  
28 commonly seen in mice exposed to epoxide chemicals?

1           A.    It's indicating that epoxides have that  
2 capability of forming a tumor response.  This is biology.  
3 That the biology demonstrates tumor induction in this  
4 particular site which is common among epoxide forming  
5 chemicals.

6           Q.    Does that tend to indicate that it is not a  
7 fortuitous occurrence?

8           A.    No.  This is something which seems to concern  
9 or demonstrate a concern for epoxide-forming chemicals.

10          Q.    Okay.  And here is the EFSA 2015 document, and  
11 if we look at this paragraph on Page 191, let's see, it says  
12 the Contam panel --

13          MR. KENNEDY:  Objection.  This wasn't included in  
14 the demonstrative.  I'm not sure it was even produced in the  
15 case.

16          MR. METZGER:  It was.

17          THE COURT:  Subject to a motion to strike.  Counsel  
18 can discuss where the document can be found.

19          MR. MARGULIES:  Do you have an exhibit number?

20          MR. METZGER:  It was identified earlier.  I don't  
21 see my copy of it.

22          THE COURT:  Okay.  Let's stop this chitchat.  
23 Discuss it during the break.  Next question.

24          Q.    BY MR. METZGER:  Yes, it says the Contam panel  
25 considered that even though the harderian gland is not present  
26 in humans, this rodent organ represents a sensitive end point  
27 for detecting compounds that are both genotoxic and  
28 carcinogenic.  And it cites three studies.  And then it says

1 harderian gland tumors and tumors in other rodent organs,  
2 including the lung in mice, the brain in rats, and the mammary  
3 gland and forestomach in both species are prone to tumor  
4 formation upon exposure to epoxides or epoxide-forming  
5 carcinogens, citing Melnick 2002, such as acrylamide.

6 Is that you?

7 A. I'm that Melnick, yes.

8 Q. And if we look at your Curriculum Vitae on  
9 Page 20, is the article identified as No. 95 by Ronald L.  
10 Melnick, Carcinogenicity and Mechanistic Insights on the  
11 Behavior of Epoxides and Epoxide-Forming Chemicals published  
12 in the Annals of New York Academy of Sciences? Is that what  
13 EFSA relied on?

14 A. Yes, that's the article they are referring to.

15 Can we go back to that page again?

16 Q. Sure. On the EFSA. If you can read the  
17 paragraph that begins therefore.

18 THE COURT: Wait, wait. This is not a reading  
19 exercise. Next question.

20 Q. BY MR. METZGER: All right. So do you agree  
21 with EFSA's conclusion that therefore, the results on the  
22 harderian gland in mice cannot be disregarded in the risk  
23 assessment of acrylamide?

24 A. That is correct. And that's what I feel and  
25 that's what the panel concluded, that the results on the  
26 harderian gland in mice cannot be disregarded in the risk  
27 assessment.

28 Q. Okay. All right.

1           Have you assessed what the effects of excluding the  
2 various tumors that Dr. Rhomberg excluded on the NSRL?

3           A.    Yes, I've made that comparison.

4           Q.    And what did you find?

5           MR. KENNEDY:  Objection, your Honor.  This is  
6 Dr. Bayard's work.  I understand it has tried to be  
7 introduced, and your Honor kept it out during Dr. Rhomberg's  
8 exam.  We object to it at this point.  He's just trying to  
9 read Bayard's stuff into evidence.

10          THE COURT:  The witness can testify based on hearsay  
11 subject to a motion to strike, cross-examination.

12          Q.    BY MR. METZGER:  Dr. Melnick, what did you  
13 find?

14          A.    I did these calculations and found that the  
15 NSRL was increased as a consequence of excluding particular  
16 tumor sites.  The tunica mesothelioma in male rats was  
17 increased approximately 30 percent, 1.3.  Mammary gland  
18 tumors, excluding those from female rats, increased the NSRL  
19 by nearly a factor of three.  Excluding the harderian gland,  
20 tumors in male mice increased the NSRL by nearly fivefold, and  
21 excluding the harderian gland tumors in female mice increased  
22 the effect on the NSRL by a little over a factor of two.

23          So if you run a risk assessment and you pick the  
24 male rat or female rat or male mouse or female mouse, these  
25 are the values that are increased in the NSRL for that  
26 particular species by excluding those tumor sites.

27          Q.    So as these different tumors and tumor sites  
28 are excluded from the risk assessment, does that reduce the

1 risk?

2 A. The risk is increased by excluding sites.

3 Q. Okay. The no significant risk level is  
4 increased.

5 A. That is correct.

6 Q. Okay.

7 A. The potency is decreased.

8 Q. That was the term. I used the incorrect term.  
9 So the potency is decreased, when you exclude the tumors,  
10 because you don't have this potent effect, but that increases  
11 the no significant risk level because you can have higher  
12 exposures.

13 A. That is correct.

14 Q. Okay. All right. So now I think we're at the  
15 topic of pharmacokinetics, which you mentioned at the very  
16 beginning, and I know this is going to get complex. So let's  
17 take this slowly.

18 First, have you prepared a diagram of the human body  
19 to show essentially how chemicals are distributed and  
20 metabolized?

21 A. Yes, I have.

22 Q. All right. So there we have this nice  
23 gentleman with a cup of coffee I see in front of him, and AA.  
24 Is AA for acrylamide?

25 A. Yes, that's what the AA represents.

26 Q. All right. So would you just tell us what  
27 you've intended to convey by means of this diagram?

28 A. Okay. Since we all just finished our lunch a

1 little while ago, this is what happens to chemicals that  
2 your body may not necessarily want to see. It wants to  
3 eliminate them. So if we consider in this case a cup of  
4 coffee containing acrylamide, the exposure of this individual  
5 is an oral exposure. Drinking a cup of coffee. As a  
6 consequence of drinking coffee and having acrylamide in it,  
7 the acrylamide passes into the stomach of the individual.

8           Unfortunately, this diagram I picked didn't have an  
9 esophagus, but eventually it passes down the esophagus into  
10 the stomach, and you can see represented in the stomach the  
11 AA.

12           Q.    Okay.

13           A.    From the gastrointestinal tract there is a  
14 direct vein that feeds materials to the liver. This is called  
15 the portal vein. And as a result, the chemical passes into  
16 the liver before it gets systemically distributed. Okay?

17           Q.    Um-hm.

18           A.    In the liver -- the liver has a major role in  
19 metabolism of foreign agents for the purpose of trying to get  
20 rid of them from the body.

21           Q.    So we call that detoxification?

22           A.    Yes, well, it wants to get rid of them, okay?

23           Q.    All right.

24           A.    Whether or not it's toxic at that moment can  
25 depend on what happens in the course of the metabolism.

26           Q.    Okay.

27           A.    In the liver there are a number of enzymes  
28 which will act on that agent. One of those enzymes will

1 oxidize acrylamide to glycidamide. Glycidamide is the epoxide  
2 form of oxidation of acrylamide.

3 Q. Is that enzyme you're referring to that famous  
4 one called cytochrome P-450 2E1? Is that it?

5 A. That is the common enzyme for low molecular  
6 weight vinyl type of compounds, yes. There's a bunch of  
7 cytochrome P-450s. 2E1 is the form which is the primary  
8 metabolism on compounds like vinyl chloride, butadiene as well  
9 as acrylamide.

10 Q. Okay.

11 A. So in the liver metabolism is occurring, and  
12 I'm going to show in a little more detail in the next slide,  
13 but what I'm representing here is that this is how glycidamide  
14 can get into our bloodstream.

15 Q. Wait a second. It was acrylamide. How did we  
16 get to glycidamide?

17 A. By that cytochrome P-450 2E1 metabolism  
18 oxidized acrylamide to glycidamide.

19 Q. So it converts or changes the acrylamide into  
20 the genotoxic glycidamide?

21 A. This is extra additionally called an activation  
22 step because the glycidamide is the activated form of major  
23 concern for acrylamide exposure.

24 Q. All right. So now we have glycidamide in a  
25 vein, right?

26 A. Right. And as a consequence, so you can see  
27 within here how materials get distributed in the venous blood,  
28 which is shown as blue, where it's passing into the heart and

1 the lungs and get distributed. Eventually, it comes back to  
2 the heart and then passes into the arterial blood, and you can  
3 see even on the right side there at AA. That's acrylamide  
4 which hasn't been metabolized, which also exited the liver  
5 unmetabolized, can also get distributed to other organs within  
6 the body. And some of it will go back to the liver. Some  
7 will go on to other organs.

8           So what this diagram is intended to do is just to  
9 show how chemicals which are ingested can be distributed  
10 throughout the body and a role for liver in the metabolism of  
11 that particular agent.

12           Q. All right. I believe you're going to tell us  
13 now about specifically metabolism of acrylamide in the liver.

14           A. Okay. So this is now the liver where we're  
15 just talking about the metabolism of acrylamide. So you can  
16 see in the upper left, that's acrylamide. Acrylamide can  
17 undergo two different pathways of metabolism. One is the  
18 cytochrome P-450. And that we would call an activation step  
19 forming glycidamide, or the acrylamide may be conjugated with  
20 glutathione. You may remember this morning when I mentioned  
21 cysteine binding to acrylamide. Glutathione is the three  
22 amino acid molecule which can bind to acrylamide.

23           And in the liver this is catalyzed by an enzyme  
24 called glutathione S-transferase. Once that happens, like I  
25 mentioned with cysteine, that metabolite is no longer able to  
26 be oxidized to glycidamide. So that's a detox pathway.

27           Glycidamide itself can also be conjugated with  
28 glutathione by glutathione S-transferase, forming conjugates,

1 glutathione conjugates of glycidamide, or it can undergo  
2 hydrolysis to glyceramide.

3           So you have competing pathways and the direction of  
4 that pathway depends in essence on the affinity of the enzymes  
5 for acrylamide.

6           So the P-451 typically has a stronger affinity than  
7 the glutathione transferases. But one thing I also want to  
8 point out is that these enzymes, the epoxide hydrolase and the  
9 glutathione S-transferase, are what we call polymorphic,  
10 meaning that there are different forms of that enzyme in  
11 different people within the population. Polymorphism would  
12 represent at least 1 percent of a population having an altered  
13 form of that. And some of these polymorphisms result in  
14 lacking the activity of certain of these enzymes.

15           So, for example, there are a number of glutathione  
16 transferases. But there are polymorphisms that some people  
17 lack an enzyme which is capable of causing that detoxification  
18 pathway as efficiently as another individual.

19           So to me, polymorphism is an important issue because  
20 not everybody is the same in terms of how they will activate  
21 and detoxify the enzyme.

22           And one other thing on cytochrome P-450 2E1, it's  
23 also an induceable enzyme.

24           Q.    Meaning what?

25           A.    Meaning there are certain agents which will  
26 increase the level of cytochrome 450 in people exposed to  
27 certain drugs. Alcohol, for example, is an inducer of  
28 cytochrome P-450 2E1.

1           So the metabolism can vary substantially in  
2 individuals because of the levels of activities of these  
3 enzymes which are activating and detoxifying acrylamide.

4           Q.    So why is this all important?

5           A.    The importance is we're trying to characterize  
6 what I mentioned earlier, the dosimetry of glycidamide.  
7 Because the belief is that glycidamide is the primary  
8 carcinogen of exposure to acrylamide.  It doesn't mean  
9 acrylamide doesn't do anything.  It's just that glycidamide is  
10 the stronger genotoxic agent that binds to the DNA.

11           So in order to understand the risk associated with  
12 exposure to acrylamide, you want to understand what is the  
13 dosimetry, which I mentioned before is the concentration in a  
14 tissue over the time that that compound is in there.

15           So like in the liver, you can see acrylamide enters.  
16 As soon as you finish your coffee, the acrylamide is being  
17 metabolized.  Some is being distributed.  Some is  
18 disappearing.  Glycidamide is formed.  It's also being  
19 metabolized away, or it may bind to certain structures.

20           So to understand the risk of exposure to an agent  
21 such as acrylamide, there are a number of ways to calculate  
22 exposure or dose.  One is just what are you exposed to.  
23 Milligrams per kilogram.

24           Another is what is the internal dose of acrylamide.

25           Q.    In a particular tissue?

26           A.    Right.  In particular tissues.  And taking it  
27 further, what is the tissue dose over time for glycidamide.

28           And that's what physiologically based

1 pharmacokinetic models are attempting to characterize so that  
2 they could be used in a risk assessment as opposed to simply  
3 saying what is the exposure that the individual received.

4 Q. All right. So are there various factors that  
5 affect glycidamide in tissues?

6 A. Yes. This is the kind of information. And  
7 this isn't all of it, but it's as many as I could fit on the  
8 slide, that are involved in affecting the blood levels of  
9 glycidamide exposure to acrylamide. And these are the types  
10 of parameter values that would be included in a  
11 physiologically based pharmacokinetic model. So when I say  
12 physiologically based, this is not just two compartments of  
13 something moving. This is taking into account breathing  
14 rates, cardiac output, movement of materials in the blood.

15 So it accounts for the full physiological basis of  
16 the organism, human or rodent, and it's pharmacokinetic, make  
17 it changes its action that's happening. It's changing levels.  
18 So this is what would lead into the formation of a  
19 physiologically based pharmacokinetic model.

20 Q. If a physiologically based pharmacokinetic  
21 model is well developed and supported, what goal is  
22 accomplished? What do you do with that?

23 A. Well, you can, as I mentioned, there's a lot of  
24 variability among humans. We can include different parameter  
25 values into the model to see how individuals may respond  
26 differently. We can identify the tissue level in humans  
27 compared to rodents to see how well they compare. If we have  
28 a good physiologically based pharmacokinetic model, then that

1 information could be used in the risk assessment as opposed to  
2 body weight scaling for the pharmacokinetic portion of the  
3 risk assessment.

4 Q. So if a physiologically based pharmacokinetic  
5 model is validated and is well done and documented, does  
6 that -- is the concept that this helps improve the risk  
7 assessment?

8 A. Yes, definitely. This is the kind of work that  
9 we've done with the 13 butadiene, for example. To  
10 characterize the epoxide -- there's two epoxides with  
11 butadiene concentrations in rodents and humans.

12 Q. When you say this is what we have done, is that  
13 the national toxicology program? Who is the "we"?

14 A. The we is Michael Cohen, who is a mathematical  
15 modeler, and myself, and we've published numerous papers on  
16 this.

17 Q. And those are listed in your CV?

18 A. Yes.

19 Q. All right.

20 A. So I just want to finish on this.

21 Q. Yes, please.

22 A. So some of the types of information that you  
23 want are, for example, how does it get absorbed from the  
24 GI tract? What is the rate? Remember, this is kinetic  
25 modeling. So we're interested in rates. What are the  
26 parameters physiological, the heart in terms of how much blood  
27 goes to various organs. What is the organ volume.

28 A partition code is a value which relates to the

1 distribution between the blood and the tissue itself. So  
2 there would be a partition coefficient for a small crossing  
3 from the blood into the liver or blood into the kidney.

4 Then we have the metabolic processes. What is the  
5 rate of oxidation of acrylamide to glycidamide, to P-450 2E1.  
6 The rate of glutathione conjugation, what I showed in the  
7 liver. These are rates that you can input into a model, but  
8 if you don't have them, you base it on the experimental data  
9 that you have available, and you run the model to match the  
10 experimental data such that the model will tell you this is  
11 the best fitting parameter value that will match the  
12 experimental data.

13 Q. Okay. And you have all of these other. Rates  
14 of glutathione conjugation, hydrolysis of glycidamide,  
15 transfer of glycidamide into blood. Binding of macro  
16 molecules and excretion of glycidamide into urine. These are  
17 a bunch of the things that you have --

18 A. You either have some of this information, for  
19 example, binding to hemoglobin. You can do that externally in  
20 vitro. In terms of setting up a model, if you have no idea  
21 what the metabolic rate could be, you may have, for example,  
22 in vitro measurements of the oxidation of a chemical like  
23 acrylamide to glycidamide, and you can plug in that kind of  
24 information into the model.

25 Now, the model is going to run. These are runs of  
26 iterations looking for the best parameter values. And it may  
27 say well, it's a little bit off. But this is the best I can  
28 do in terms of fitting the model to the data. You don't fit

1 data to a model. The data is sacred. The model is what's  
2 being adjusted to fit the data.

3 Q. Got it.

4 All right. So your next graph is entitled  
5 physiologically based pharmacokinetic model. Could you  
6 explain what this is.

7 A. So this is now in a different diagram. The  
8 person that I showed earlier of what a model might look like.

9 So next, with an oral dose, you characterize it  
10 entering into the GI tract. Some of it may pass into the  
11 feces. Or it may enter into the GI blood and pass by the  
12 portal vein into the liver blood and eventually come into the  
13 liver where it can undergo metabolism.

14 So the model is intended to mimic the human or  
15 rodent, depending on the nature of your data, to the extent  
16 possible.

17 I don't have a connection between the venous blood  
18 and arterial blood, but that's basically the heart and lung,  
19 where the heart is pumping, and the lung is oxidizing.

20 Q. Okay. So what did you consider next regarding  
21 the pharmacokinetics?

22 A. So I just hope it's clear that the model is  
23 producing parameter values that are uncertain or unknown or  
24 not well characterized to fit available data.

25 Now, one type of information which is useful to have  
26 is the formation of adducts.

27 Q. Which are?

28 A. Adducts are basically addition products that

1 are formed when a material binds to something else and forms a  
2 new product.

3 Q. Dr. Rappaport testified all about that in 2014.  
4 I remember it now.

5 All right. So what about DNA and hemoglobin adducts  
6 for acrylamide or glycidamide?

7 A. So glycidamide is a relatively small molecule.  
8 It's only a few carbons. Three carbons with the epoxide. And  
9 it will find places on DNA because as I mentioned earlier,  
10 acrylamide and glycidamide in particular is an electrophile.  
11 It's looking for electrons that it can bind to. When it's in  
12 the formation of an epoxide, it's less stable than the  
13 acrylamide itself. It is looking for those electrons to allow  
14 that strain on the epoxide to be relieved, and it can find  
15 those electrons in DNA. It can bind to where there may be  
16 certain free amines on the DNA basis that I mentioned earlier  
17 briefly and form a DNA adduct.

18 Also, both acrylamide and glycidamide have been  
19 shown to form adducts with hemoglobin. There's a particular  
20 site, the terminal valine amino acid, where these tend to show  
21 good binding, and this is another characteristic of epoxide  
22 and other electrophilic compounds.

23 Q. And why are you looking at hemoglobin adducts  
24 in particular?

25 A. In the next slide I want to try to explain the  
26 concept of area under the curve.

27 Q. Okay.

28 A. Okay. Because I believe you've heard that

1 term. It may have been explained adequately. Maybe it  
2 wasn't. But just to keep everybody in the same understanding.

3           So with what is plotted here is the blood  
4 concentration over time. And if we gave a dose, as I show  
5 there, of a compound such as acrylamide, there's going to be a  
6 rise in the blood levels of acrylamide or glycidamide,  
7 depending on what is measured because it may have come from  
8 passing through the liver if it wasn't all metabolized as the  
9 acrylamide. Some acrylamide will wind up in the blood. And  
10 glycidamide will also wind up in the blood.

11           So you can see this is not an experimental data set.

12           Q. This is illustrative?

13           A. Illustrative, right. So because starting off  
14 with essentially zero, you see an increase, and with time it's  
15 going to decrease because it's being metabolized away and  
16 eventually excreted. That's what the body wants to do. It  
17 wants to get rid of it. But unfortunately, the body activated  
18 it as well.

19           So this would represent the AUC for glycidamide or  
20 acrylamide in the blood. And that then becomes a marker of  
21 the internal dosimetry because we're looking at time and  
22 concentration at the same -- on the same graph. So that area  
23 under the blue curve is what is called the area under the  
24 curve.

25           For hemoglobin it's a little bit different. You can  
26 see it rises, and these values are not necessarily scaled  
27 properly. But it rises, and it remains eventually flat. And  
28 the reason for this is that once it binds to hemoglobin, it

1 forms a stable adduct, but it doesn't disappear until the red  
2 blood cell undergoes degradation. And the red blood cell has  
3 a half life somewhere around three, four months.

4           So it can remain in the blood for some time, and  
5 this then becomes a valuable biomarker for exposure, but it's  
6 also then used in trying to determine the relative hemoglobin  
7 adduct levels for glycidamide across species.

8           So what I showed here, though, was a theoretical  
9 curve. Now, the model -- this might be the output of a model.  
10 But I didn't show data. And if I showed X's on that blue  
11 line, that would have said wow, that model did a great job of  
12 representing the experimental data. We've got some pretty  
13 good confidence.

14           If I put those little dots of what the measurements  
15 were, and they are all over the place, or maybe one of the  
16 compounds increases whereas the model is showing a decrease  
17 and say wait a second. That's not a very good fit of the  
18 model to the data.

19           So what you need to do is if you think you have a  
20 good fit, because you fit it to a particular data set, is to  
21 try to validate that you got it right.

22           Q.    How do you do that?

23           A.    Validation would be to test your model with a  
24 different set of data. So if you have blood time course data  
25 in rodents, if you have it in humans and you try to fit a  
26 model to that, you may look at an alternative exposure and see  
27 whether the model still predicts the levels that are  
28 determined experimentally.

1           So what we're looking at, does the model predict the  
2 actual outcome, and the outcome is the data. So the data is  
3 sacred. The model needs to fit the data.

4           Q.    Okay. All right. So how does this concept  
5 that you just described, explained, how does this now fit into  
6 the risk assessment?

7           A.    Okay. So the risk assessment, as I mentioned,  
8 could be done based on the actual exposure, but if you have a  
9 model and the model is valid and good, then maybe you can use  
10 the model in replacement of the body weight scaling factor.

11           So a body weight scaling factor is to adjust the  
12 animal cancer potency for differences in animals and humans  
13 for pharmacokinetic and pharmacodynamic differences. There's  
14 two aspects. The pharmacokinetics, characterizing the  
15 internalized dosimetry. What is the dose that gets to the  
16 tissue. And the pharmacodynamics is what is the change that  
17 occurs in the tissue as a consequence of that dose of the  
18 chemical.

19           So a pharmacodynamic property might be causing DNA  
20 damage, as an example, or a mutation. What is that  
21 relationship. Now, there's very little pharmacodynamic  
22 information. So the focus has now been for acrylamide on the  
23 pharmacokinetic factor.

24           And Prop 65 says you can make a pharmacokinetic  
25 adjustment when the data can be taken into account with  
26 confidence, and that's what I want to explore. Can we take it  
27 into account with confidence.

28           So the PK factor, as I mentioned, can be the ratio

1 of the blood area under the curve of glycidamide. That would  
2 be the carcinogen in exposed humans compared to the blood area  
3 under the curve of glycidamide in the exposed animal.

4           So we're comparing a ratio. Is there greater, less,  
5 or what is that value? Okay? And that's what has been  
6 attempted in this particular case.

7           Alternatively, it is the ratio of the area under the  
8 curve of the hemoglobin adducts in exposed humans compared to  
9 exposed animals. So when I showed you the AUC graphs, we're  
10 comparing those area under the curve for humans versus rodents  
11 to see how do these stack up with respect to how the body is  
12 absorbing, metabolizing, and eliminating glycidamide.

13           Q. Okay. So what's the next step?

14           A. I looked at what Dr. Rhomberg did. And in my  
15 feeling the PK factors that he used didn't meet this level of  
16 confidence, that they are not reliable.

17           And this is what I hope to explain over the next  
18 series of slides. But for mice, they used a pharmacokinetic  
19 model for the tumors induced in mice, and this was a model  
20 that actually was not used by EPA. The model that was  
21 available at that time was the model by Young. And it's been  
22 referred to, I know, in this case from 2007. Young developed  
23 a PBPK physiologically based pharmacokinetic model for rats,  
24 mice, and humans. That's pretty much the title of their  
25 publication.

26           Now, why wouldn't EPA use that model for rats since  
27 in 2010 when they did their risk assessment, they were -- they  
28 had rat tumor data. And here was the Young model which was

1 available. But they claimed you can't because --

2 Q. You can't what?

3 A. Use that model for risk assessment. So as I  
4 mentioned, there are a number of parameter values which come  
5 out of a model.

6 Now, when you have multiple data sets, so for  
7 example, you may have one data set in which the animal was  
8 exposed by what we call gavage, it's a bolus dose injected  
9 into the animal's stomach. It's with a syringe with a bill  
10 hard ball at the end so you don't scratch the esophagus. And  
11 it places a bolus dose of that chemical right into the stomach  
12 of the animal.

13 Alternatively, you may expose the animal in drinking  
14 water, such as was done in the two-year study, or in the feed.  
15 And in that case, rather than seeing it all come in at once,  
16 it's coming in more slowly. And the data will be different,  
17 but the parameter values, these are called rate constants.  
18 They are expected to be constant by definition.

19 So unfortunately, within the Young model, they  
20 needed different rate constants to fit different data sets.  
21 And EPA said, which I agree, is that you need to find a single  
22 set of data of parameters that will fit all of the data.

23 And another thing which I hadn't mentioned was you  
24 also need to look at a sensitivity analysis of your parameter  
25 values. Now, why do you do a sensitivity analysis? Because  
26 there's uncertainty. When your model gives you a parameter,  
27 say, a rate for oxidation of glycidamide, or for GST. If you  
28 don't have that parameter value very carefully determined, if

1 this is coming out of your model and you have multiple values,  
2 one thing is which one do you use?

3           Secondly, is how big of an impact is it on the  
4 outcome of the model if that parameter value is off by a  
5 little bit? And that's what you do with a sensitivity  
6 analysis. You are trying to check up on your parameters to  
7 see is a small error in that parameter going to have a big  
8 impact or a little impact on the output of the model. Because  
9 if it has a big impact, you've got to be careful that you have  
10 the right parameter value because you're going to get answers  
11 all over the place. If it's a small value, it won't matter so  
12 much if that parameter is off by a little bit.

13           So you can test your model by varying parameter  
14 value, maybe by 10 percent or so, and seeing how well did that  
15 affect the fit of the model to the data.

16           And that's what a sensitivity analysis entails. So  
17 EPA, and I agree, said that by having multiple parameters to  
18 fit multiple data sets and no sensitivity analysis, the model  
19 was not ready for use in risk assessment.

20           Q. Are you referring to the Young model?

21           A. Exactly. That's the Young paper in which the  
22 model was produced.

23           Q. So in the EPA risk assessment for acrylamide,  
24 the EPA rejected and did not use the Young pharmacokinetic  
25 model?

26           A. That is correct. Now, remember, that was for  
27 rats.

28           Q. Okay.

1           A.    Because at that time EPA only had rat tumor  
2 data.  But the Young model was available in 2007.  And it had  
3 also a model for rats.  But that rat model then was not  
4 accepted for use by EPA in their risk assessment of  
5 acrylamide.

6           Q.    So what about mice?

7           A.    Okay.  So Dr. Rhomberg derived a PK factor for  
8 mice.  And his determination of that value, again, this would  
9 be the area under the curve for mice, was based on the Young  
10 model, the Young 2007 model, of which there was information in  
11 terms of a prediction of that area under the curve, in the  
12 blood of mice from the Young model.

13                  However, the data that was used for that model was  
14 by a co-author of the Young paper, Dan Doerge.  This is from  
15 the FDA laboratories outside of Little Rock, Arkansas, that  
16 there is actual data, actual serum measurements.  And rather  
17 than using the serum measurements, which is data, they used --  
18 he used --

19           Q.    Who is the "they"?

20           A.    I'm sorry.  Dr. Rhomberg used, and he was  
21 basing it somewhat on the EFSA document, but he used the  
22 model-based estimate.  And now, as I said a couple of times,  
23 the data is sacred.  The model is trying to mimic, to explain  
24 the data.

25                  So this is a criticism of the use of the mouse model  
26 for the AUC.  This is the blood concentration over time rather  
27 than using actual data.

28           Q.    So I'm not understanding.  There was data for

1 mice, area under the curve. What did Dr. Rhomberg use?

2 A. He used the model prediction of the area under  
3 the curve.

4 Q. Is there any precedent or authority for doing  
5 that?

6 A. Well, in my view I don't know if there's  
7 precedent or not, but data --

8 Q. You should use the data.

9 A. If I can use the word, trump's model  
10 predictions -- one is a predicted value based on fitting model  
11 parameters which have uncertainties in them compared to actual  
12 data. If you have actual data, that is primary.

13 Q. Okay. And did you assess what the impact  
14 Dr. Rhomberg's use of a prediction rather than the data that  
15 was available, how that affected his calculated NSRL?

16 A. This causes a decrease right now in the PK  
17 factor.

18 Q. Okay.

19 A. And it's -- if you use body weight scaling, the  
20 PK factor for pharmacokinetics is approximately 1.9. I'm  
21 sorry. We're dealing with mice. I don't know what the number  
22 is offhand. But by using the model instead of the data, the  
23 decrease is approximately 10 or 20 percent.

24 Q. The decrease is the PK factor?

25 A. Yes.

26 Q. And what effect does that have?

27 A. That's comparable in decrease in the potency  
28 index for the chemical by that same proportion.

1           Q.    Okay.  And did Dr. Rhomberg, in doing this, use  
2 data from gavage or from dietary administration?

3           A.    Okay.  In a paper by Doerge, again, this is  
4 from the FDA laboratories outside of Little Rock.  They  
5 provided information on area under the curve AUC for mice for  
6 both dietary as well as by gavage.  And what they found was  
7 that there's a difference.  And as I mentioned, with gavage  
8 it's a bolus dose which is good for characterizing a  
9 parameter, but you can wind up with a difference if the  
10 material is coming in more slowly, such as through drinking  
11 water or consuming a diet.

12                The difference was by using the gavage  
13 administration as opposed to the dietary, it decreases the PK  
14 factor by 60 to 70 percent.  And the consequence of that on  
15 the NSRL is to increase the NSRL by 3.3 for male mice and 2.3  
16 for female mice.

17                So which one is more appropriate?  Well, most humans  
18 don't just gulp through coffee like we do with the gavage  
19 experiment in animals.  And they may consume coffee over the  
20 course of a day.

21                The NTP's carcinogenicity study was a drinking water  
22 study.  So it wasn't a situation in which the animals received  
23 all of their dose was one bolus by gavage.  So to me the more  
24 appropriate value to use for the AUC would be the drinking  
25 water or the dietary administration AUC that had available  
26 data.  It was there.  But that wasn't used to calculate a PK  
27 factor for mice.  And as I say, this has a reasonably big  
28 increase in the NSRL for male mice and for female mice by 2.3

1 and 3.3 fold.

2 Q. Okay. Well, what about the serum glycidamide  
3 under the curve for humans?

4 A. Well, this one becomes a major problem because,  
5 as I mentioned, to get a PK factor, you're comparing the area  
6 under the curve in humans compared to rodents. Okay? So up  
7 to here, we were discussing the issues and problems with how  
8 the rodent AUC was determined.

9 But now we're looking at how do we get an AUC for  
10 humans. As I mentioned, the Young model, it's multiple  
11 models. They also developed a model for humans. However,  
12 that model was never validated.

13 Q. What does that mean in this context?

14 A. Well, in this context there was no human blood  
15 data in order to determine whether the predictions of the  
16 blood levels were correct. Because the model is predicting  
17 that AUC blood levels of glycidamide in human blood, and they  
18 have no blood data to which they can parameterize their model  
19 or validate it.

20 So you can make a prediction with a model. A model  
21 is a hypothesis until it's demonstrated to be accurate. So  
22 you have a hypothesis, but it's untested because you don't  
23 have the blood data that enables you to say yes, the model  
24 that we created actually does reflect the blood concentrations  
25 in humans.

26 And if you had one set of good data and you created  
27 a model, you can't use the same data set to validate the model  
28 because if you use the same data set, you're going to be right

1 100 percent of the time. Because you create a model with the  
2 data set, and then you can say well, let's see how well it did  
3 against the data set. And, of course, you're going to get it  
4 right. So you need an alternative data set to see whether you  
5 can actually show that your model can predict other  
6 circumstances.

7           Now, for the human glycidamide PBPK model, there was  
8 some data available, but it wasn't blood data. It was urinary  
9 metabolites that were determined from a paper by Fuhr taken  
10 from six healthy individuals who consumed a meal containing  
11 acrylamide. So what the model has for its data set are  
12 concentrations of those glutathione conjugated metabolites  
13 that I showed in the liver that eventually get excreted in the  
14 urine.

15           So you have these metabolites in the urine. And  
16 from that information, you're trying to predict the blood  
17 concentration of glycidamide which led to those excretions in  
18 the urine.

19           So to me this is not a very strong data set in which  
20 to create a model because you're using urine measurements to  
21 predict rates of metabolism in the liver and consequent  
22 concentrations of glycidamide in the blood.

23           And as I mentioned, those enzymes are also  
24 polymorphic, and there was a study done by Duale in which they  
25 compared in humans the ratio of glycidamide to acrylamide  
26 hemoglobin adducts because this gives a reflection whether  
27 it's constant or variable. And they saw a ninefold  
28 variability among 44 individuals in whom they measured this

1 adduct level. This is just the measurement in which it's  
2 dietary exposure that happens naturally to humans.

3           So you got a variability effect, and unfortunately,  
4 the agencies don't know how to deal with variability. And  
5 they don't adjust for it. But if you had a good  
6 physiologically based pharmacokinetic model and you knew a  
7 distribution of these parameter values, you can plug that in  
8 and come up with the determination of how human variability  
9 impacts the risk.

10           Q. And what do you conclude regarding this?

11           A. Well, that the use of a human model has no  
12 reliability for determining a pharmacokinetic factor. It's  
13 totally unreliable. It's not based on sufficient data. And  
14 in my view, you can't use the human proposal for the area  
15 under the curve for glycidamide based on the model and the  
16 limited data set that was available for creating the model.  
17 So my view is that the pharmacokinetic adjustment factors that  
18 were used have no scientific basis.

19           Q. All right. And I think you mentioned that for  
20 Prop 65, you could use a pharmacokinetic factor where the data  
21 was -- did you say with confidence, where you could -- what  
22 was that?

23           A. I'll have to go back to see how Prop 65 words  
24 it. You need to have good confidence in -- a pharmacokinetic  
25 adjustment may be made when available data can be taken into  
26 account with confidence.

27           Q. And in your opinion can the available data be  
28 taken into account with confidence?

1           A.    I have no confidence in the PK factor that is  
2 used for the mouse tumor response.

3           Q.    So what was the effect of Dr. Rhomberg's using  
4 PK factors that he derived based upon the Young and the Doerge  
5 or the Young model and the Doerge data instead of using the  
6 body weight scaling?

7           A.    I have a table coming up with two slides after  
8 we do the rat on this.

9           Q.    Oh, we still have the rat to do.   Okay.

10          A.    But as a hint to your question, it raises the  
11 NSRL by about four- to fivefold.

12          Q.    Okay.  I didn't mean to cut you off.  Can you  
13 tell us what was the significance regarding the rat?

14          A.    Okay.  So the rat was based on hemoglobin  
15 adducts.  The rat was based on glycidamide-hemoglobin adducts  
16 that were measured in rats and humans.  There were six  
17 individuals per dose group.  And the hemoglobin adducts were  
18 measured 24 hours after acrylamide dosing.

19          A couple of concerns that I have on that.  One is  
20 the recovery of urinary metabolites in this study was  
21 34 percent for humans and 50 percent for rats.  Now, I know  
22 we're talking about hemoglobin adducts.  But my concern is  
23 that they may not have formed sufficient number of hemoglobin  
24 adducts from glycidamide.  And if we can go back one slide.  
25 Let me explain.

26          This is the actual data from Fuhr that was used for  
27 creating the human model.  But I just want to point out the  
28 different relationships that you can see here for the

1 glycidamide adduct, which is the bottom one versus the  
2 acrylamide conjugates in the urine at 24 hours.

3           So what you can see is for the acrylamide, the top  
4 two, this is acrylamide glutathione conjugates in the urine.  
5 By 24 hours it's not changing in the top. It's almost  
6 complete for the middle one, but if you look at the third one,  
7 that's for glycidamide urinary adducts. They are still  
8 increasing with time. And, in fact, in many cases it's only  
9 50 percent recovery at 24 hours compared to 72 hours, which to  
10 me implies that there's more glycidamide in the human which  
11 hasn't been excreted totally.

12           And therefore, when we consider the use of the PK  
13 factor for rats, it may have some uncertainties, inaccuracies.  
14 The 1.2 has been used by -- OEHHA used it, but I don't think  
15 anyone has looked at this kind of consideration. Are the  
16 glycidamide concentrations fully accounted for in the  
17 estimation of the glycidamide-hemoglobin adducts for the area  
18 under the curve.

19           So if you go to the next slide, with 34 percent  
20 recovery, I have some concern that we may not have a full  
21 determination of the -- we're using the hemoglobin adducts as  
22 a surrogate for glycidamide concentrations that we may not  
23 have fully evaluated in this particular study for phenyls, the  
24 phenyl paper may not have fully evaluated the glycidamide  
25 concentration to hemoglobin by stopping their study at  
26 24 hours.

27           THE COURT: At this time we'll take the afternoon  
28 recess for 15 minutes.

1           You can step down, Dr. Melnick. And I'm going to  
2 call another case.

3           (Recess taken.)

4           THE COURT: All right. Back on the record.

5           Counsel, you may proceed.

6           MR. METZGER: Thank you, your Honor.

7           Q. Dr. Melnick, thank you for the explanation  
8 regarding the pharmacokinetic modeling.

9           Now what I'd like to ask you is if you've assessed  
10 the effect from Dr. Rhomberg's use of pharmacokinetic factors  
11 instead of body weight scaling on the derivation of the no  
12 significant risk level.

13           A. Certainly. I hope it's in the next slide.  
14 Yes, that looks like it.

15           Q. All right. So let me first ask you to address  
16 how the use of the PK factors versus body weight scaling  
17 affected cancer potency.

18           A. Okay. That is the second column, labeled  
19 potency, with the cancer slope factors for animals compared  
20 and converted to the cancer slope factor for human equivalent  
21 dose.

22           So the values in that particular set there indicate  
23 how the PK factor influences potency of comparing body weight  
24 scaling versus PK factor.

25           And in each of these cases you can see there is a  
26 decrease in cancer potency, for male rat from 3.6 to 2.3.

27           Female rat, 4.0 to 2.4.

28           Male mouse, 6.1 to 1.3.

1           And female mouse, 7.3 to 1.9.

2           So in each case potency is reduced by using a PK  
3 factor instead of body weight scale.

4           Q.    And how did the use by Dr. Rhomberg of the PK  
5 factors that he selected affect the no significant risk level  
6 compared to body weight scaling?

7           A.    So in each case here, the reversal, with less  
8 potency there's an increase in the NSRL.  And it's showing  
9 NSRLs at one times 10 to the minus 5.  That there is an  
10 increase with the body weight scaling compared -- with the PK  
11 factor compared to body weight scaling.  And that ratio is  
12 shown in the fourth column, what is the effect on NSRL.

13           And this would be the ratio of that previous column  
14 for the PK factor relative to the body weight scaling  
15 adjustment.  And you can see for the male rat, the PK factor  
16 is increased 50 percent.  Same with the female rat.

17           Q.    The NSRL or the PK factor?

18           A.    This is the NSRL.  The effect of applying a PK  
19 factor instead of body weight scaling on the NSRL.  So there's  
20 a 50 percent increase in the NSRL based on male rat and female  
21 rat tumor responses.  But with the mouse, it's more than a  
22 fivefold increase for the male mouse and a three and a half  
23 fold increase for the female mouse.

24           So these are substantial increases.

25           Q.    On the NSRL?

26           A.    On the NSRL, yes.

27           Q.    Okay.  And did you assess -- oh, by the way,  
28 before we leave that, this is the NSRL which is one cancer per

1 1,000, correct?

2 A. That is correct.

3 Q. And Dr. Rhomberg calculated an ASRL by  
4 multiplying the NSRL by 10; is that correct?

5 A. To make an adjustment of one per hundred  
6 thousand or 10 to the minus 5 to one times 10 to the minus 4,  
7 it is a multiple of 10, yes.

8 Q. All right. So for Dr. Rhomberg's ASRL, the  
9 effect of the body of the pharmacokinetic factor instead of  
10 body weight scaling, that would be a 15-fold increase for the  
11 male rat and the female rat?

12 A. Right. So I showed this as based on one per  
13 hundred thousand, if you multiply that by 10, that would be  
14 the consequence at one times 10 to the minus 4.

15 Q. All right. And have you also assessed the  
16 effect on the NSRL of using the tumor sites and body weight  
17 scaling?

18 A. Yes. So this is a combination of both of these  
19 factors which I've been discussing this afternoon.

20 Q. What two factors, please, for the record?

21 A. I'm sorry. Including all tumor sites as  
22 opposed to excluding tumor sites and using body weight scaling  
23 as opposed to a PK factor.

24 Q. Right. So if you include all tumor sites and  
25 apply a body weight scaling from animal potency to human  
26 potency, the NSRL would be derived would be 1.0 for male rat,  
27 .8 for female rat, .3 for male mouse, and .4 for the female  
28 mouse. This is at the one times 10 to the minus 5 cancer

1 risk.

2 Now, the next column is the NSRLs that Rhomberg --  
3 Dr. Rhomberg has proposed for risk at one times 10 to the  
4 minus 5, not his one times 10 to the minus 4. But in this  
5 case it's based on the exclusion of sites.

6 So I just want to look at what is the effect if we  
7 include all sites versus Rhomberg -- Dr. Rhomberg's exclusion  
8 of sites, and use body weight scaling.

9 You can see the effects on the NSRL for the male rat  
10 is approximately twofold higher at 10 to the minus 5 risk.

11 4.4 for the female rat.

12 25 for the male mouse?

13 25 fold?

14 A. 25 fold.

15 And 7.8 for the female mouse. The consequence of  
16 this is that the male mouse or the female mouse are no longer  
17 the most sensitive species for the risk assessment. The rat  
18 has become, by Dr. Rhomberg's calculations, the sensitive  
19 species for calculating risk.

20 Q. And what is the import of that?

21 A. Well, as I indicated, I don't see a basis for  
22 excluding tumor sites. And I don't see a basis for applying a  
23 pharmacokinetic factor; however, if you do that, you are  
24 decreasing the potency of the response, but most important is  
25 leading to an increase in the NSRL by quite a substantial  
26 number, especially for male mice.

27 Q. Okay. All right. So let's talk now about the  
28 quantitative risk assessment. And was there a particular part

1 of the final statement of reasons adopted by OEHHA regarding  
2 quantitative risk assessment that you considered to be  
3 important in your analysis?

4 A. Yes. This is in the addendum to the final  
5 statement of reasons.

6 Q. And what is the risk?

7 A. That the necessity is to show that a beneficial  
8 health effect outweighs the risks. That is the requirement as  
9 stated within the final statement of reasons. If that cannot  
10 be done, then the application of the one times 10 to the  
11 minus -- something different than 10 to the minus 5 is not  
12 available. That one times 10 to the minus 5 then becomes the  
13 standard unless the health benefits can be demonstrated to  
14 outweigh any health risks.

15 Q. Okay. And do you have opinions on health  
16 benefits and health risks that we're going to talk about?

17 A. We can talk about that.

18 Q. Okay. All right. So first of all, is there a  
19 methodology for quantitatively assessing health benefits and  
20 health detriments of a food?

21 A. A methodology has been written into the  
22 literature in 2012. It's called by the acronym BRAFO, or  
23 benefit risk analysis for foods.

24 MR. KENNEDY: Object and move to strike. The  
25 benefit analysis has already been done by the agency.

26 THE COURT: Objection overruled.

27 Q. BY MR. METZGER: All right. And who did this?

28 A. Who developed this methodology?

1 Q. Yes.

2 A. Okay. This was a project funded by the  
3 European Commission coordinated by the International Life  
4 Sciences Institute. I've served as a reviewer for some of the  
5 European Commission projects. What they tried to do is  
6 identify an important health issue and encourage investigators  
7 from different countries within the European union to develop  
8 an approach to answer that particular question. And that's  
9 what was done. And it was titled then How to Perform a  
10 Benefit Risk Analysis For Foods.

11 Q. And has this methodology been subjected to  
12 publication and peer review?

13 A. Yes, this was published in the peer-reviewed  
14 literature.

15 Q. All right. Could you explain to the Court what  
16 this BRAFO technology or methodology is that quantitatively  
17 assesses health benefits and detriments?

18 A. It's basically a comparison of health risks and  
19 health benefits of a reference condition. And I provide this  
20 as the reference scenario being coffee at the current  
21 acrylamide levels.

22 And the alternative would be coffee at reduced  
23 acrylamide levels.

24 So the comparison to look for this analysis is to  
25 see how these match up. For example, it's a four-tier  
26 process. If there's no benefits from the alternative, that  
27 would be, for example, reducing acrylamide in coffee, then why  
28 do it? The reference would be advised.

1           However, if there are only benefits with the  
2 alternative, removing acrylamide from coffee, then that is the  
3 preferred scenario.

4           So it's a qualitative determination. Are there  
5 benefits from the alternative or not. And if there are  
6 benefits, then we can -- and that's all, then we can stop at  
7 Tier 1.

8           However, if risks dominate benefits from the  
9 alternative, then the reference is advised. So when you have  
10 benefits and risks, if the risks are greater from the  
11 alternative, removing acrylamide from coffee, then you stay  
12 with the current -- the reference.

13           However, if benefits of the alternative dominate the  
14 risks, then the alternative is preferred. So it's comparing  
15 benefits and risks for two different scenarios.

16           It then can get more complex in the assessment  
17 because what if there's both risks and benefits. And this now  
18 goes into quantitative analyses because now we need to find  
19 some parameter values that allow a comparison of benefits and  
20 risks.

21           So, for example, a willingness to pay to avoid an  
22 adverse disease is a numerical value that might be obtainable  
23 from this comparison or to avoid -- or a disability, how many  
24 changes in the quality adjusted years or avoiding adverse  
25 health disabilities.

26           So you start to look at what are you gaining and  
27 what are you losing between the two with respect to quality  
28 and disabilities and see if one dominates the other in a

1 quantitative way.

2 Q. Let me ask you, under Tiers 3 and 4, which  
3 involve quantitative integration of risks and benefits, could  
4 one do an analysis using the BRAFO methodology where you,  
5 apart from just acrylamide, but where you would quantify  
6 health benefits of coffee, if there are any, and health  
7 detriments of coffee?

8 A. That could certainly be done.

9 Q. So this methodology could be used to do that.

10 A. Right, yes.

11 Q. Okay. And could you tell us -- give us some  
12 examples of how this methodology has been used to quantify  
13 health benefits and detriments of foods?

14 A. So in one of the publications from the BRAFO  
15 work -- and there were probably about five publications that  
16 came out. One of them conducted a benefit/risk analysis for  
17 potatoes and cereal products for acrylamide mitigation, and  
18 that they conclude is the reference versus the use of  
19 asparaginase to reuse acrylamide levels.

20 So it's something similar to what we're talking  
21 about with respect to coffee. How do the benefit risks  
22 compare in potatoes and cereal products before removing the  
23 acrylamide or after you remove it, reduce the asparaginase. I  
24 believe the reductions that they were considering were only  
25 about 30 percent.

26 And that group concluded that you can stop at Tier  
27 1. You don't need to go to Tier 2, 3, or 4 because of the  
28 beneficial effects of reducing acrylamide in processed foods.

1 And that was it.

2 So Tier 1, if you recall I mentioned, if there are  
3 only held benefits with the alternative, then why go further.  
4 And that's what they concluded, and I agree. For potatoes and  
5 cereal products, there is a benefit.

6 But they conclude that reducing acrylamide reducing  
7 actions should be applied as long as any adverse side effects  
8 are recognized and minimized to the extent possible. And that  
9 makes public health sense.

10 So the fact that that can be done in terms of making  
11 this comparison with potato and cereal products, I conclude  
12 that that same analysis is applicable to acrylamide in coffee.

13 Q. Okay.

14 A. And that hasn't been done. There has been no  
15 BRAFO analysis conducted by the defendants for acrylamide in  
16 coffee.

17 Q. Okay. All right. So now let's talk about  
18 sound considerations of public health and the use of a 10 to  
19 the minus 5 standard or a 10 to the minus 4 cancer risk  
20 standard.

21 What have you taken into account in answering that  
22 question?

23 A. Well, I don't think I need to state, but it's  
24 on my slide that I prepared, is that obviously cancer is a  
25 devastating disease. It's costly. We know from records that  
26 the number of new cancer cases per year in the United States  
27 is 1.7 million. 176,000 in California. And there's a big  
28 cost for health care, lost wages, and caregiving. 230- to

1 \$300 billion per year. So cancer is a disease we would  
2 attempt to reduce to the incident possible. And myself coming  
3 from an environmental cancer program, this is one which I feel  
4 very strongly about, is cancer prevention. The diet is linked  
5 to about 30 to 35 percent of human cancers --

6 Q. Let me ask you, that's about a third of human  
7 cancers, and I think 40 percent of humans or something get  
8 cancer?

9 THE COURT: Did you attempt to limit that to those  
10 areas of cancer that could increase risk? In other words,  
11 some specific cancers that were associated according to what  
12 you said with acrylamide. The statement about cancer. Was it  
13 limited to that? Or just cancer generically.

14 THE WITNESS: This is a general survey of cancer  
15 rates within the United States and separate for states.

16 Q. BY MR. METZGER: So you've indicated here that  
17 the diet is linked to about a third of human cancer. Where  
18 does that come from?

19 A. There are reports in the literature where  
20 people have made these types of estimations. In terms of  
21 whether it's genetic factors, lifestyle habits, obesity. But  
22 in these types of estimates, the diet was linked with  
23 approximately 30 to 35 percent of human cancers, that there  
24 are dietary components which are linked to increased cancers.

25 Q. And what are these dietary components or  
26 constituents that are linked to human cancer?

27 A. Well, it's probably pretty complex because it  
28 might be carcinogens in food. It might be high lipid -- high

1 fat diets can contribute. There's just an array of factors,  
2 but one of those factors would be environmental contaminants.

3 Q. All right. So tell us -- you had some articles  
4 about primary prevention of cancer. We discussed that  
5 earlier. How does that play into this puzzle here?

6 A. Well, as I mentioned, the National Toxicology  
7 Program where I worked conducts studies to identify agents  
8 that can cause cancer. And this information then is used by  
9 regulatory agencies to set limits on exposure of the public.  
10 And this is identified as primary prevention. Prevent the  
11 cancer from developing. And one way of doing that is reducing  
12 or eliminating exposure to those agents that cause cancer.  
13 And by doing that, it's serving as a public health protective  
14 approach.

15 Q. And in the human diet, what constituents or  
16 what are the carcinogens that are prevalent in the human diet?

17 MR. KENNEDY: Foundation.

18 THE COURT: Overruled.

19 THE WITNESS: Okay. Well, there are those caused by  
20 overcooking meats. Processed meats are contributors. There  
21 could be acrylamide as a factor. I'm trying to think of some  
22 of the others offhand.

23 Q. BY MR. METZGER: Is acrylamide the most  
24 prevalent carcinogen in the human diet?

25 MR. KENNEDY: Lack of foundation.

26 THE COURT: Overruled.

27 THE WITNESS: It's very prevalent in the human diet.  
28 And of which, I believe, 40 percent for adults of acrylamide

1 exposure comes from coffee.

2 Q. BY MR. METZGER: All right. So let's talk  
3 briefly about the carcinogenicity of acrylamide. And is that  
4 something that you considered in forming your opinion  
5 regarding sound considerations of public health?

6 THE COURT: Before we go there, let me ask you this:  
7 Did you attempt to eliminate all of those other cancers caused  
8 by foods other than coffee from all these statistics?

9 THE WITNESS: No, I haven't. I don't think  
10 anybody's done that.

11 THE COURT: Mr. Metzger.

12 Q. BY MR. METZGER: All right. So what is your  
13 assessment of acrylamide as a carcinogen in the context of  
14 sound considerations of public health?

15 A. Okay. Well, based on my experience and  
16 knowledge with epoxide-forming chemicals, I consider it of  
17 high concern for acrylamide-induced cancers. This was  
18 evaluated by IARC the last time, I believe, in 1994 and termed  
19 a probable, probably carcinogenic to humans. This was based  
20 on sufficient evidence in animals and, as I mentioned, those  
21 were the Johnson and Freedman studies prior to the NTP  
22 publication of their studies.

23 And part of the reason for this was many times a  
24 carcinogen is active at one site. For acrylamide it's  
25 carcinogenic at multiple sites in both sexes of two species,  
26 rats and mice.

27 When a compound does that, it's likely to be also  
28 carcinogenic in humans. However, there is inadequate evidence

1 for the carcinogenicity of acrylamide in humans. The data  
2 don't exist. And consequently, IARC classified this as  
3 probably carcinogenic to humans.

4 And in the IARC review process, what they include,  
5 when inviting participants, is there's four groups. One  
6 related to exposure. One evaluates the epidemiological data.  
7 One evaluates the animal cancer data. And the fourth one  
8 makes considerations of mechanistic information.

9 Even at that time in 1994, it was known that  
10 acrylamide and its metabolite, glycidamide, were both known  
11 that they form covalent DNA adducts in mice and rats.  
12 Acrylamide in glycidamidic form, covalent adducts with  
13 hemoglobin in humans and in rats.

14 So this shows that glycidamide is distributed  
15 systemically in exposed humans, and I believe even one of the  
16 studies that looked at urinary metabolites, I believe it was  
17 the phenyl study, found glycidamide excreted in the urine.

18 So this is a compound that I have large concerns for  
19 because of what it can do, that it is being systemically  
20 distributed in humans, in the body of humans. And it induces  
21 gene mutations and chromosomal aberrations in germ cells as  
22 well as somatic cells in mice or rats. So this is a bad  
23 compound.

24 Q. Okay. Tell us, if you would, based on your  
25 research and your publications regarding epoxide chemicals,  
26 their effects in different tumors in animals.

27 A. Okay. Well, there are certain sites, as  
28 mentioned in my 2002 paper, where epoxides tend to form tumors

1 in animal models, rats or mice. And this commonality also  
2 shows up for acrylamide and glycidamide.

3 So the mammary gland in rats, this was the site that  
4 was used in the FAO/WHO risk assessment, is a site which is  
5 vinyl chloride in 1,3-butadiene induced tumors, and  
6 1,3-butadiene are established human carcinogens.

7 The mammary gland in mice was a site of tumor  
8 induction by acrylamide and glycidamide.

9 The NTP conducted a study of acrylamide but also  
10 conducted a study at that same time of glycidamide. So when I  
11 show glycidamide up here, that is based on a separate study of  
12 glycidamide in rats and mice.

13 So the mammary gland was a target for both  
14 acrylamide and glycidamide, but the mammary gland in mice is  
15 also a target for vinyl chloride, 1,3-butadiene and ethylene  
16 oxide. Ethylene oxide is a Group 1 known human carcinogen.

17 And as we discussed earlier on the harderian gland,  
18 this was the site used by EFSA for their acrylamide risk  
19 assessment. Tumors are induced again by butadiene, ethylene  
20 oxide, glycidamide, acrylamide, as well as other  
21 epoxide-forming chemicals.

22 And lastly, in the lung of mice. Both acrylamide  
23 and glycidamide induced tumors in the mouse lung, but so does  
24 vinyl chloride, butadiene, ethylene oxide. So the picture I'm  
25 trying to demonstrate here is glycidamide or exposure to  
26 acrylamide which produces glycidamide is causing tumors  
27 similar to known human carcinogens that are either epoxides or  
28 metabolites to epoxides.

1           So we're seeing a lot of similarity between the two,  
2 which leads me to believe that this is a likely, very likely  
3 human carcinogen.

4           Q.    And based on this, is it your opinion that  
5 tumors of the mammary gland, the rats, the mice, and the  
6 harderian gland should be included in human cancer risk  
7 assessments for epoxide chemicals?

8           A.    Definitely.  Because as I mentioned before, for  
9 the harderian gland, I consider this to be the canary in the  
10 coal mine for epoxide-forming chemicals.  I assume everybody  
11 knows what that means.

12          Q.    Okay.  Thank you, Dr. Melnick.

13          Let's change topics and talk about the FDA guidance  
14 for industry acrylamide in foods.  You've reviewed that,  
15 correct?

16          A.    Yes, I have.

17          Q.    Okay.  And what was your assessment of that  
18 publication?

19          A.    Well, first of all, there were a number of  
20 statements that the FDA made that I think are relevant to this  
21 case.  So, for example, reducing acrylamide in foods may  
22 mitigate potential human risks from exposure to acrylamide.

23          So the FDA is recognizing that there are potential  
24 health risks, and reducing that would be a valuable  
25 consideration.

26          However, they also indicate, and this has been cited  
27 in this case a number of times.  FDA is not aware of any  
28 proven mitigation measures for acrylamide in food and that a

1 viable commercial process is not yet available.

2 Q. I think you said food instead of coffee.

3 A. Oh. I'm sorry. I meant coffee. I guess I  
4 can't read that well from here. I meant coffee.

5 Q. Okay.

6 A. Okay. My criticism of this is that the  
7 statements in that second bullet are based on outdated  
8 sources. One was from an article by Seal in 2008. And the  
9 other is the coffee industry's tool box or the tool box for  
10 industry on acrylamide for the Food Drink Europe, what they  
11 use as their source of information for making this kind of  
12 statement.

13 I went through this morning a number of mitigation  
14 approaches. And those don't seem to have made it into the  
15 FDA's guidance for industry. And there may be an explanation  
16 in part for this.

17 Q. What is that?

18 A. Well, I believe that the FDA concealed  
19 information from the FDA in 2000 --

20 Q. The FDA? I'm sorry?

21 A. The Nestle Company concealed information from  
22 FDA in a meeting which, from a document written by one of the  
23 coffee producers, this was Mwangi, M-W-A-N-G-I, that the  
24 purpose of the meeting was to persuade FDA to not set  
25 regulatory limits for acrylamide in coffee and told their  
26 managers that we would not divulge any data that would be  
27 damaging to us.

28 So to me the suppression of information might have

1 had some bearing on FDA not being aware of mitigation  
2 measures.

3 MR. KENNEDY: Object. Move to strike as sheer  
4 speculation.

5 THE COURT: Motion granted. No foundation for the  
6 witness's statements. The whole answer will be stricken.

7 MR. KENNEDY: Object. No foundation, ask that the  
8 answer be stricken.

9 THE COURT: I just said that. The answer is  
10 stricken.

11 MR. KENNEDY: Okay. I'm sorry. I thought you were  
12 telling me to say the magic words.

13 THE COURT: I'm sorry?

14 MR. KENNEDY: I'm sorry. I thought you were telling  
15 me to say the magic words. I thought I had.

16 THE COURT: No. I granted the motion to strike.

17 Q. BY MR. METZGER: Dr. Melnick, did you review  
18 some confidential documents in this case that were produced  
19 which indicated that there was a meeting between Nestle  
20 managers and the FDA and that the Nestle folks decided that  
21 they would not disclose information to the FDA at that  
22 meeting?

23 A. Yes, that --

24 MR. KENNEDY: Object, your Honor. Lack of  
25 foundation, multiple levels of hearsay --

26 THE COURT: Let me hear the answer.

27 THE WITNESS: That meeting was described in these  
28 confidential documents that I received, that the meeting

1 occurred in 2010 with Stadler and Mwangi. The meeting had  
2 representatives from the FDA. But they wrote their purpose,  
3 which I've already stated, but it was divulged in their  
4 documents where Mwangi was describing his accomplishments for  
5 the year.

6 THE COURT: Dr. Melnick, were you at any of these  
7 meetings?

8 THE WITNESS: No, I wasn't.

9 THE COURT: All right. The answer is stricken.

10 Q. BY MR. METZGER: So now let's talk about  
11 another of the defense's favorite documents, the USDA  
12 Scientific Committee report, or the Dietary Advisory  
13 Committee.

14 Have you reviewed that, Dr. Melnick?

15 A. Yes, I have.

16 Q. And what have you considered in that report  
17 with respect to sound considerations of public health for  
18 coffee consumption?

19 A. Okay. That document writes that moderate  
20 coffee consumption can be incorporated into a healthy dietary  
21 pattern, along with other healthful behaviors.

22 That statement is based on observational studies in  
23 healthy individuals. Observational studies have limitations  
24 in terms of their adequacy for determining causation. So it's  
25 simply a statement that this is what they believe. However,  
26 they do raise concerns about caffeinated coffee consumption by  
27 pregnant women, children, and adults and adolescents or other  
28 vulnerable individuals. And they also recommend minimizing

1 cream and sugar consumption. And they even make the comment  
2 that individuals who do not consume caffeinated coffee should  
3 not start to consume it.

4           So to me, if there's a health benefit, why would  
5 they recommend not consuming it for any type of health benefit  
6 if it doesn't exist?

7           Q.    Okay. Now, regarding this statement that  
8 moderate coffee consumption can be incorporated into a healthy  
9 dietary pattern, does the FDA also say that soft drinks,  
10 sodas, sugar sweetened beverages can be -- in moderate  
11 consumption can be incorporated into a healthy diet?

12           A.    Yes, they do.

13           Q.    Do they also say the same for alcohol?

14           A.    Yes.

15           Q.    So now let's talk a little about the FDA and  
16 coffee. First of all, has the FDA ever authorized any health  
17 claim for coffee?

18           A.    I was not able to find any health claim. And  
19 we searched for those, and, in fact, EFSA rejected health  
20 claims for coffee. There's no evidence that any government  
21 agency has concluded that drinking coffee prevents cancer or  
22 any chronic disease. And in reading the testimonies of  
23 Dr. Kessler and Dr. Alexander, they also concluded that coffee  
24 does not prevent any disease.

25           So there's no evidence for supporting health claims  
26 in any sources that I'm aware of.

27           But one thing that concerns me about this document  
28 from the USDA scientific report, the Dietary Guideline

1 Advisory Committee, is that the issue of acrylamide in food,  
2 as he mentions, has been known since 2002. There have been  
3 hundreds of papers regarding health concerns for acrylamide in  
4 food. And when I looked into this document and try to search  
5 for acrylamide to see how this Dietary Guideline Advisory  
6 Committee would react to the presence of acrylamide in foods,  
7 I found that it wasn't there.

8           There's no comment in this report regarding  
9 acrylamide in foods. And obviously, if there's no comment  
10 about it, there's no recommendation on an acceptable risk  
11 level for this carcinogen in coffee.

12           So this report is totally silent on issues related  
13 to acrylamide in foods and human health.

14           Q. Okay. So let's talk about the FDA and its  
15 regulation of carcinogens.

16           Have you reviewed over the years the Food, Drug, and  
17 Cosmetic Act, how that addresses carcinogens in food?

18           A. I've seen it. I haven't gone thoroughly  
19 through reviewing it. But I'm aware of it from just  
20 experience because, as I mentioned, the NTP is made up of  
21 several agencies. FDA is one of the agencies that's part of  
22 the NTP. So I'm aware that the Delaney clause prohibits FDA  
23 from adopting regulations that allow carcinogenic food  
24 additives. And their policy for regulating carcinogens is one  
25 per million. One times 10 to the minus 6.

26           However, I'm aware of a couple of rare exceptions  
27 that FDA has regulated carcinogens to allow more than one  
28 cancer per 10,000.

1 Q. Okay. And let's talk about those rare  
2 circumstances. What are those?

3 A. Well, the circumstances, first, are that the  
4 food has proven to have a health benefit. And there is no  
5 practical way of reducing those carcinogens in the food. The  
6 chemicals, and these were mentioned by Dr. Kessler, of what  
7 FDA has done. They have adopted a level of seven times 10 to  
8 the minus 5 for PCBs, polychlorinated biphenyls, in fish. And  
9 that's because fish contains omega 3 fatty acids which have  
10 been established as effective in reducing coronary heart  
11 disease. However, PCBs have been banned since approximately  
12 1980.

13 So they are not being produced into the environment  
14 anymore. But PCBs are very stable. So they are in the  
15 environment, but the PCBs become incorporated into the fat or  
16 lipid components in tissue, and they can remain there and are  
17 essentially impossible to get out unless you removed the  
18 source of the fat within the fish.

19 So if you pick up a fish which has PCBs, you can't  
20 set -- it can't be eliminated. It's an issue that is  
21 unsolvable at this time.

22 Q. So what is your understanding as to why the FDA  
23 allowed a higher carcinogenic risk for PCBs in fish?

24 A. They had --

25 MR. KENNEDY: Object. Lack of foundation.

26 THE COURT: Overruled. You may answer.

27 THE WITNESS: They had an identified health benefit  
28 which was recognized. The reduction of coronary heart disease

1 and the condition in which it could not be removed.

2           So in wanting people not to avoid fish, they adopted  
3 a level of higher risk. How they came up with 7.2 times 10 to  
4 the minus 5 I really don't know.

5           Q. BY MR. METZGER: Okay. And the other example  
6 that Dr. Kessler mentioned was arsenic in rice. And what is  
7 your understanding of how that came about?

8           A. This was for whole grains, that they reduce  
9 cancer and coronary disease. So there's an established health  
10 benefit. However, arsenic is a naturally occurring element,  
11 and it can't be removed from rice. And the FDA adopted a  
12 level of 3.9 times 10 to the minus 5. But how they got to  
13 that number I don't know.

14           I think this is something related to your question  
15 earlier this morning. How do you select the number? I really  
16 don't know how FDA selected 7.2 and 3.9. It might be that  
17 that was a level that wasn't overly excessive and could be  
18 accommodated. But I really don't know.

19           Q. Dr. Melnick, are you aware of any other food  
20 that the FDA has allowed a cancer risk at 10 to the minus 4?

21           A. No, I'm not aware of any. I haven't seen  
22 anything like that.

23           Q. So are these, PCBs in fish and arsenic in rice,  
24 rare exceptions to the FDA's one in a million cancer risk  
25 policy?

26           A. These are definitely exceptions, and these are  
27 higher concentrations or risk levels than 10 to the minus 4,  
28 even for the PCBs in fish and arsenic in rice.

1           Q.    Okay.  You've also indicated here the EPA, how  
2 it has regulated acrylamide in water.

3                   And what is the significance of that to you?

4           A.    Well, the significance is that the regulation  
5 is at a half part per billion, which is close to the level of  
6 the NSRL for -- from consumption of coffee.  This has a use  
7 for clarifying potable water, for treatment in waste water.  
8 But again, it was one in which it could not be practically  
9 removed from drinking water.

10                   So EPA for carcinogens identifies what they call a  
11 maximum contaminant level goal and a maximum contaminant  
12 level.  The goal is zero.  But if you can't achieve it and  
13 they feel that it serves a purpose, they can establish a  
14 maximum contaminant level, and this would be the standard for  
15 acrylamide in water.

16           Q.    All right.  Thank you.  Now, in assessing sound  
17 considerations of public health, have you evaluated whether  
18 the epidemiologic studies regarding coffee consumption and  
19 cancer or chronic disease provide support for a health  
20 benefit?

21           A.    Well, there's no evidence right now available  
22 demonstrating an actual health benefit from coffee  
23 consumption.  Now, there have been observational  
24 epidemiological studies that have shown inverse relationships,  
25 but the FDA has already noted that observational studies  
26 cannot determine whether such an observed relationship is one  
27 in which the substance caused that reduction in disease or  
28 whether it's coincidence.  There's a reduction, but the basis

1 for it cannot be determined.

2 In contrast, intervention studies cannot  
3 establish -- in comparison to intervention studies, the  
4 observational studies cannot establish cause and effect. This  
5 is what FDA has addressed for a number of years.

6 Q. And are any of the epidemiologic studies  
7 regarding coffee consumption and cancer or chronic disease,  
8 are any of those intervention studies?

9 A. No. I don't think they can actually be done.

10 Q. Okay. All right. So in assessing sound  
11 considerations of public health, in your opinion is it  
12 important to consider both health benefits and health  
13 detriments?

14 A. Yes. That's similar to what I was talking  
15 about earlier with BRAFO. You consider both the benefits and  
16 the detriments in making a consideration for public health.

17 Q. Okay. And what have you concluded regarding  
18 any health benefit from coffee consumption?

19 A. There's no agency or expert that can conclude  
20 that coffee prevents any disease. The prevention of a disease  
21 would be a reflection of the health benefit. But there's no  
22 statements within the government or even in the defendants in  
23 this case demonstrating causation for reduction of disease.

24 Q. In the absence of any health benefit of coffee  
25 consumption, in your opinion do sound considerations of public  
26 health justify allowing acrylamide exposure in excess of the  
27 NSRL?

28 A. Well, I think that's the whole basis of this

1 consideration is that to consider it, it needs to demonstrate  
2 that there's a benefit. Without the benefit, in my view this  
3 does not justify allowing a level higher than the NSRL.

4 THE COURT: When you say allowing, are you talking  
5 about without a warning? You're not talking about  
6 prohibiting.

7 THE WITNESS: No.

8 MR. METZGER: Correct, your Honor.

9 Q. Right. Proposition 65 doesn't say you can't  
10 expose people to carcinogens even at high levels, right?

11 A. Right. It's a labeling act. It's not a  
12 banning act.

13 Q. Right. All right. So now let's talk about  
14 acrylamide in coffee. What is your assessment regarding  
15 acrylamide in coffee regarding sound considerations of public  
16 health?

17 A. As a strong proponent of primary prevention,  
18 reducing exposures to carcinogens can reduce what would be  
19 preventable cancers among the exposed population. I think you  
20 want to go forward a slide for the reading audience.

21 Q. Sure.

22 A. So in my view a sound policy, public health  
23 policy, this is what public health is all about, from the view  
24 of primary prevention, is to reduce the risk of diseases,  
25 cancers among the exposed population.

26 And my concern, which I think I've expressed enough  
27 today, is that acrylamide is not a good chemical. It is a  
28 genotoxic carcinogen. There's no doubt about that. And

1 earlier, when we're talking about the margin of exposure,  
2 again, this is -- I think I mentioned the dose associated with  
3 a 150 percent excess cancer risk compared to human exposure  
4 levels has been determined by both FAO/WHO, as well as EFSA,  
5 in saying that margin of exposure is too low. It signifies a  
6 high human health concern. And I share that concern that  
7 these agencies have expressed.

8 Q. Is there any health benefit to acrylamide in  
9 coffee?

10 A. No -- for acrylamide?

11 Q. Acrylamide.

12 A. No, there's no health benefit for acrylamide.

13 This is -- you know, back in the 60's, when people were  
14 treating individuals with -- who had cancer, they were using  
15 these kinds of compounds to destroy cancer cells. But what  
16 they found on a number of cases was that they got an increase  
17 in another type of cancer. Particularly non-Hodgkin's  
18 lymphoma.

19 So a number of the chemotherapeutic drugs that were  
20 used back in the 50's and 60's were these same type of  
21 electrophilic compounds. The mustards, et cetera. But I  
22 would not recommend using acrylamide as a health benefit, as a  
23 chemotherapeutic drug. There's better ones out there.

24 Q. Is acrylamide an essential constituent of  
25 coffee in your opinion?

26 A. No. It doesn't provide any value in coffee.  
27 It doesn't provide flavor. There's no nutritive value from  
28 acrylamide. That's for sure.

1           Q.    All right.  And in assessing sound  
2 considerations of public health, what significance do you  
3 attribute to the fact that, as you have testified, acrylamide  
4 concentrations in coffee can be reduced by about 90 percent  
5 without negatively affecting palatability?

6           A.    Well, therefore, the approach that I would  
7 recommend very strongly is because it can be selectively  
8 reduced, and it can be reduced without affecting significantly  
9 palatability, there's no reason why that approach should not  
10 be taken because it can be done.  It's doable.

11           So if you can remove the acrylamide, which I believe  
12 can be done, I would prefer that coffee had lower levels of  
13 acrylamide rather than having a label.

14           Q.    A cancer warning label, you mean.

15           A.    Yeah.  You know, I'm thinking from the public  
16 health perspective.  You know, I would prefer that people  
17 don't get exposure to acrylamide as opposed to reading the  
18 label and in some cases ignoring it because this is a compound  
19 which we want to reduce human exposure to.

20           Q.    And have you considered that for some people,  
21 even if they read the label, if they are dependent on caffeine  
22 in coffee, that they are going to drink it anyway because they  
23 feel compelled to?

24           A.    People will do that, yes.

25           Q.    So in your opinion, getting the acrylamide out  
26 is the best solution?

27           A.    That would be my preference.  Very strongly.

28           Q.    Just like the potato chip manufacturers got it

1 out of potato chips?

2 A. It can be done. So if it can be done, I would  
3 prefer to see it done.

4 Q. In any event, do you see any justification for  
5 allowing, devising a 10 to the minus 4 risk, allowing that  
6 much more cancer risk for acrylamide in coffee?

7 A. I see no justification for an alternative  
8 cancer risk for acrylamide in coffee. As I just stated, it  
9 can be removed. The potato industry was successful in  
10 reducing its levels. I think it can be reduced substantially  
11 without having large impact on palatability. And therefore,  
12 to me, saying the risk level could be one times 10 to the  
13 minus 4 seems to be an arbitrary value with no supportive  
14 rationale. There's no health benefit that can be identified  
15 by allowing a one times 10 to the minus 4 risk level.

16 Q. What is your ultimate conclusion? Is that it?

17 A. Well, no. I have one more what I consider a  
18 sound consideration for public health.

19 Q. What is that?

20 A. And that is the people of California expressed  
21 that in passing Prop 65. That was they want to find what are  
22 the hazardous chemicals that are posing threats to their  
23 health and well-being. And they were dissatisfied that the  
24 government agencies failed to provide them with adequate  
25 protection. They were asking for sound considerations of  
26 public health. That is why they declared their rights to be  
27 informed about exposures to chemicals that cause cancer, birth  
28 defects, or other reproductive harm. And they wanted to see

1 enforcement of those laws controlling the hazardous chemicals  
2 that threaten public health.

3 And to me, that is a strong expression of sound  
4 consideration for health that was expressed by the citizens of  
5 California.

6 MR. METZGER: All right. Thank you very much,  
7 Dr. Melnick.

8 THE WITNESS: Well, I have my conclusions.

9 Q. BY MR. METZGER: Oh. More conclusions?

10 A. No. Just my overall conclusions.

11 Q. Okay. What are your overall --

12 THE COURT: Do you have a question, Mr. Metzger,  
13 that you'd like to ask the witness?

14 MR. METZGER: Yes, I do.

15 Q. What are your overall conclusions, Dr. Melnick?

16 A. If you remember, I indicated at the beginning  
17 that the topics to be covered would include my overall  
18 conclusions, and these are them posted on the screen over  
19 there. That there's no health benefit from acrylamide in  
20 coffee and that the concentrations can be selectively reduced  
21 by significantly affecting the sensorial properties of coffee.

22 Because of the beneficial effect of reducing  
23 acrylamide in foods, similar to the BRAFO statement on  
24 potatoes, I believe an acrylamide reducing action should be  
25 applied to coffee as long as there's no further demonstration  
26 of adverse effects identified.

27 With respect to that pharmacokinetic adjustment,  
28 which I hope people were able to grasp, I find that

1 pharmacokinetic adjustments cannot be taken into account with  
2 confidence, and the exclusion of cancer sites produces  
3 significant increases in the NSRL for acrylamide in coffee.

4 I see no justification for supporting an alternative  
5 cancer risk level for this genotoxic carcinogen in coffee.

6 MR. METZGER: Thank you very much, Dr. Melnick.

7 THE COURT: Mr. Kennedy, are you going to have any  
8 questions? You don't have to do it today.

9 MR. KENNEDY: Okay.

10 THE COURT: I just want to make sure you had  
11 questions.

12 MR. KENNEDY: We're not going to pass.

13 THE COURT: Okay. We're going to resume the trial  
14 tomorrow morning at 9:00 o'clock.

15 MR. METZGER: Your Honor, could we just chat with  
16 you briefly about the remainder of the week?

17 THE COURT: Sure.

18 MR. METZGER: What your plans are.

19 THE COURT: Yes.

20 MR. METZGER: So I expect that Dr. Melnick will  
21 be -- his testimony will conclude tomorrow, on Tuesday.

22 And that leaves in question what is to be done for  
23 the remainder of the week. I don't know if your Honor has  
24 seen it. I have a new witness, a percipient witness that I  
25 just discovered, and I notified counsel and the Court about  
26 this witness. I'd like to have that witness testify on  
27 Wednesday. And that witness is willing to give a deposition  
28 before testifying, if your Honor feels that that's necessary.

1 THE COURT: Well, where did this witness come from?  
2 How come after seven and a half years you discovered a new  
3 percipient witness?

4 MR. METZGER: I put this all in my declaration.  
5 Your Honor probably hasn't seen it. Do you want to take a  
6 moment to read it, or should I give you a narration?

7 THE COURT: Why don't you give me a quick summary.

8 MR. METZGER: Okay. So on Friday I was contacted by  
9 a gentleman by the name of Harvey Durand, who is the president  
10 of Healthy Cafe, LLC, which actually is the assignee for a  
11 patent for reducing acrylamide in coffee. And he has informed  
12 me that he has some very significant information regarding  
13 this case.

14 That was just on Friday. And I immediately notified  
15 counsel this morning, the first court day after I discovered  
16 this.

17 I believe he has relevant information as a  
18 percipient witness to give, especially regarding the coffee  
19 industry's unwillingness to adopt or implement this technology  
20 even though it improved the flavor of coffee and specifically  
21 because it incidentally reduced the concentration of  
22 acrylamide.

23 So he has some percipient knowledge about this. I  
24 don't think his testimony will be very long. But he first  
25 came to my attention when he called me on Friday.

26 THE COURT: All right. And defendants?

27 MR. KENNEDY: If he's been able to keep this secret  
28 this long, I don't see how he thinks testimony is going to

1 bear on what the industry should have known since he probably  
2 hasn't come out of the woodwork until Friday. So we would  
3 object. If the Court is inclined to let him testify, we would  
4 request a deposition.

5 THE COURT: Well, I have concern at this late stage.  
6 Supposing next week the defendants discover a new witness. Do  
7 you think we ought to let this trail along like this? Each  
8 party coming up with new witnesses?

9 MR. METZGER: Well, your Honor, the only reason I  
10 learned about him --

11 THE COURT: I'm not blaming you. A guy comes out of  
12 the woodwork --

13 MR. METZGER: -- was because of the publicity from  
14 the case, that he contacted me. And I'll tell you, there's  
15 probably 50 people that contacted me. But this is the only  
16 one that I thought, oh, this gentleman actually has some  
17 relevant information because he's had conversations with  
18 executives of the coffee industry. And these are party  
19 opponent admissions that they don't want to do it because of  
20 the litigation.

21 THE COURT: He's just had conversations, and he's  
22 been around for a while. So all of a sudden, he wants to  
23 interject himself in this case. At any rate, I'll give  
24 counsel for the defendant an opportunity to file some papers  
25 tomorrow morning. But we'll discuss it tomorrow.

26 I'm a little concerned about having a new witness  
27 come forward, especially a witness who is introducing himself  
28 to the proceedings.

1                   We'll be in recess until tomorrow morning at 9:00  
2 o'clock.

3                   MR. METZGER: Thank you, your Honor.

4

5                   (Proceedings concluded at 4:25 P.M.)

6

7

8

9

10

11

12

13

14

15

16

17

18

19

20

21

22

23

24

25

26

27

28

1  
2  
3  
4  
5  
6  
7  
8  
9  
10  
11  
12  
13  
14  
15  
16  
17  
18  
19  
20  
21  
22  
23  
24  
25  
26  
27  
28

SUPERIOR COURT OF THE STATE OF CALIFORNIA  
FOR THE COUNTY OF LOS ANGELES

DEPARTMENT 323 HON. ELIHU M. BERLE, JUDGE

CERT, )  
) )  
PLAINTIFF, )  
) ) CASE NO. BC 435759  
VS. )  
) ) BC 461182  
STARBUCKS CORP, ET AL., )  
) )  
DEFENDANTS. )  
\_\_\_\_\_ )

I, MARK SCHWEITZER, OFFICIAL COURT REPORTER PRO TEM  
OF THE SUPERIOR COURT OF THE STATE OF CALIFORNIA, COUNTY OF  
LOS ANGELES, DO HEREBY CERTIFY THAT THE FOREGOING TRANSCRIPT,  
DATED OCTOBER 2, 2017, P.M. SESSION, COMPRISES A FULL, TRUE,  
AND CORRECT TRANSCRIPT OF THE PROCEEDINGS HELD IN THE  
ABOVE-ENTITLED CAUSE.

DATED THIS 2ND DAY OF OCTOBER, 2017.

/S/ MARK SCHWEITZER  
MARK SCHWEITZER, RPR, CRR, CSR NO. 10514

# **EXHIBIT “E”**

1  
1 SUPERIOR COURT OF THE STATE OF CALIFORNIA

2 FOR THE COUNTY OF LOS ANGELES

3 DEPARTMENT 323

HON. ELIHU M. BERLE, JUDGE

4  
5 CERT, )  
6 )  
7 ) Plaintiff, )  
8 ) vs. ) SUPERIOR COURT  
9 ) CASE NO. BC 435759  
10 ) BC 461182  
11 ) STARBUCKS CORP, ET AL., )  
12 )  
13 ) Defendants. )  
14 )  
15 )  
16 )  
17 )  
18 )  
19 )  
20 )  
21 )  
22 )  
23 )  
24 )  
25 )  
26 )  
27 )  
28 )

REPORTER'S TRANSCRIPT OF PROCEEDINGS

Tuesday, October 3, 2017

(A.M. Session)

APPEARANCES OF COUNSEL:

FOR THE PLAINTIFFS: METZGER LAW GROUP  
BY: RAPHAEL METZGER, ESQ.  
ABRAHAM I. PARISER, ESQ.  
401 East Ocean Boulevard  
Suite 800  
Long Beach, California 90802  
(562) 437-4499  
sbrust@toxictorts.com  
rmetzger@toxictorts.com  
apariser@toxictorts.com

FOR THE ROASTER AND DOE DEFENDANTS:  
MORRISON/FOERSTER  
BY: JAMES M. SCHURZ, ESQ.  
425 Market Street  
San Francisco, California 94105-2482  
(415) 268-7124  
jschurz@mofo.com

(Appearances continued on next page.)

DAVID A. SALYER, CSR, RMR, CRR  
Official Pro Tem Court Reporter  
License No. 4410

1 APPEARANCES OF COUNSEL: (CONTINUED)

2 FOR KEURIG: SKADDEN, ARPS, SLATE, MEAGHER  
& FLOM, LLP  
3 BY: RAOUL D. KENNEDY, ESQ.  
4 525 University Avenue  
Palo Alto, California 94301  
5 (650)470-4550  
rkennedy@skadden.com

6 FOR HN FERNANDEZ, ET AL.:

7 NORTON ROSE FULBRIGHT, LLP  
8 BY: JEFFREY B. MARGULIES, ESQ.  
555 South Flower Street  
9 41st Floor  
Los Angeles, California 90071  
10 (213)892-9286  
jmargulies@nortonrosefulbright.com

11  
12  
13  
14  
15  
16  
17  
18  
19  
20  
21  
22  
23  
24  
25  
26  
27  
28



1 CASE NUMBER: BC 411192/BC435759  
2 CASE NAME: CERT CASES  
3 LOS ANGELES, CALIFORNIA TUESDAY, OCTOBER 3, 2017  
4 DEPARTMENT 323 ELIHU M. BERLE, JUDGE  
5 REPORTER: DAVID A. SALYER, CSR 4410  
6 TIME: 9:00 A.M.

7 -o0o-

8 THE COURT: Calling the trial, CERT versus Starbucks.  
9 All counsel are present and Dr. Melnick is on the  
10 stand.

11 RONALD MELNICK,  
12 witness, resumed the stand and testified further as follows:

13  
14 THE COURT: Good morning, Dr. Melnick.  
15 You understand you're still under oath?

16 THE WITNESS: I understand that.

17 THE COURT: And Mr. Kennedy is going to proceed with  
18 cross-examination.

19 MR. KENNEDY: Your Honor, two housekeeping matters.

20 One, we've prepared binders with some -- I can't say  
21 all, but some of the documents that we're going to be using  
22 this morning.

23 I tried to put them in more or less the same order.

24 Secondly, I'm on some medication that may require me to  
25 ask the Court's indulgence for a bathroom break.

26 THE COURT: Any time you need a break, just give me a  
27 signal and we'll take a recess.

28 MR. KENNEDY: I appreciate that, your Honor. You get

1 old, these things happen.

2 THE COURT: That goes for any counsel and the witnesses  
3 and even the court reporter.

4 Counsel, you may proceed.

5

6

**CROSS-EXAMINATION**

7

BY MR. KENNEDY:

8

Q. Good morning, Dr. Melnick.

9

10 As you told us in your statement of opinions -- you  
11 told us in your statement of opinions while roasting coffee  
12 beans is necessary to make coffee products and to reduce  
13 microbial contaminants to some extent, the presence of  
14 acrylamide in coffee provides no health benefits.

15 THE COURT: Mr. Kennedy, could you just hold on one  
16 second.

17 I just want to open up the LiveNote on my computer.

18 MR. KENNEDY: Plaintiff's Exhibit 600 --

19 THE COURT: Mr. Kennedy, wait just one second.

20 All right. Thank you, Mr. Kennedy. You may proceed.

21 MR. KENNEDY: Yes, your Honor.

22 Q. Dr. Melnick, directing your attention to the  
23 screen and to Exhibit 60077, that's part of the opinions of  
24 Ronald Melnick that you submitted in this case; is that  
25 correct?

26 A. That's correct.

27 Q. Going to page 3 of Exhibit 60077, you say, in  
28 part, "While roasting coffee beans is necessary to make coffee  
products and to reduce microbial contaminants to some extent,

1 the presence of acrylamide in coffee provides no health  
2 benefits."

3 Correct?

4 A. That's correct.

5 Q. And you do agree that roasting coffee beans is  
6 necessary to make coffee products, correct?

7 A. That's correct.

8 Q. And going to page 8 of Exhibit 60077, you also  
9 say, "While acrylamide is formed as product of the Maillard  
10 reaction which produces many aromatic and flavorful chemicals,  
11 acrylamide itself is not an essential component of coffee."

12 Correct?

13 A. Yes, that's correct.

14 Q. So you agree that acrylamide is formed in the  
15 product from the Maillard reaction. No disagreement on that?

16 A. No disagreement on that.

17 Q. Then directing your attention to the  
18 demonstrative slides that you used yesterday, Exhibit 71356,  
19 slide four, and that's on the screen now, the "No Significant  
20 Risk Level" slide, correct?

21 A. That is correct.

22 Q. And directing your attention to the third bullet  
23 point that talks about "an alternative level must be supported  
24 by sound considerations of public health."

25 Then you give, "For example, where chemicals in food  
26 are produced by cooking necessary to render the food palatable  
27 or to avoid microbial contamination."

28 Do you see that?

1 A. Yes, I do.

2 Q. And then in the fourth bullet point you say, "If  
3 beneficial effects do not outweigh the risks, then the 10 to  
4 the minus 5 standard applies." And you cite addendum FSOR.

5 What are you referring to there?

6 A. The final statement of reasons.

7 Q. The addendum to the final statement?

8 A. The addendum, yes.

9 Q. And that's the final statement for 75203?

10 A. Yes.

11 Q. And then Exhibit 71356, the demonstratives, you  
12 also quote from the final statement at slide 43, do you not,  
13 where you say that, "The person responsible for the exposure  
14 must be able to show that the beneficial health effects of the  
15 additive outweigh the risks."

16 Correct?

17 A. Correct.

18 Q. And directing your attention next to  
19 Exhibit 71356, which was the more complete statement from the  
20 addendum.

21 And what they're talking about there is they explain  
22 the commentor who this is all pertaining to talks about  
23 chemicals that are intentionally added to a food product,  
24 correct?

25 A. Yes.

26 Q. And acrylamide is not something which is  
27 intentionally added to a food product, is it?

28 MR. METZGER: Objection. That's actually a legal

1 conclusion.

2 THE COURT: Overruled.

3 You may answer.

4 THE WITNESS: It's not intentionally added, correct.

5 Q. BY MR. KENNEDY: If you go into a roasting  
6 plant, there isn't a station that says here's where we  
7 intentionally add the acrylamide, is there?

8 A. No, there isn't.

9 Q. Okay. So the addendum is talking about the  
10 effect of intentionally added substances, which acrylamide  
11 isn't, correct?

12 MR. METZGER: Objection, legal conclusion.

13 THE COURT: Overruled.

14 You may answer.

15 THE WITNESS: But it is a chemical which has been  
16 included in the final product. But it is a consequence of  
17 roasting.

18 Q. BY MR. KENNEDY: No, no.

19 We're talking about, it is not something that's  
20 intentionally added?

21 A. That is correct.

22 Q. And the addendum has nothing to do with  
23 acrylamide, does it?

24 MR. METZGER: Objection, legal conclusion.

25 THE COURT: It's argumentative.

26 I think we've already established that there's not some  
27 product of acrylamide that's being added, that it happens in  
28 the process of roasting coffee.

1 MR. METZGER: And they intentionally roast coffee.

2 THE COURT: Let's move on.

3 Q. BY MR. KENNEDY: Sticking with the  
4 demonstratives from yesterday, Exhibit 61950, let's go, for  
5 example, to slide 14.

6 This talks about the effect of steaming and pressure on  
7 acrylamide levels, correct?

8 A. Yes, that's correct.

9 Q. A different way of preparing the beans?

10 A. Right.

11 Q. And similarly, slide 15 talks about the effect  
12 if you did vacuum roasting, correct?

13 A. Yes.

14 Q. And slide 16 talks about if you used heat  
15 curing, correct?

16 A. Correct.

17 Q. And 17 talks about supercritical extraction?

18 A. Correct.

19 Q. And 18 talks about cysteine addition?

20 A. Cysteine.

21 Q. Cysteine.

22 And at the time -- and you prepared these slides,  
23 didn't you?

24 A. Yes, I did.

25 Q. And at the time you prepared them, were you  
26 familiar with the final statement of reasons that we talked  
27 about here this morning already, correct?

28 A. Some of that was discussed over the weekend.

1 Q. Well, you've told us about the addendum to the  
2 final statement?

3 A. Oh, the statement of reasons? Yes.

4 Q. You read it. You're familiar with the  
5 statement?

6 A. Yes, sure.

7 Q. Going to Exhibit 71356, the May 1990 final  
8 statement of reasons, as you read that over and became  
9 familiar with it, you learned that the word "necessary" in the  
10 necessary cooking exception is not intended to favor one  
11 cooking practice over another.

12 If a food could be boiled or broiled to avoid  
13 contamination or render the food palatable but broiling  
14 produces more chemical byproducts than boiling, broiling does  
15 not become necessary --

16 MR. METZGER: Unnecessary.

17 MR. KENNEDY: Unnecessary, thank you.

18 "The agency's intention is that whatever method of  
19 cooking is chosen, the amount of cooking which is necessary to  
20 avoid bacterial contamination or to render the food palatable  
21 should provide a basis for the application of a risk level  
22 other than a risk of 1 times 10 to the minus 5."

23 Q. You saw that language, didn't you?

24 A. Yes, I have.

25 Q. And from that you concluded, did you not, that  
26 the particular method of cooking was really irrelevant to  
27 whether an ASRL would apply, didn't you?

28 MR. METZGER: Objection, legal conclusion,

1 argumentative. It's all roasting. So it's one method of  
2 cooking.

3 THE COURT: Objection overruled.

4 The witness may answer.

5 THE WITNESS: I concur that roasting is the method for  
6 preparing coffee.

7 Q. BY MR. KENNEDY: And if there's some other  
8 variation on how to render coffee beans palatable, that  
9 doesn't make any difference under the language we've just  
10 talked about, does it?

11 MR. METZGER: Objection, legal conclusion,  
12 argumentative.

13 THE COURT: Overruled.

14 The witness may answer.

15 THE WITNESS: I think some of the slides I presented  
16 and that you showed were variations on roasting or methods  
17 that could be used during roasting to remove acrylamide.

18 So they're not changing from roasting to boiling or  
19 broiling. I don't think that ever came up.

20 Q. You don't think the language we have up there  
21 says that the ASRL exception applies regardless of whether  
22 there are other methods that might produce less of the  
23 carcinogen? You don't think that's what it means?

24 MR. METZGER: Objection, argumentative, legal  
25 conclusion.

26 THE COURT: Overruled.

27 THE WITNESS: I think the variation in roasting is  
28 still a roasting process.

1 I think we're comparing roasting to boiling. I don't  
2 think anyone would, at this point, claim that boiling is an  
3 alternative. But roasting and applying supercritical CO2  
4 extraction is still roasting.

5 So I don't think the supercritical CO2 extraction  
6 deviates from these particular statements.

7 Q. BY MR. KENNEDY: And you'll agree it's his  
8 Honor's prerogative to decide what these words mean.

9 A. You asked me.

10 Q. And you're trying to do your best to help him  
11 reach the right answer; is that correct?

12 A. Certainly.

13 Q. Now, you spent a lot of time yesterday talking  
14 about various ways of mitigating acrylamide in coffee.

15 You recall that discussion, don't you?

16 A. Sure.

17 Q. And in the course of reading over the section  
18 75203 and the statement of reasons, you didn't find the word  
19 "mitigation" anywhere, did you?

20 A. I would have to look again to recall.

21 Q. You're assuming that there's a mitigation  
22 requirement, correct?

23 A. Oh, no, no. I believe mitigation is not a  
24 requirement.

25 Q. Okay.

26 A. It's a labeling act.

27 I think mitigation is an option that might be  
28 considered by the industry or facilities which are involved in

1 a Prop 65 case in terms of how to avoid labeling, but the act  
2 is a labeling act.

3 It doesn't indicate that the judge would necessarily  
4 say you must mitigate.

5 Q. Or say unless you mitigate you must put a  
6 warning label on it, right?

7 MR. METZGER: Objection, argumentative, legal  
8 conclusion.

9 THE COURT: Overruled.

10 THE WITNESS: No. I think the decision would be the  
11 level is above the NSRL. Under that condition a labeling  
12 would be required.

13 And I would imagine -- I would prefer to see acrylamide  
14 removed from coffee as opposed to labeling. And hopefully an  
15 interaction could occur such that the labeling could be  
16 avoided if there were attempts to remove or reduce  
17 substantially the acrylamide from coffee.

18 That would be my preferred finality to the situation.

19 THE COURT: Why do you think that's not happening?

20 THE WITNESS: You want my honest answer?

21 THE COURT: I hope your answer is honest.

22 THE WITNESS: My honest answer is it appears to me that  
23 the coffee company thinks that they can win on litigation and  
24 don't need mitigation.

25 THE COURT: Do you think there would be some  
26 competitive advantage for some innovator to come into the  
27 market with a coffee that has had acrylamide eliminated?

28 THE WITNESS: I think it would be a huge advantage.

1 THE COURT: So do you have any opinions as to why no  
2 one has done that?

3 You testified yesterday about this German company. Do  
4 you know of any information why the German company has not  
5 entered the American market?

6 THE WITNESS: I don't know why that hasn't happened.  
7 But I would imagine if there were two products side by side on  
8 a shelf, one had a label saying that this is known to the  
9 State of California to have a carcinogen, acrylamide, and side  
10 by side was another product that didn't have that label, I  
11 would imagine very strongly people would opt for the one that  
12 doesn't have that label.

13 Why that hasn't developed further, I don't know the  
14 reason, but I know the industry is quite united among most of  
15 the coffee roasters.

16 THE COURT: All right. Thank you.

17 Mr. Kennedy?

18 Q. BY MR. KENNEDY: Now, in terms of mitigation of  
19 acrylamide in coffee, you yourself have never devised a method  
20 for doing that, have you?

21 A. No, I haven't.

22 Q. You've never worked for a company that was doing  
23 that or trying to do it, have you?

24 A. No, I haven't.

25 Q. And outside of this case, you've never written  
26 or lectured on the reduction of acrylamide in coffee?

27 A. That's correct.

28 Q. And you've never visited a company that was

1 involved with trying to reduce acrylamide in coffee?

2 A. I didn't know of any companies that were trying  
3 to reduce acrylamide in coffee. I only know about it largely  
4 from the confidential papers that I received.

5 Q. And you learned about Novozymes from  
6 confidential sources?

7 A. You asked me in coffee. I did not know of  
8 coffee companies that were working on mitigating acrylamide.

9 Q. I'll try again.

10 You've never visited any company that you understood  
11 was working on reduction of acrylamide in coffee?

12 A. That is correct.

13 Q. Okay. And you've never drunk a cup of coffee  
14 that reflected anybody's attempted mitigation method, have  
15 you?

16 A. No, I haven't.

17 Q. So what you know about reduction of acrylamide  
18 in coffee is what you've learned working on this case,  
19 correct?

20 A. That is correct.

21 Q. And you've learned in the course of working on  
22 this case that the FDA doesn't think there's a commercially  
23 viable process for reducing acrylamide in coffee?

24 MR. METZGER: Objection. Vague as to time.

25 THE COURT: Objection sustained.

26 Can you pinpoint -- if there is a differentiation in  
27 time, then pin it down?

28 If not, at any time.

1 MR. KENNEDY: Sure, your Honor.

2 THE COURT: Fine tune that, please.

3 MR. KENNEDY: Sure.

4 Why don't we put up Defendants' Exhibit 71830, the  
5 guidance for industry document we talked about yesterday.

6 Q. You're familiar with that, aren't you?

7 A. Yes, I am.

8 Q. That's a March 2016 publication, correct?

9 A. That is correct.

10 Q. And that's a joint effort of the U.S. Department  
11 of Health and Human Services, correct?

12 A. Correct.

13 Q. And of the Food and Drug Administration?

14 A. Yes.

15 Q. And of the Center for Food Safety and Applied  
16 Nutrition, correct?

17 A. Yes.

18 Q. Let's go over to page 23.

19 As of March, 2016 they told us they did not -- I'm  
20 sorry.

21 If we went to page 27, it might be much better. My  
22 apologies.

23 Coming to the end of the second full paragraph, right  
24 before the, E, Properties and Cooking Interaction., the last  
25 sentence.

26 "A viable commercial process is not yet available,  
27 Reference 30."

28 That's what those three agencies had to say as of

1 March, 2016, correct?

2 A. That's what's written here.

3 Q. And let's go over to page 35 of that same  
4 document, 71830.

5 Let's go to reference 84.

6 And the reference is comments submitted by Novozymes.  
7 So apparently these three agencies were aware of something  
8 about Novozymes, correct?

9 A. I haven't seen those comments, so I don't know  
10 what they contain.

11 Q. Okay. In any event, you disagree with those  
12 three agencies, correct?

13 A. Yes, I do.

14 Q. You think there are at least two commercially  
15 viable ways of reducing acrylamide in coffee, correct?

16 A. I think there's more than two.

17 Q. Well, you told us about long-term storage and  
18 you've told us about Novozymes.

19 Are there more than that?

20 A. Curing.

21 There was another depending on the nature of the coffee  
22 product, cysteine.

23 I would have to look at my full list, but it's more  
24 than two.

25 Q. Okay. Let's go back to page 3 of the guidance  
26 exhibit.

27 And going to the black bordered box at the top.

28 Can we enlarge that, Tom.

1           This explains that the guidance represents the current  
2 thinking of the FDA, but you can use an alternative approach  
3 if it satisfies the requirements. And to discuss an  
4 alternative approach, contact the FDA staff responsible for  
5 this guidance.

6           Do you see that?

7           A.       Yes, I do.

8           Q.       You told us yesterday cancer is an absolutely  
9 horrible disease, right?

10          A.       Yes.

11          Q.       And you consider that acrylamide is a risk  
12 factor in causing cancer, right?

13          A.       Correct.

14          Q.       And you believe that with the adoption of the  
15 mitigation methods you've proposed here, that risk factor  
16 could be reduced, correct?

17          A.       That is correct.

18          Q.       Could save lives, correct?

19          A.       Correct.

20          Q.       And so you seem to have knowledge that the FDA  
21 doesn't have, correct?

22          A.       I don't know what knowledge the FDA has. All I  
23 know is that the sentence that you read to me was based on, as  
24 you read it, reference 30 which was a document from  
25 EuropeFoodDrink, which was prepared by the coffee companies.

26                So they have the information that the coffee companies  
27 provided to them.

28          Q.       Correct.

1           You came to the conclusion that the FDA was dealing  
2 with incomplete information, right?

3           A.       That is correct.

4           Q.       And that people were potentially dying because  
5 they didn't have complete information, correct?

6           A.       Potentially dying, yes.

7           Q.       Okay. And when did you first learn about this  
8 March, 2016 document?

9           A.       In the course of this case.

10          Q.       Six months ago, a year ago?

11          A.       Probably within the past six months to a year --  
12 eight months, somewhere in that range.

13          Q.       And tell us everything you've done during those  
14 intervening six to eight months to try to call the FDA's  
15 attention to the fact that there are potentially lifesaving  
16 methodologies out there that for some reason they don't know  
17 about.

18                 Do you find that funny, doctor?

19          MR. METZGER: Objection, argumentative.

20                 He's under a protective order that he cannot disclose  
21 the confidential documents to the FDA.

22          THE COURT: Well, the question as phrased is  
23 argumentative.

24                 But the witness can answer the question as to whether  
25 he's had any communications with the FDA about any new  
26 processes.

27          MR. KENNEDY: Do you want me to rephrase, your Honor?

28          THE COURT: Yes.

1 Q. BY MR. KENNEDY: Tell us, first, have you had --  
2 made any attempt whatsoever to contact the FDA to try to share  
3 the information that you have about potential reduction of  
4 acrylamide in coffee?

5 A. No, I haven't.

6 Q. And the information that you have includes  
7 publicly available information such as things on the  
8 Novozymes' website, correct?

9 A. Much of what I saw came from the documents under  
10 the confidential documents that I was under the court order  
11 not to discuss.

12 Q. So it's your testimony that the reason you  
13 haven't made any attempt to contact the FDA is you feel it  
14 might potentially be violating the protective order in this  
15 case?

16 A. That's one reason.

17 Q. What are the others?

18 A. Sometimes -- I've worked in the federal  
19 government for nearly 30 years. Sometimes policy decisions  
20 take a long time.

21 Perhaps through a court case there could be a faster  
22 means of reducing acrylamide from coffee.

23 Q. So rather than telling the FDA about it, you  
24 thought it would be quicker to come tell about it in a trial  
25 in a courtroom in L.A.?

26 A. I find that within the federal government one  
27 person's comment does not necessarily move a bureaucracy that  
28 fast.

1 Q. You didn't think you had a duty to at least try,  
2 given what's at stake here?

3 MR. METZGER: Objection, argumentative.

4 THE WITNESS: Again, these were confidential documents  
5 that I was aware of in the past six months.

6 If you relieve the court order, I would be happy to  
7 do it.

8 Q. BY MR. KENNEDY: During those six months, have  
9 you ever had a conversation with Mr. Metzger along the lines  
10 of is there something we can do to get an exception to this  
11 protective order so that we can at least share this with the  
12 FDA? Any conversations along those lines?

13 A. I don't recall any.

14 Q. Now, you talked quite a bit yesterday about the  
15 200-ton production example from Novozymes and the German  
16 company, correct?

17 A. I don't know if I spent a lot of time on it.

18 Q. It was discussed?

19 A. Yes.

20 Q. And that's the only example you have of any kind  
21 of wide-scale commercial production of at least an enzyme  
22 attempt to reduce acrylamide in coffee, correct?

23 A. That is correct.

24 Q. And turning, if we could, to your declaration,  
25 which was referred to yesterday, Exhibit 59957, this is your  
26 most recent declaration you talked about yesterday, correct?

27 A. Is there a date stamp on this one? I'm looking  
28 for it.

1 Q. This is the one that's unsigned.

2 Do you want us to go to the signature page to confirm  
3 there?

4 A. That's okay. It's probably the --

5 Q. Why don't we turn to paragraph 63 of  
6 Exhibit 59957.

7 And what you explain there is in September of 2011  
8 Helmut Guenther, a food scientist at Kraft Foods, prepared an  
9 update on acrylamide and using asparaginase to reduce levels  
10 in coffee in which you say he updated the European coffee  
11 industry regarding the collaborative effort between Novozymes  
12 and Hermanson. That's the German company we talked about,  
13 right?

14 A. Correct.

15 Q. And then you go on to say that, "Novozymes is  
16 aware of the current coffee industry," and skipping down over  
17 to the --

18 MR. METZGER: Objection to skipping.

19 MR. KENNEDY: All right. We will not skip.

20 THE COURT: All right. Read the whole thing.

21 MR. KENNEDY: (Reading:)

22 "Novozymes is aware of the current  
23 coffee industry position that using enzymes  
24 is not seen as an option to reduce for  
25 efficiency, quality and cost and food  
26 safety reasons (as detailed in the Food  
27 Drink Europe Acrylamide Toolbox) and is  
28 addressing this by showing data which

1           achieved reductions of up to 70 percent  
2           instead of our industry findings of a 10  
3           max 45 percent reduction.

4           "This is together with mentioning that  
5           coffee has been processed at industrial  
6           scale already.

7           "According to the presentations of  
8           Sara Lee, more than 200 tons of coffee have  
9           been processed on industrial scale and sold  
10          in the market" --

11         MR. METZGER: To the market.

12         MR. KENNEDY: (Reading:)

13                 -- "to the market.

14                 "Additionally they are referring to the  
15                 opportunity to combine the enzyme process  
16                 with other green coffee treatments,  
17                 (steaming), stating that under the current  
18                 green price environment they believe coffee  
19                 roasters are interested in the possibility  
20                 of modifying blends to lower costs without  
21                 impacting quality and without increasing  
22                 level of acrylamide."

23         That's what it says, correct?

24         A.         Yes, that's from the --

25         Q.         Were you present at the presentation to Sara  
26         Lee?

27         A.         No, I wasn't.

28         This is a quote that I obtained from the confidential

1 documents.

2 Q. And you've never talked to anybody that has told  
3 you for sure they were present at the presentation to Sara  
4 Lee, have you?

5 A. No, I haven't.

6 Q. And you don't know one way or another whether  
7 Mr. Guenther was present at the presentation to Sara Lee,  
8 do you?

9 A. You would have to go back to see how he worded  
10 it since that's where the page split is.

11 Q. Well, you certainly never talked to him to find  
12 out whether he was there or not, have you?

13 A. No, I haven't spoken to Guenther.

14 Q. Or made any effort to find out whether  
15 Mr. Guenther was present at the Sara Lee presentation?  
16 You haven't done that either, have you?

17 A. Under the assumption that he was there in order  
18 to have acquired that information as opposed to simply just  
19 making it up?

20 Q. You don't know whether Mr. Guenther talked to  
21 someone who was at Sara Lee, whether he talked to someone who  
22 talked to someone at Sara Lee? You don't know how he came to  
23 have this information, do you?

24 A. Could we go back to page 21 again?

25 Q. Sure.

26 THE COURT: I'm sorry. Could you speak in the  
27 microphone. I can't hear.

28 THE WITNESS: I just asked to move the page.

1 THE COURT: But in general, please speak closer to the  
2 microphone.

3 Q. BY MR. KENNEDY: Let's go back to the beginning  
4 of paragraph 63.

5 A. I'm under the assumption that he heard directly  
6 in terms of making a presentation, but I wasn't there and I  
7 haven't spoken with him so I can't confirm that assumption.

8 But I would see no reason why he would make such a  
9 statement to the European coffee industry if he who works for  
10 the Kraft Foods would be misleading.

11 It seems to me he's writing this in an encouraging way.

12 Q. You assumed, correct?

13 MR. METZGER: Assumed what?

14 Q. BY MR. KENNEDY: You assumed that Mr. Guenther's  
15 comments were accurate?

16 A. I assumed they were accurate because he's  
17 representing a coffee company indicating that there is  
18 information that is relevant to producing a product that would  
19 be -- have reduced levels of acrylamide.

20 Q. Again, I don't mean to be argumentative, but you  
21 don't believe everything you're told by a coffee company, do  
22 you?

23 MR. METZGER: Objection, argumentative.

24 THE COURT: Objection sustained.

25 Q. BY MR. KENNEDY: Now, going to the 200 tons of  
26 coffee referenced there, you don't know anything about that  
27 other than what's on this piece of paper, do you?

28 A. I learned about that this year when I received

1 these documents.

2 Q. Okay. You don't know whether anybody actually  
3 drank any of that coffee, do you?

4 A. Again, I would assume it's more likely that the  
5 coffee was drank rather than poured into the Boston Harbor.

6 Q. How about into coffee ice cream.  
7 You don't know whether it went there or not?

8 A. The 200 tons?

9 Q. Yeah.

10 A. I don't know if the 200 tons went into coffee  
11 ice cream.

12 Q. Or coffee candy?

13 MR. METZGER: I thought coffee ice cream and coffee  
14 candy were not a part of the case.

15 THE COURT: Objection sustained.

16 The witness doesn't know. He's just speculating.  
17 Let's move on to something the witness knows about.

18 Q. BY MR. KENNEDY: And you haven't talked to  
19 anybody who could tell you anything about the success or lack  
20 of success of that market, can you?

21 MR. METZGER: Objection, 352, your Honor.

22 THE COURT: Overruled.

23 THE WITNESS: No, I haven't.

24 Q. BY MR. KENNEDY: And have you seen anywhere that  
25 after that 200-ton production they ever sold any more of it?

26 A. I haven't seen any additional information on  
27 that.

28 Q. The world coffee market is tens of millions of

1 metric tons, correct?

2 A. Correct.

3 Q. Okay. So if somebody has a successful like  
4 product, you would expect them to sell more than 200 tons,  
5 wouldn't you?

6 MR. METZGER: Objection, calling for speculation.

7 The coffee industry has boycotted it.

8 THE COURT: The objection is sustained.

9 Let's go on to another question.

10 Q. BY MR. KENNEDY: Has the Novozyme process for  
11 coffee been approved by the FDA?

12 MR. METZGER: Objection, vague.

13 THE WITNESS: The enzyme has been --

14 THE COURT: Overruled.

15 You may answer. Go ahead.

16 THE WITNESS: I don't know if the Novozymes process,  
17 but I know the enzyme has been approved by FDA, the use of  
18 that enzyme in foods.

19 Q. BY MR. KENNEDY: Okay. And the product has not  
20 been what you would consider thoroughly safety tested, has it?

21 A. I haven't seen information on safety testing of  
22 asparaginase-treated coffee.

23 Q. And you would certainly want to see further  
24 analysis done before, for example, a pregnant woman started  
25 drinking coffee that had been treated with the Novozymes  
26 process, correct?

27 MR. METZGER: Objection. That's argumentative as  
28 phrased and compound.

1 THE COURT: Overruled.

2 You may answer.

3 THE WITNESS: Although I don't expect this to create a  
4 problem, I would still feel comfortable -- more comfortable if  
5 the product was tested for safety.

6 But by selectively removing this amino acid, there  
7 would not be any reason to believe that a highly toxic  
8 material would arise.

9 Q. BY MR. KENNEDY: But you would like to see more  
10 testing as a cautious scientist, correct?

11 A. That would be correct.

12 Q. And you're familiar with a researcher named Fei,  
13 F-E-I, Xu, X-U, at the University of Redding.

14 In fact, you cited some of his articles in this case,  
15 correct?

16 A. I'm familiar with the college and the Xu papers,  
17 yes.

18 Q. And going to Plaintiff's Exhibit 57084,  
19 plaintiff's exhibit, it talks about the effect of asparaginase  
20 on flavor formation in roasted coffee.

21 You've seen that, haven't you?

22 A. Yes, I have.

23 Q. And going over to the third page of that  
24 document, if we could, under figure 2, we've highlighted  
25 there:

26 "Two-way ANOVA," A-N-O-V-A, of groups three  
27 and four showed that furfural and 5 methyl  
28 furfural increased as a result of steaming,

1           while furfuryl alcohol increased with  
2           increasing asparaginase dose with levels in  
3           treatment being significantly higher."

4           You see that, don't you?

5           A.       Yes, I do.

6           Q.       And furfuryl is a carcinogen, isn't it?

7           A.       Yes.

8           Q.       In fact, it's on the Prop 65 --

9           MR. METZGER: Hold it. Let him answer.

10          THE COURT: Let the witness complete his answer.

11          THE WITNESS: Yes. It's shown to induce tumors in  
12          rodents.

13          In fact, that was one of the chemicals that was  
14          discussed at the IARC meeting last June when I was there.

15          Q.       BY MR. KENNEDY: And it's on the Prop 65 list,  
16          isn't it?

17          A.       I believe it was recently added to the list.  
18          But I also believe there's no NSRL established yet or cancer  
19          potency value established for furfuryl by OEHHA.

20          Q.       You agree with me it is a good step?

21          A.       I believe acrylamide is worse than furfuryl  
22          alcohol.

23          Q.       Do you have any published research to that  
24          effect?

25          A.       Acrylamide is a probable human carcinogen from  
26          IARC evaluation based on its multi-site carcinogenicity in  
27          rodents as well as the types of chromosomal damage,  
28          mutagenesis.

1 Furfuryl does not have the same extent of information  
2 as acrylamide, and for a number of reasons like that the IARC  
3 panel voted unanimously that furfuryl alcohol should be listed  
4 as a possible human carcinogen.

5 Q. Let's go back to Exhibit 61950, the  
6 demonstratives from yesterday and going to your slide 44.

7 Do you remember this is where you talked about a  
8 benefit-risk analysis and discussed the BRAFO proposal?

9 A. Yes.

10 Q. Now let's go back once again to Exhibit 71536,  
11 the final statement of reasons for section 25703.

12 Slide 11, please.

13 And in the course of reading the final statement, you  
14 saw that among others things they agreed with you that:

15 "On the other hand, there's extensive  
16 information in the scientific literature  
17 that indicates that chemicals having  
18 mutagenic or carcinogenic properties are  
19 formed as a result of cooking food.

20 "The chemicals formed and their  
21 amounts vary with such factors as the  
22 method of cooking (e.g., boiling, pan  
23 frying, grilling, et cetera,) the  
24 temperature and duration of cooking and the  
25 type of food.

26 "Chemicals that have been found in  
27 cooked food include benzoapyrene and other  
28 polycyclic aromatic hydrocarbons" -- how do

1           you pronounce that?

2           A.       Tryptophan.

3           Q.       (Reading:)

4           -- tryptophan 1 and other amino acids  
5           pyrolusites, nitrosamines and aldehydes. A  
6           number of these chemicals have been listed  
7           as known to the state to cause cancer."

8           Going to slide 12:

9                    "In light of the offsetting public  
10           health benefit that the cooking of food  
11           provides, the agency takes the position  
12           that businesses which utilize cooking  
13           necessary for the processing or preparation  
14           of food should not be strictly held to the  
15           10 to the minus 5 standard."

16          You see that, don't you?

17          A.       Yes, I do.

18          Q.       Wouldn't you agree the State has already made  
19          the risk-benefit analysis?

20          A.       For acrylamide in coffee?

21          Q.       For cooking exception.

22                    For carcinogens formed as a result of necessary  
23          cooking, hasn't the state said we find the benefits and  
24          palatability outweigh the risks of carcinogen --

25                    MR. METZGER: Objection, legal conclusion,  
26          argumentative.

27                    THE COURT: Overruled.

28                    THE WITNESS: I'm not sure if that necessarily means

1 when there is a condition in which a chemical carcinogen can  
2 be easily removed.

3 I don't think that exception would hold under that kind  
4 of a scenario.

5 Q. BY MR. KENNEDY: As we've already established,  
6 even if broiling causes more carcinogens than boiling, that  
7 doesn't make a difference, but you think it does here?

8 MR. METZGER: Objection, argumentative, compound.

9 THE WITNESS: Yes, I do.

10 THE COURT: Overruled.

11 Q. BY MR. KENNEDY: Now, let's turn next to a  
12 document you discussed yesterday, the Dietary Guidelines  
13 Advisory Committee.

14 Going to Exhibit 61950, the demonstratives, slide 51.

15 There you summarize some of your thoughts about the  
16 USDA scientific report of the 2015 Dietary Guidelines Advisory  
17 Committee.

18 Do you recall that?

19 A. Yes, I do.

20 Q. Okay. Have you read Dr. Kessler's testimony in  
21 this case?

22 A. Yes, I have.

23 Q. And you recall he described this advisory  
24 committee as being about as good as science gets?

25 You recall that, don't you?

26 A. That is what he said.

27 Q. Do you agree?

28 A. No.

1 Q. Well, tell us, what's your opinion of the  
2 advisory committee?

3 A. Well, I'm referring to a particular situation  
4 because I haven't reviewed all aspects of the advisory  
5 committee as well as the members. But I noted yesterday in  
6 looking at the Dietary Guidelines, that it does not mention  
7 acrylamide.

8 The issue of acrylamide in foods has been known since  
9 2002. It has been raised as a health concern for more than  
10 10, 12 years. Going back to the WHO/FAO risk assessment on  
11 acrylamide in foods.

12 I think the Dietary Guideline Advisory Committee was  
13 deficient in addressing, to me, what is an important health  
14 concern.

15 Q. Okay. And you then had four bullet points on  
16 this slide. You felt those were the most significant  
17 takeaways from what you got out of the report?

18 A. The most significant takeaway for me was what  
19 wasn't in the report, and that was acrylamide.

20 Q. You found that -- how would you describe in your  
21 own words -- disappointing, surprising, incomplete?

22 A. Deficient.

23 Q. Outdated?

24 A. Deficient.

25 Q. Okay. So we can add the advisory committee to  
26 the list of entities that you feel are dealing with either  
27 outdated or deficient information, correct?

28 MR. METZGER: Objection, argumentative, cumulative.

1 THE COURT: Objection sustained.

2 Q. BY MR. KENNEDY: Now, going to your fourth  
3 bullet point, "Recommends that individuals who do not consume  
4 caffeinated coffee should not start to consume it."

5 It's not quite a complete thought, is it?

6 Let's go to the report itself, Exhibit 71322.73.

7 Sorry, 71073.

8 My mistake, your Honor.

9 71073.023, Kessler 58.

10 And can you scroll down to the bottom of the page, last  
11 sentence beginning with "furthermore."

12 The sentence actually read, didn't it, "Furthermore,  
13 individuals who do not consume caffeinated coffee should not  
14 start to consume it for health benefits alone."

15 You cut that off, didn't you?

16 A. I have no problem with that phrase --

17 Q. Why didn't you --

18 A. -- for health benefits.

19 Q. Why didn't you include it on your slide?

20 A. That's the rationale that -- I have no -- those  
21 words are correct.

22 Q. But yours weren't, were they?

23 MR. METZGER: Objection, argumentative.

24 THE COURT: Overruled.

25 THE WITNESS: It was simply that the committee did not  
26 recommend the consumption of caffeinated coffee for people who  
27 were non-coffee drinkers to start if it's for health benefits.

28 I recall Dr. Kessler said in his testimony he's not a

1 coffee drinker. He said also, after seeing this information,  
2 maybe I should consume it.

3 So maybe this is a statement written for Dr. Kessler,  
4 since he's not a coffee consumer and he believes that there  
5 are potential health benefits.

6 Q. Okay. And if you can go just above where you  
7 quoted the -- can you go back to slide 51 for a minute.

8 The first bullet is "Moderate Coffee Consumption."

9 Now, let's go back to the report itself, .023.

10 And right above moderate coffee, if we go up just two  
11 paragraphs, Tom, we get to "Conclusion."

12 We have two conclusions.

13 You'll notice the first conclusion is:

14 "Strong and consistent evidence shows that  
15 consumption of coffee within the moderate  
16 range, three to five cups or up to  
17 400 milligrams a day of caffeine, is not  
18 associated with increased risk of major  
19 chronic diseases such as cardiovascular  
20 disease, CVD, and cancer and premature  
21 death in healthy adults. Grade: Strong."

22 You didn't include that in your summary of what the  
23 advisory committee found, did you?

24 A. No, I didn't.

25 Q. Is that because you disagree with that?

26 A. Well, I think it's an complete consideration.

27 Q. You didn't think it was worth putting on a slide  
28 when you were talking about considerations of health benefits,

1 correct?

2 MR. METZGER: Objection, argumentative.

3 THE COURT: Sustained.

4 THE WITNESS: For example --

5 THE COURT: No.

6 Next question.

7 Q. BY MR. KENNEDY: And going to the next paragraph  
8 on your conclusions, it states, does it not:

9 "Consistent observational evidence  
10 indicates that moderate coffee consumption  
11 is associated with reduced risk of type 2  
12 diabetes and cardiovascular disease in  
13 healthy adults. In addition, consistent  
14 observational evidence indicates that  
15 regular consumption of coffee is associated  
16 with reduced risk of cancer of the liver  
17 and endometrium and slightly inverse or  
18 null associations are observed for other  
19 cancer sites."

20 You didn't include that in any of your demonstratives,  
21 did you?

22 A. No.

23 Since the evidence was moderate and it was based on  
24 observational evidence, I didn't think it was necessary.

25 Q. Okay. Other than you yourself, are you aware of  
26 anybody who's criticized the 2015 advisory committee findings?

27 A. For being deficient in addressing acrylamide?

28 Q. Better question, yes.

1 A. I --

2 Q. Start there.

3 A. I haven't read the comments that have been  
4 forwarded to the advisory committee, but to me it's an obvious  
5 deficiency within that committee's report.

6 Q. But as you sit here now, you can't think of  
7 anybody else who shares your criticisms of them?

8 A. Again, I haven't seen any comments that were  
9 made to this report, so I can't say anybody who shares.

10 But I would be sure that anybody who's worked in  
11 toxicology and knows the issues related to acrylamide as a  
12 carcinogen, a genotoxic carcinogen, a germ cell mutagen and  
13 that it's present in food would feel it should be included in  
14 any type of dietary guidelines.

15 But I can't name people because I haven't seen any  
16 comments that were written to this report.

17 Q. Okay. Let's go back to the demonstratives  
18 61950, slide 58.

19 That was "Sound Considerations of Public Health" and  
20 you identified four or five factors, correct?

21 A. Correct.

22 Q. And those are based on criteria that are in your  
23 reports for applying an ASRL, correct?

24 A. I would prefer if you would call it an ARL as  
25 opposed to an ASRL, because I think the definition of  
26 "significant" under Prop 65 is 1 per 100,000.

27 So this is actually just an alternative risk level, not  
28 an alternative significant risk level.

1           So in answering a question, I would be answering it  
2 under the condition that I hear an ASRL to really mean an ARL.

3           Q.       You have trouble with the term ASRL?

4           A.       The S in that acronym, yes.

5           Q.       Okay. In any event, on slide 58, you don't  
6 identify any source for these criteria, do you?

7           MR. METZGER: Objection. Vague. What criteria? What  
8 are the criteria he's talking about?

9           THE WITNESS: These are --

10          THE COURT: Objection overruled. You may answer.

11          THE WITNESS: These are my take-home messages from work  
12 in the field and primary prevention.

13                I've written in papers with Dr. Lorenzo Tomatis, who  
14 was the director of the International Agency for Research on  
15 Cancer in terms of stating that certain environmental  
16 carcinogens provide no health benefit. This is something I've  
17 written about more than ten years ago.

18                The mitigation aspects of my report indicate that the  
19 level can be significantly reduced, so that's basically just a  
20 take-home from sound considerations of public health and that  
21 it can be substantially reduced without negatively affecting  
22 palatability.

23                It's not a necessary constituent in coffee, because it  
24 has no flavor or nutritive value.

25                So these are just conclusions that are very easily  
26 reached from anyone in the public health environment.

27          Q.       And did you attempt to compare those conclusions  
28 with the final statement of reasons for section 25703?

1 MR. METZGER: Objection, lacking foundation.

2 Those were written before it was even known that  
3 acrylamide was in food.

4 THE COURT: Objection overruled.

5 THE WITNESS: No, I haven't compared them.

6 Q. BY MR. KENNEDY: Going back to the final  
7 statement, 71 -- Exhibit 71356, slide nine, again, in the  
8 course of reading the final statement you saw, didn't you,  
9 that:

10 "The agency made an exception where  
11 sound considerations of public health  
12 support an alternative level of risk.

13 "To illustrate what constitutes a  
14 sound consideration of public health, the  
15 existing regulation provides a single  
16 example.

17 "The agency believes that additional  
18 examples will better serve to illustrate  
19 what kinds of public health considerations  
20 warrant special treatment.

21 "The public health exception is  
22 justified because the act was intended by  
23 the voters as a measure to protect the  
24 public health and well-being, ballot  
25 pamphlet, Safe Drinking Water and Toxic  
26 Enforcement Act of 1986, Section 1.

27 "It might contravene this intent if  
28 the act were construed to prohibit

1 activities which protect the public health.

2 "It would be ironic and  
3 counterproductive if, as a result of  
4 warnings, the public avoided practices  
5 which protect the public health."

6 Did you have that in mind when you were preparing your  
7 list of considerations of public health?

8 A. Probably I didn't have it in mind, but I can see  
9 what it says at this point.

10 Q. And then going to slide ten from Exhibit 71356,  
11 the final statement goes on to explain:

12 "This regulatory action amends  
13 subsection B of section 12703 to add two  
14 additional examples of public health  
15 considerations: Where chemicals in food  
16 are provided by cooking necessary to render  
17 the food palatable or to avoid  
18 microbiological contamination and two,  
19 where chlorine disinfection, in compliance  
20 with all applicable state and federal  
21 safety -- where chemicals in food are  
22 produced by cooking necessary to render the  
23 food palatable or to avoid microbiological  
24 contamination and, two, where chlorine  
25 disinfection in compliance with all  
26 applicable state and federal safety  
27 standards is necessary to comply with  
28 sanitation requirements."

1           Did you have that language in mind when you were coming  
2 up with your considerations of public health?

3           A.       No. I was aware of these, but I don't see any  
4 inconsistency.

5           Q.       You don't feel that your list is adding  
6 additional requirements beyond what the section already  
7 provides?

8           A.       No.

9           I view it as a situation for sound considerations of  
10 public health, that the removal of a carcinogen is a public  
11 health consideration.

12          So in my view removal of a carcinogen is an important  
13 goal for an industry which provides a product that has a  
14 carcinogen in it at levels greater than the NSRL.

15          Q.       You would agree with me, however, the State of  
16 California has already concluded that the benefits of  
17 palatable food outweigh the carcinogenic risk?

18          We can agree on that?

19          MR. METZGER: Objection, argumentative, legal  
20 conclusion.

21          THE WITNESS: But I think we can also agree --

22          THE COURT: Hold on one second.

23          THE WITNESS: Pardon?

24          THE COURT: The objection is overruled.

25          The witness can comment on his understanding as to what  
26 the State of California has concluded.

27          With that caveat, you can answer the question.

28          THE WITNESS: Okay. I think we can also agree that

1 removal of acrylamide does not prevent the roasting of coffee  
2 to make it palatable and remove microbiological contaminants.

3 So I think there is a consistency in terms of the view  
4 from a public health consideration.

5 Q. BY MR. KENNEDY: Now, you talked yesterday about  
6 acrylamide being responsible for, what, up to 40 percent of  
7 the -- excuse me, for coffee being responsible for up to  
8 40 percent of the acrylamide in the adult diet, correct?

9 A. Yes.

10 Q. Okay. And you base that 40 percent statement on  
11 a paper by a gentleman named Mucci, correct?

12 A. I would have to go through my documents again.  
13 I think it was made by -- it could be Mucci. It might be  
14 Friedman as well.

15 I would have to look at a variety of sources. I didn't  
16 scan the full literature to see any other -- the total  
17 evaluations of acrylamide from dietary sources.

18 But I've seen 40 percent as a numerical value that has  
19 been attributed to coffee in adults.

20 Q. And in your deposition you told us that you had  
21 relied on Mucci and Friedman, as you recalled here, correct?

22 A. At that point, I believe so.

23 But, again, I would have to re-look at the references  
24 in total.

25 Q. And let's go to defendants' exhibit 69866.

26 Let us know if that is the Mucci article, "Prospective  
27 Study of Dietary Acrylamide and Risk of Colorectal Cancer  
28 Among Women."

1           A.       I really don't recall if this was the article  
2 from which I got that value.

3           Q.       If there is any doubt about it, let me move on  
4 to Friedman.

5           Let's take a look at Exhibit 68647.

6           THE COURT: Before we go there, let me ask a general  
7 question.

8           To your knowledge, is there any other food product that  
9 when processed in cooking or otherwise, it creates acrylamide  
10 or releases acrylamide?

11          THE WITNESS: Potatoes.

12          Potatoes actually have higher --

13          THE COURT: Potatoes and potato chips. Any others?

14          THE WITNESS: Breads, baking breads.

15          That's in many products from baking at high  
16 temperatures.

17          THE COURT: And to your knowledge in terms of your  
18 experience, has there been any concern about the risks of  
19 cancer, other than the coffee and potato chips or french  
20 fries?

21          THE WITNESS: I would have to, again, look at the full  
22 literature on that.

23          It is in a number of products. It might be in some of  
24 the baby foods as well.

25          THE COURT: In processing baby foods?

26          You're talking about baby foods that include some kind  
27 of a grain or is it just any baby food, the processing, or  
28 vegetables?

1 THE WITNESS: I'm not sure if it's just grain. I think  
2 it's in vegetables, as well.

3 But, again, it's the condition in which you have free  
4 asparagine in reducing sugars that are heated to sufficiently  
5 high temperature.

6 THE COURT: To your knowledge, has anyone raised a  
7 concern of the risk of cancer from any of those other  
8 products?

9 THE WITNESS: I'm not sure how concerns are raised,  
10 whether they're --

11 The totality of acrylamide in foods has been raised as  
12 a concern. That was done by the WHO/FAO who considered the  
13 total acrylamide in human diets, as well as EFSA in their  
14 documents raised a concern of total acrylamide.

15 THE COURT: We've seen these articles about the concern  
16 for dietary content and toxicity of acrylamide but mainly  
17 discussing the product of coffee and the potatoes you  
18 mentioned.

19 Any other particular foodstuffs?

20 THE WITNESS: Those are the ones that are highlighted  
21 because potatoes had the highest levels. But coffee has the  
22 highest consumption levels on a daily basis.

23 So coffee becomes more of a target of concern because  
24 in the adults the level -- the acrylamide source can approach  
25 40 percent in adults, though it is present in other foods.

26 THE COURT: Any acrylamide released in the processing  
27 of tea leaves?

28 THE WITNESS: I would have to look at the tables. I

1 don't know foods, in general, that have been shown to contain  
2 acrylamide, but I know the list the fairly large.

3 THE COURT: Thank you.

4 Mr. Kennedy?

5 MR. KENNEDY: I think the next exhibit may be of  
6 interest to your Honor.

7 THE COURT: Yes. Go ahead.

8 Q. BY MR. KENNEDY: Going to Exhibit 68647, that's  
9 Friedman and Levin, "Review of Methods for the Reduction of  
10 Dietary Content and Toxicity of Acrylamide," correct?

11 A. Yes.

12 Q. And let's go to page 4.

13 And they have a number of pie charts, don't they, here,  
14 breaking down important sources of acrylamide in various  
15 populations.

16 And in Sweden it's up at 39 or 40 percent as coffee,  
17 correct?

18 A. Yes.

19 Q. Okay. And if we use the United States -- again,  
20 other things, your Honor, would be -- let's stay with Sweden.

21 Coffees, 39 percent, bread is 11 percent, fried potato  
22 products and chips are 36 percent, crackers, cookies, et  
23 cetera, 11 percent, cereal products 2 percent.

24 Other, 1 percent.

25 Now, let's take a look at the pie chart for the United  
26 States.

27 Coffee, 8 percent.

28 You didn't mention that when you talked about this

1 article, did you?

2 MR. METZGER: Objection, argumentative.

3 THE COURT: Sustained.

4 Q. BY MR. KENNEDY: You'll agree with me the  
5 article you relied upon shows that in the United States coffee  
6 is responsible for 8 percent of acrylamide intake?

7 A. I would have to look to see if this is for  
8 adults or the total population.

9 So if it's including children, you have a large  
10 population which is diluting out the effect -- the acrylamide  
11 source of coffee in adults.

12 So it depends on how you present data in terms of how  
13 you can make a conclusion. But if this is total population,  
14 that includes children, probably includes non-coffee drinkers  
15 as well.

16 So I think the issue here is really the acrylamide  
17 exposure among coffee drinkers and that percentage of  
18 acrylamide that comes from the diet, not from non-coffee  
19 drinkers.

20 So you have a dilution factor in here which needs to be  
21 accounted for.

22 Q. They have children in Sweden, don't they?

23 A. I think so.

24 MR. METZGER: Objection, argumentative.

25 THE COURT: Sustained.

26 Q. BY MR. KENNEDY: Do you have any reason to think  
27 that they would have different populations for different pie  
28 charts here and not say something about it?

1 MR. METZGER: Objection, argumentative.

2 THE COURT: Sustained.

3 Q. BY MR. KENNEDY: In any event the Levin article  
4 doesn't support a 40 percent coffee acrylamide factor for the  
5 United States, does it?

6 A. Not for the total population, including children  
7 and non-coffee consumers.

8 But that number will grow substantially if you take out  
9 the non-coffee consumers. And whether it reaches 39 percent  
10 or even goes higher, I can't tell you.

11 But I would imagine in Sweden, if we take out the  
12 children from that population, it would probably go higher as  
13 well.

14 But when you look at data, you have to understand the  
15 full conditions under which these data are being presented.

16 We're sort of cherry-picking numbers to make a point.

17 MR. METZGER: It says Swedish adults. The other is  
18 children.

19 Q. BY MR. KENNEDY: Going back to the Mucci article  
20 again, Exhibit 69866, that was a study just of Swedish women,  
21 wasn't it?

22 A. That's what it says.

23 Q. There were no Americans at all in that article,  
24 as far as you know?

25 A. No.

26 I believe, though, when I referred to these articles, I  
27 said it had been estimated from 8 to 40 percent among adults,  
28 citing Mucci, the Friedman and Levin, as well as EFSA.

1 Q. Changing topics.

2 Your Ph.D. is in food science?

3 A. That is correct.

4 Q. And since getting your Ph.D. in 1970, you've  
5 never actually worked anyplace where your title is food  
6 scientist, correct?

7 A. No.

8 After receiving my Ph.D., I did post-doctoral research  
9 at the University of California and Berkeley in which I became  
10 more involved in cell biology, cell physiology.

11 I sought an academic position from there. And after my  
12 academic career, I went and joined the U.S. government in the  
13 National Toxicology Program.

14 So I did not work for a food company, although I did  
15 summer work at a food company when I was in college.

16 Q. But the answer is since 1970 you've never worked  
17 anyplace where your title was food scientist, correct?

18 A. That's correct.

19 Q. And what's organoleptic testing?

20 A. It's sensory testing. Tasting is an example.

21 Q. And you don't have any expertise or training in  
22 that, do you?

23 A. I've done taste testing. In coursework, it  
24 included taste testing. But I don't have any experience after  
25 my Ph.D. with taste testing.

26 Q. And you're not here expressing any opinions on  
27 whether there is or is not an effect on the taste and aroma of  
28 coffee with the various acrylamide reduction methods you've

1 talked about, correct?

2 MR. METZGER: Objection.

3 THE WITNESS: No.

4 I think I indicated that there have been demonstrations  
5 of acceptable quality of coffee which has been treated to  
6 reduce acrylamide levels.

7 Q. BY MR. KENNEDY: And we talked yesterday about  
8 FDA tolerances for PCB's in fish.

9 Do you remember that?

10 A. Yes, I do.

11 Q. And you pointed out that the FDA has found that  
12 fish have a positive health benefit?

13 A. Correct.

14 Q. Okay. And they found that in about 2004,  
15 correct?

16 A. I don't recall the year.

17 Q. And do you recall that they gave the fish the  
18 deviation level in 1984, approximately 20 years before the  
19 health claim was made.

20 Do you remember that?

21 A. I don't know the dates.

22 Q. Now, one of the critiques you had of  
23 Dr. Rhomberg was his use of a PK factor, correct?

24 A. Yes, that is correct.

25 Q. And OEHHA in some of its work has used PK  
26 factors, correct?

27 A. I know it was used in the 2005 assessment for  
28 acrylamide, if that's what you're referring to.

1 I don't know if it's been used in other documents. I  
2 assume it might have been, but I haven't read all of their  
3 risk assessment documents.

4 Q. And you're critical of OEHHA for doing that, for  
5 the same reasons that you're critical of Dr. Rhomberg,  
6 correct?

7 A. Well, there's multiple aspects which I thought I  
8 presented hopefully clearly yesterday in terms of the use of a  
9 PK factor for mouse tumor responses versus rat tumor  
10 responses.

11 OEHHA used a PK factor for the rat tumor response  
12 because when they did their risk assessment, the mouse tumor  
13 data were not available.

14 The mouse tumor response, to me, was massively  
15 incorrect.

16 The rat, I have less of a problem with what they did.  
17 But I pointed out certain aspects which lead to uncertainties  
18 in the 1.2 numerical value that was used. And I think I  
19 mentioned those yesterday, but I can mention them again if you  
20 would like.

21 Q. This 1.2 is a value that was used by  
22 Dr. Rhomberg, correct?

23 A. For rats but not for mice.

24 Q. And OEHHA used it, correct?

25 A. In the document that never was finalized, yes.

26 Q. And Dr. Bayard used it, correct?

27 A. That is correct.

28 Q. And if you had been doing it, you wouldn't have

1 done it, used a PK factor, correct?

2 A. If I was involved in the risk assessment, I  
3 would have looked carefully at the data and pointed it out to  
4 a group that was conducting the risk assessment to be sure  
5 before applying it whether we had high confidence in that  
6 value or not.

7 And I expressed areas in which my confidence was not  
8 total on the use of that 1.2 value.

9 I'm not saying that there's not a PK factor that might  
10 be usable, but the data upon which that PK factor is based, to  
11 me, has much uncertainty.

12 Using an uncertain PK factor is a concern, because it  
13 might be underestimating the numerical value that is used in  
14 the estimation of risk.

15 MR. KENNEDY: Your Honor, I have a hypothetical  
16 question for the doctor that I've written out.

17 With the Court's permission, may I --

18 THE COURT: Can you show it to Mr. Metzger?

19 MR. KENNEDY: Sure.

20 I predict an objection.

21 THE COURT: Since it's one page, I hesitate to see how  
22 many subordinate clauses there are.

23 MR. METZGER: There are several.

24 THE COURT: All right. Present it to me.

25 MR. METZGER: So I do have several objections.

26 THE COURT: Mr. Metzger?

27 MR. METZGER: Should I state my objections now?

28 THE COURT: Go ahead.

1 MR. METZGER: So the hypothetical --

2 THE COURT: For a complete record, Mr. Kennedy, why  
3 don't you ask the question first and then Mr. Metzger can  
4 assert his objections.

5 MR. KENNEDY: Would it help if I approach and give the  
6 witness a copy or just read it?

7 THE COURT: You can give a copy to the witness, please.

8 Before the witness says anything, Mr. Metzger, we  
9 should have it on the record so we all know what we're talking  
10 about.

11 You may read the question.

12 Q. BY MR. KENNEDY: Dr. Melnick, I want you to  
13 assume the following.

14 First, that cooking necessary to achieve palatability  
15 is, by itself, a sound consideration of public health which  
16 supports an alternative significant risk level or ARL, if you  
17 prefer, of more than one excess case of cancer in an exposed  
18 population of 100,000.

19 Second, that the ASRL or ARL cannot impede the cooking  
20 necessary to achieve palatability.

21 Third, that there is no duty to mitigate or reduce the  
22 amount of acrylamide created by the cooking necessary to  
23 achieve palatability.

24 Under those assumed circumstances, what would be the  
25 proper ASRL or ARL?

26 MR. METZGER: Objection. This is going to be lengthy.

27 The hypothetical is grossly compound, argumentative,  
28 ambiguous, assumes erroneous facts, assumes erroneous law.

1 I am now going to address the particulars of it.

2 The phrase that says that "cooking necessary to achieve  
3 palatability is by itself a sound consideration of public  
4 health which supports an alternative significant risk level or  
5 alternative risk level," that is argumentative legally and  
6 factually.

7 It assumes that -- just a moment. It assumes that a  
8 particular level has been calculated pursuant to a  
9 quantitative risk assessment as required in the regulation  
10 which has not been done.

11 It assumes that palatability cannot be achieved without  
12 acrylamide present or without reducing acrylamide.

13 The second part, that the ASRL cannot impede the  
14 cooking necessary to achieve palatability, that is  
15 argumentative legally. That's not in any regulation.

16 Mr. Kennedy is making up regulation and law.

17 It assumes that an alternative level cannot be achieved  
18 without palatability.

19 It assumes that acrylamide cannot be reduced without  
20 negatively affecting palatability.

21 The third part that there is no duty to mitigate or  
22 reduce the amount of acrylamide is purely a legal question.  
23 It's a question of duty.

24 It's also irrelevant because Proposition 65 does not  
25 require any company to reduce the amount of any carcinogen.

26 Companies are free to expose Californians to  
27 100 percent carcinogens as long as they give a warning.

28 So this is all grossly argumentative legally and

1 factually.

2 I think I've covered enough of it.

3 I object to the entire hypothetical question.

4 THE COURT: All right.

5 MR. KENNEDY: Yes, your Honor.

6 Mr. Metzger has just given us his view of the case.

7 With one exception, I believe the hypothetical not only  
8 sets forth our view of the case but is supported by quotations  
9 that have already come in.

10 If I might, Tom, can we put up Exhibit 71341, slide 2.

11 Your Honor, the source of the word "impede" I got from  
12 the June, 1989 cleanup example. You will recall, that was --  
13 the cleanup of toxic tort sites was the first of the three  
14 examples that the agency adopted.

15 At that time they said,

16 "The agency was informed that  
17 in most cleanups water is taken up, treated  
18 and returned to the same source of ground  
19 or surface water.

20 "The proposed regulation would  
21 prevent liability for chemicals received in  
22 the water.

23 "It's the intention of the agency  
24 that ground and surface water cleanups not  
25 be impeded."

26 As your Honor knows, they thereafter decided one  
27 example wasn't enough. They really needed two more. And the  
28 cooking and chlorine examples came along. Nothing to suggest

1 the same desire to prevent liability.

2 And we're talking about a statute which, as Mr. Metzger  
3 has pointed out, deals with a duty to warn. The liability is  
4 whether you have to warn or not and the exemplar conduct not  
5 be impeded.

6 So it's our position that the ASRL, to the extent a  
7 numerical one is required -- and obviously we're not conceding  
8 that -- it has to be done in a way that will not impede the  
9 roasting of coffee through any mechanism or means it's  
10 entitled to.

11 Clearly, this is not the only argument that could be  
12 made. We, however, feel it would be a benefit to your Honor  
13 to know if you should accept our position supported by the  
14 language we have as to what this esteemed toxicologist  
15 believes on the subject.

16 MR. METZGER: All right. Now I have a further  
17 objection.

18 THE COURT: Yes.

19 MR. METZGER: That with this clarification, this is  
20 irrelevant because it is concerning toxic waste or water  
21 cleanup.

22 There is no claim being made in this case that roasting  
23 need be impeded. Everything that Dr. Melnick has testified to  
24 regarding reduction of acrylamide fully allows roasting of the  
25 coffee. It's not in any way being impeded.

26 He's not proposing that coffee be -- that coffee beans  
27 be boiled or fried or grilled or anything like that. This is  
28 purely a roasting process.

1 Proposition 65 and these things, these regulations, do  
2 not in any way say that it cannot be optimized to reduce  
3 levels of carcinogens.

4 This is all just argument. It's their position of the  
5 case. I consider it wrong.

6 But to include basically their whole legal argument,  
7 including all this stuff from 1989 before acrylamide was known  
8 to be present in food is compound, argumentative. It's  
9 nonsensical.

10 THE COURT: All right. Well, as we all know, expert  
11 witnesses are entitled to be asked hypothetical questions.

12 The witness is being asked to assume certain facts. It  
13 will be up to counsel to argue whether the facts that are  
14 being asked to be assumed have been established.

15 The witness is called upon to assume certain legal  
16 positions that counsel is arguing about, and that will  
17 necessitate further argument of counsel after the conclusion  
18 of the testimony.

19 So I recognize that a number of the clauses and  
20 assumptions have not been established in this case yet.

21 Nevertheless the witness may be asked the question with  
22 these assumptions. And we'll discuss it with counsel later  
23 on, if any of the assumptions are appropriate from either a  
24 factual or a legal position.

25 MR. METZGER: So you're allowing the witness to answer  
26 subject to a motion to strike, your Honor?

27 THE COURT: Yes.

28 MR. METZGER: All right.

1 THE COURT: Mr. Kennedy, do you want to rephrase the  
2 question to refresh the witness's recollection of the  
3 question.

4 MR. KENNEDY: Certainly, your Honor.

5 Q. Dr. Melnick, I want to ask you to assume the  
6 following.

7 That cooking is necessary to achieve palatability is by  
8 itself a sound consideration of public health which supports  
9 an alternative significant risk level, whether you call it  
10 ASRL or ARL, of more than one excess case of cancer in an  
11 exposed population of 100,000.

12 Second, that the ASRL/ARL cannot impede the cooking  
13 necessary to achieve palatability.

14 And, third, that there is no duty to mitigate or reduce  
15 the amount of acrylamide created by the cooking necessary to  
16 achieve palatability.

17 Under those assumed circumstances, what is the proper  
18 ASRL or ARL, if you prefer to call it that?

19 MR. METZGER: May I just confirm my objections are  
20 preserved?

21 THE COURT: Yes.

22 MR. METZGER: Thank you.

23 THE WITNESS: I believe that the citizens of California  
24 wanted to be notified --

25 THE COURT: No, we're not going there. We're not going  
26 there. We're not going to discuss political process of  
27 approving propositions.

28 Please focus on the question.

1 THE WITNESS: You're asking me to provide an arbitrary  
2 number, as I see it, a number that is different than 1 per  
3 100,000, if I accept these assumptions. That's the way I'm  
4 reading your question and hypothetical situation.

5 Q. BY MR. KENNEDY: If you believe it has to be  
6 arbitrary, that's your prerogative. I'm asking you if you can  
7 answer the question as phrased.

8 I'm asking you to assume the correctness of all three  
9 of those assumptions.

10 MR. METZGER: Objection. Now it's argumentative. The  
11 witness has answered. He doesn't like the answer.

12 THE COURT: I haven't heard an answer yet.

13 There was a question about the question.

14 Let's focus on the question. If the witness doesn't  
15 have an answer to the question, it's appropriate to say I  
16 don't have an answer or I don't know. Those are acceptable  
17 responses.

18 THE WITNESS: I don't have the arbitrary value that  
19 would be appropriate.

20 It would not be one that is necessarily the level of  
21 that agent, acrylamide, in coffee, such that we simply accept  
22 what's there. So I cannot give you my arbitrary value.

23 But I don't think that arbitrary value should be just  
24 selected by anybody without quantifying the benefits and risks  
25 associated with these conditions.

26 Q. Does that complete your answer?

27 A. Yes.

28 I cannot give you a numerical value.

1 MR. KENNEDY: I have no further questions.

2 Thank you, Dr. Melnick.

3 THE COURT: Mr. Metzger, any redirect?

4 MR. METZGER: Yes, your Honor.

5 THE COURT: How long is it going to take?

6 MR. METZGER: I would -- I haven't timed it, but I  
7 would say probably a half hour.

8 Should we take a break?

9 THE COURT: At this time we'll take a recess at this  
10 time. We'll be in recess for 15 minutes.

11 (Recess.)

12 THE COURT: Mr. Metzger, are you ready to proceed?

13 MR. METZGER: Yes, your Honor.

14 THE COURT: Back on the record.

15 All counsel are present and Dr. Melnick is on the and  
16 stand.

17

18

**REDIRECT EXAMINATION**

19 BY MR. METZGER:

20 Q. Dr. Melnick, are you in any way suggesting that  
21 the coffee industry should not roast coffee?

22 A. No, I've never made that statement.

23 Q. Are you in any way suggesting that the coffee  
24 industry should not roast coffee sufficiently to reduce  
25 microbial contamination to the levels that they currently are?

26 A. No.

27 Q. Are you in any way suggesting that the coffee  
28 industry should fry, pan fry, broil, boil or prepare coffee or

1 process coffee in any manner other than roasting?

2 A. No, I'm not.

3 Q. So one of the documents that you were shown was  
4 the FDA guidance regarding acrylamide.

5 There was a statement in there that the FDA is  
6 unaware -- presently unaware of a viable option for reducing  
7 acrylamide in coffee.

8 You mentioned that there was a reference for that,  
9 reference number 30.

10 What was that reference?

11 A. Yes. That was the FoodDrinkEurope document that  
12 was prepared in 2013.

13 Q. Okay. And that was actually drafted by one of  
14 the defendants in this case.

15 Are you aware of that?

16 A. I didn't know that.

17 MR. KENNEDY: Objection, assuming facts not in  
18 evidence.

19 Move to strike.

20 THE COURT: The objection is sustained to the question.

21 The answer will be stricken.

22 Q. BY MR. METZGER: You are aware that  
23 FoodDrinkEurope is the food and beverage industry of Europe,  
24 correct?

25 A. Yes, it is.

26 Q. Okay. And do you recall reading among the  
27 confidential industry documents that you were provided that  
28 that statement in FoodDrinkEurope was actually prepared by

1 Nestle?

2 MR. KENNEDY: Objection, assuming facts not in  
3 evidence.

4 THE COURT: Overruled.

5 THE WITNESS: I believe Nestle was involved in it. I  
6 don't know if it was totally Nestle or not.

7 Q. BY MR. METZGER: All right. Now, have you  
8 reviewed a subsequent publication by Food Drink Europe from  
9 May of 2016?

10 MR. KENNEDY: Object. Beyond the scope of cross.

11 THE COURT: Overruled.

12 THE WITNESS: I'm aware that there has been an update  
13 on the FoodDrinkEurope.

14 Q. BY MR. METZGER: And in that updated  
15 FoodDrinkEurope document, the industry has now taken the  
16 position that the asparaginase treatment of Novozymes does  
17 have applications in certain contexts in reducing acrylamide  
18 in coffee; isn't that true?

19 MR. KENNEDY: Object. Best evidence, your Honor.

20 THE WITNESS: Yes. I've seen that statement.

21 THE COURT: The objection is overruled.

22 The answer will stand.

23 Q. BY MR. METZGER: All right. Do we have that  
24 article that was posted up there, the Mucci -- the Friedman?

25 Perhaps the defense could put it up since they had  
26 it up.

27 THE COURT: All right. Please put that up.

28 MR. METZGER: It was the one with the pie charts on it.

1 MR. KENNEDY: If I might, your Honor, I think it's  
2 69866.

3 MR. PARISER: That's Mucci.

4 MR. METZGER: The one that had the pie charts,  
5 whichever it was.

6 There we go. That's it.

7 Q. So we're looking at the document Bates numbered  
8 Smucker. 19474 is the page.

9 The title here is "Important Sources of Acrylamide in  
10 Various Populations," with a subheading "(Percentage of Total  
11 Acrylamide in the Diet.)"

12 May I approach that, your Honor, so I can actually  
13 read it?

14 THE COURT: Yes.

15 Q. BY MR. METZGER: I don't know if you can see it,  
16 Dr. Melnick.

17 A. I see it on my screen.

18 Q. You were shown this pie chart for Sweden. It  
19 had coffee at 39 percent.

20 But the heading above that is actually "Sweden,  
21 adults," parentheses -- it looks like 18 through 74 years of  
22 age?

23 A. Yes, I see that.

24 Q. So that is an adult population. That is coffee  
25 consumption for an adult population, correct?

26 A. That's what it is, Sweden adults.

27 Q. And then when you were shown for the United  
28 States, you were indicating that, well, there might be

1 children included in there.

2 In fact, it says United States and in parentheses  
3 "2 plus populations."

4 So that's including children age two and more, correct?

5 A. Correct.

6 Q. So are these comparable pie charts?

7 A. Definitely not.

8 That's what I was trying to address, but I hadn't  
9 noticed it at that very moment because it was sprung on me.

10 But, yes, my concern was that the population value for  
11 the United States of 8 percent may have included children and  
12 non-coffee consumers, and therefore this would be a total  
13 underestimate for adults.

14 Q. Right. And Dr. Scrafford included children in  
15 her exposure assessment, likewise, correct?

16 A. I believe so, yes.

17 Q. Yeah.

18 A. And Dr. Rhomberg excluded in his risk assessment  
19 the age up to 16.

20 Q. Right. Because he agreed with Dr. Bayard that  
21 it was improper to include children who were not consumers,  
22 correct?

23 A. Correct.

24 Q. All right. Now, is acrylamide as the defense  
25 has used the phrase, "an inevitable byproduct"? I want to ask  
26 the question using that phrase.

27 Is acrylamide an inevitable byproduct of roasting  
28 coffee?

1           A.       Most of the acrylamide is not an inevitable  
2 byproduct of roasting coffee to provide a palatable product.

3           Q.       Why is that?

4           A.       Because if you remove asparagine from the  
5 coffee, you will reduce most of the acrylamide that is formed.  
6 Therefore, most of it is not an inevitable byproduct.

7           Q.       And how do you remove the asparagine from the  
8 coffee?

9           A.       You can treat it with asparaginase.

10          Q.       Okay. And that's one of the -- is one of the  
11 companies that has developed that technique or methodology the  
12 Novozymes?

13          A.       Yes. Novozymes has developed the enzyme, yes.

14          Q.       Right. And is that the specific technique that  
15 the 2016 FoodDrinkEurope recognizes as a viable option for  
16 reducing acrylamide in certain coffees?

17          A.       Yes, it is.

18          Q.       Now, do you believe that there should be an  
19 alternative risk level for coffee?

20          A.       No, I don't.

21          Q.       Why not?

22          A.       Because coffee can be prepared by roasting,  
23 producing a palatable product in which the acrylamide levels  
24 could be achieved at levels below the current NSRL.

25          Q.       So why do you believe there shouldn't be an  
26 alternative risk level?

27                 Can you explain that further?

28          A.       It's unnecessary.

1 THE COURT: Is there any, any alternative risk level  
2 that you would think would be appropriate?

3 THE WITNESS: Not if you can achieve the current NSRL,  
4 then no alternative risk level would be appropriate, in my  
5 view.

6 Q. BY MR. METZGER: And why is that?

7 A. Why can't --

8 Q. Why would no alternative risk level be  
9 appropriate if, as you have indicated, coffee can be produced  
10 with acrylamide levels below the NSRL that's still palatable?

11 A. That's basically the reason, is that you have a  
12 product in which acrylamide is below the NSRL, so there's no  
13 reason to require or need an alternative risk level.

14 Q. And what would devising or allowing an  
15 alternative risk level do in that circumstance?

16 A. An alternative risk level would allow higher  
17 concentrations of acrylamide in the coffee products.

18 Q. And why is that -- why should that not be done?

19 A. Because acrylamide is a potent carcinogen which  
20 is, for public health considerations one in which you want to  
21 reduce human exposure not allow it.

22 Q. Not increase it?

23 A. Not increase it.

24 Q. Okay.

25 A. Sure.

26 THE COURT: So aside from what may happen in future  
27 innovation in the wonderful world of chemistry, living today  
28 in terms of what should be provided today, do you find that no

1 alternative risk level would be acceptable?

2 THE WITNESS: Because it hasn't been adequately  
3 scaled up.

4 But there are methodologies available that can reduce  
5 the acrylamide to levels of 90 percent or more reduction that  
6 in a very short interval, or what could have been done years  
7 ago was to have produced a product that would have met the  
8 NSRL.

9 THE COURT: Well, while we're waiting for that  
10 development, what should be done now?

11 THE WITNESS: The coffee industry should pursue --  
12 well, there are a number of steps that can be done.

13 One is they can start packaging appropriately so that  
14 coffee can be stored for a certain amount of time to start to  
15 reduce the level of acrylamide to a reasonably high extent.

16 I'm not sure if it was in the range of 30, 40 percent.

17 They can apply methods that are available.

18 For example, the supercritical CO2 method, which is  
19 available at most facilities, might require a little bit of  
20 work.

21 But there are methods available that can, right now,  
22 reduce acrylamide to levels probably below the NSRL.

23 And I would also suggest that they make better use of  
24 the asparaginase treatment since it's already been implemented  
25 in the German roaster. Why it hasn't pursued -- there may be  
26 a number of reasons beyond what I can imagine, but it exists  
27 and should be done.

28 Q. And can it be done? That is, can the industry

1 reduce acrylamide in instant coffee?

2 A. Oh, yes.

3 Q. And in decaffeinated coffee?

4 A. Certainly. This works for all coffee products.

5 Q. And when you say "this," what are you referring  
6 to?

7 A. The treatments to reduce acrylamide. There are  
8 methods to reduce it in all coffee products.

9 Q. And does the asparaginase treatment, would that  
10 reduce it in all types of coffee?

11 A. Yes. Because this occurs prior to roasting. So  
12 therefore all acrylamide which forms subsequent to roasting  
13 will be at a lower level. So all coffee products will contain  
14 less acrylamide with asparaginase treatment.

15 Q. And would simply storing roasted coffee in  
16 sealed containers at room temperature or at 37 degrees, would  
17 that likewise reduce acrylamide in all types of coffee that  
18 are roasted?

19 A. It would be -- not in the drink because it's  
20 binding to the matrix.

21 When the matrix is present, it can be reduced, if you  
22 use 37 degrees, up to nearly 50 percent.

23 So as long as the matrix material is there, as I tried  
24 to mention yesterday, the acrylamide will bind with those free  
25 sulphidal groups similar to the way it is detoxified in the  
26 human body.

27 Q. You're losing me, doctor.

28 What I'm trying to understand is can the industry

1 simply by roasting coffee using -- I'm so sorry.

2 Can the industry simply by using the Novozyme  
3 asparaginase treatment reduce acrylamide in all different  
4 types of roasted coffee?

5 A. Yes.

6 Q. And by storing coffee that has been roasted,  
7 whether in whole bean or ground form, storing it in sealed  
8 foil bags or containers, the cans, simply storing it for  
9 several months, is that something that the coffee industry  
10 could do right now to substantially reduce acrylamide in  
11 coffee?

12 A. Yes. Under an inert gas or nitrogen environment  
13 in the can, yes.

14 Q. And are there some coffee companies that  
15 actually store their coffee that way or produce their coffee  
16 that way right now?

17 A. Oh, yes.

18 Q. Okay. And all that they would need to do is to  
19 hold that for a certain period of time to reduce the  
20 acrylamide concentrations; is that correct?

21 A. That is correct.

22 Q. And then there are all these other technologies  
23 that you mentioned which are a little more complicated than  
24 that; is that correct?

25 A. The asparaginase and storage is the easiest.

26 Q. Okay?

27 A. Asparaginase is already developed.

28 Q. Okay. And is it your understanding that, in

1 fact, the reason that although there are 200 tons of coffee  
2 produced and sold to market in Europe using the Novozymes  
3 asparaginase treatment, that the reason there has not been a  
4 market for that is because the coffee industry has boycotted  
5 any reduction in acrylamide treatment?

6 MR. KENNEDY: Assuming facts not in evidence.

7 THE COURT: Objection sustained.

8 Q. BY MR. METZGER: Did you review any documents  
9 that indicated that?

10 A. Some of the documents that were confidential  
11 indicated that, yes.

12 Q. Okay.

13 MR. METZGER: Just one moment, your Honor.

14 I have no further questions.

15 Thank you very much, Dr. Melnick.

16 THE COURT: Any recross?

17 MR. KENNEDY: Very short, your Honor.

18  
19 RE CROSS EXAMINATION

20 BY MR. KENNEDY:

21 Q. As I understand it, to correctly decide this  
22 case, your opinion is that his Honor should reject the FDA  
23 statement that there is no commercially viable means, correct?

24 MR. METZGER: Objection, argumentative.

25 THE COURT: Overruled.

26 THE WITNESS: He should recognize that that statement  
27 is not true.

28 Q. BY MR. KENNEDY: And instead of accepting that,

1 he should accept your proposals for how acrylamide should be  
2 reduced, correct?

3 A. These are not my proposals. These are evidence  
4 based on data.

5 Q. The proposals that you've told us about, he  
6 should accept those, correct?

7 A. To accept the proposal that acrylamide could be  
8 reduced?

9 Q. Yes.

10 A. Yes, I think he should accept the idea that  
11 acrylamide can be reduced in coffee.

12 Q. And these are all proposals that you know about  
13 but have never shared with the FDA, correct?

14 MR. METZGER: Objection. It's cumulative,  
15 argumentative.

16 THE COURT: It's been asked and answered.

17 MR. KENNEDY: Thank you very much.

18 I have no further questions, your Honor.

19 Thank you.

20 THE COURT: Anything further?

21 MR. METZGER: I do have one final follow-up on that.

22 THE COURT: One final follow-up. Go ahead.

23 MR. MARGULIES: I have a question, if I may. I'll just  
24 do it from here, your Honor.

25

26 **CROSS-EXAMINATION**

27 BY MR. MARGULIES:

28 Q. Dr. Melnick, with regard to the storage of

1 coffee, you'd be concerned if storage would create other toxic  
2 byproducts, right?

3 A. Would I be concerned?

4 I've never seen evidence to that effect.

5 Q. So you were relying on the Baum study, right,  
6 the radioactive label to acrylamide that they looked at and  
7 then collected on the filter paper?

8 A. Well, that's the demonstration of why it  
9 decreases.

10 I'm relying on products that are available on the  
11 market, such as Illy coffee and Starbucks coffee, in which  
12 coffee does not have to be consumed within one week or two  
13 weeks of which the proposal had been made that staling occurs  
14 within two weeks.

15 Q. Simple question, doctor.

16 You relied on the Baum study, correct?

17 A. As I mentioned, to demonstrate how it occurs.

18 Q. All right. And Baum said that the mechanisms  
19 underlying the loss of acrylamide during storage are as yet  
20 unknown, correct?

21 A. No.

22 They demonstrated that it's binding to the matrix.

23 Q. Okay. And what was the metabolite of acrylamide  
24 that you believed was the carcinogenic compound that was  
25 causing cancer in lab animals?

26 A. Glycidamide would be the primary. But there may  
27 also be some effects associated with acrylamide itself but  
28 primarily associated with glycidamide.

1 Q. Did Baum discuss whether the acrylamide in the  
2 stored coffee might, in fact, be oxidized into glycidamide?

3 A. No, that wouldn't happen. That's an enzymatic  
4 reaction.

5 Q. You don't recall them saying, "Further, since  
6 coffee has been reported to contain hydrogen peroxide, it is  
7 not unlikely that oxidation into the acrylamide-derived  
8 epoxide glycidamide might potentially also contribute to some  
9 extent to acrylamide loss"?

10 A. But that was a hypothesis. That's a  
11 speculation.

12 If they demonstrated it --

13 Q. But you would be concerned. You wouldn't want  
14 that to happen in a method that you are suggesting should  
15 reduce acrylamide, right?

16 MR. METZGER: Objection. As to what. What is "that to  
17 happen"?

18 THE COURT: Overruled.

19 THE WITNESS: I would not want to see that happen, but  
20 if you don't remove the acrylamide, it's going to form  
21 glycidamide in the consumer.

22 Q. BY MR. MARGULIES: But if at the end of the day  
23 it's simply forming glycidamide in the can, you haven't really  
24 achieved anything by storing it, correct?

25 A. It's a complex question because you're dealing,  
26 then, with distribution of glycidamide in the body from the  
27 coffee itself.

28 I don't know what would happen once you start to boil

1 coffee to make it a consumable product, what would happen to  
2 the glycidamide.

3 So it's not a simple question. It sounds simple, but  
4 it's not really a simple question to answer.

5 You need to look at a cup of coffee to see if there is  
6 any glycidamide in it, but I haven't seen any data to indicate  
7 that.

8 Q. So you've offered a simple solution, but there  
9 is a lot of complexity to it that would need to be resolved  
10 before the coffee company would adopt it to reduce acrylamide,  
11 right?

12 MR. METZGER: Objection, argumentative.

13 THE COURT: Overruled.

14 THE WITNESS: That would be easy to be done.

15 The analysis of stored coffee for glycidamide? That  
16 could be done in a week.

17 Q. BY MR. MARGULIES: Has it ever been done?

18 A. I don't work for the coffee companies. Maybe  
19 they have. I don't know.

20 Q. But you're here offering an opinion that this is  
21 a safe way to reduce acrylamide exposure, which really means  
22 glycidamide exposure, correct?

23 A. I would assume that Starbucks and Illy would not  
24 say you could store your coffee for 60 weeks or up to two  
25 years knowing that glycidamide is being formed.

26 MR. MARGULIES: Move to strike as non-responsive.

27 THE COURT: The motion is granted.

28 The last answer will be stricken.

1 MR. MARGULIES: No further questions. Thank you.

2 THE COURT: Anything further, Mr. Metzger? Last  
3 question.

4

5 **FURTHER REDIRECT EXAMINATION**

6 BY MR. METZGER:

7 Q. Dr. Melnick, have you seen any data whatsoever  
8 that glycidamide is formed in stored coffee?

9 A. No.

10 But let me address one thing, because it relates to the  
11 point you just made, that it might occur through hydrogen  
12 peroxides.

13 Those are products of lipid oxidation and when storing  
14 the coffee under a nitrogen atmosphere, those peroxide  
15 products don't form. You don't get the lipid oxidation.

16 So therefore it's not going to happen.

17 Q. Oh, I'll just end there.

18 MR. METZGER: Thank you, your Honor.

19 THE COURT: Thank you.

20 May Dr. Melnick be excused?

21 MR. MARGULIES: Yes, your Honor.

22 THE COURT: You may step down, Dr. Melnick.

23 THE WITNESS: Thank you.

24 THE COURT: Plaintiff have any further witnesses?

25 MR. METZGER: Not live witnesses.

26 Well, actually, we do.

27 THE COURT: Not the deposition testimony?

28 MR. METZGER: There is this additional witness who I

1 mentioned yesterday who we would like to call tomorrow. He's  
2 currently having -- undergoing a surgical procedure today so  
3 he's not available today. But he's available --

4 THE COURT: This is a newly discovered witness?

5 MR. METZGER: It's a newly discovered witness.

6 THE COURT: And defendants have not known about this  
7 witness either?

8 MR. KENNEDY: No. We've not known about him  
9 previously.

10 THE COURT: Is this a self-identified individual who  
11 has volunteered to become a part of this case?

12 MR. METZGER: He did contact me upon reading media  
13 reports of the case.

14 He is the assignee -- well, his company is the assignee  
15 of an acrylamide reduction technology which, as he described  
16 it to me, uses herbs to -- the initial goal was to make a more  
17 flavorful coffee, which was achieved.

18 And then, in doing further studies to see what  
19 resulted, there was the incidental finding of a substantial  
20 reduction of acrylamide.

21 THE COURT: And did you check out all this information  
22 that he gave you?

23 MR. METZGER: Well, I do have the patent application.  
24 Yes.

25 THE COURT: Okay. And has defendant had an opportunity  
26 to take his deposition?

27 MR. METZGER: No.

28 One thing that puzzled me is that the defendants'

1 production of documents did not include anything about this,  
2 although he had communications, written communications, with  
3 several of the defendants regarding this technique.

4 So had they produced that, I would have discovered him  
5 much earlier, but they did not.

6 THE COURT: And they have not taken his deposition?

7 MR. METZGER: No.

8 THE COURT: And supposing they take his deposition and  
9 then they decide, well, they need three experts to respond to  
10 him. Then what do we do?

11 This is a slippery slope we go to.

12 MR. METZGER: I do not intend to have him offer any  
13 expert testimony. I'm merely going to have -- largely have  
14 him offer party opponent admissions of certain defendants in  
15 this case.

16 THE COURT: Mr. Kennedy?

17 MR. KENNEDY: We filed a short brief on this this  
18 morning, your Honor.

19 Our principal concern is it's our understanding he will  
20 be trying to introduce hearsay conversations with some small  
21 number of the defendants, none of which, as far as I know, I  
22 represent, but I wouldn't be surprised that those people are  
23 then going to want the opportunity to respond to what he  
24 claims they said or didn't say.

25 Beyond that, we will withdraw our request for his  
26 deposition. We have no desire to delay the proceedings.

27 So at least remove that obstacle.

28 I think your Honor is in the best position to know what

1 would be helpful to you at this point.

2 THE COURT: The Court is going to exclude this witness.

3 The witness has not been previously identified, not  
4 been made available for deposition.

5 There is a concern that it is somebody who apparently  
6 has some other motivation, some other interest, an economic  
7 interest in some patent or some process that may or may not be  
8 relevant, and he's self-identified.

9 He's interested in volunteering to become a witness. I  
10 don't think at this stage of the proceedings it's appropriate.

11 The Court will exclude that witness.

12 All right. Mr. Metzger, any other witnesses besides  
13 those witnesses that are going to have their testimony  
14 reviewed through depositions?

15 MR. METZGER: Plaintiff has no other live witnesses.  
16 It's just the deposition excerpts of the defendants' PMKs.

17 Then there is also the discovery responses, likewise,  
18 to be reviewed by your Honor, but no one live. This is our  
19 last live witness now that you've excluded, Mr. Durand.

20 THE COURT: The defendants, any further witnesses?

21 MR. KENNEDY: Yes, your Honor. As we mentioned  
22 previously, we have a rebuttal witness from Covance who is  
23 here in the courtroom.

24 In addition, in light of Dr. Spingarn's testimony that  
25 even the FDA data is unreliable, we also served an offer of  
26 proof this morning offering Dr. Troxell, which would be a  
27 very, very short -- both of these would be very short true  
28 rebuttal responses to factual inaccuracies.

1 I think Mr. Metzger is going to hurt himself if I don't  
2 let him stand up, but those are our next two live witnesses.

3 THE COURT: All right. Are you ready to call your next  
4 witness?

5 MR. METZGER: Excuse me, your Honor.

6 They want to call this Dr. Mastovska as an undesignated  
7 expert.

8 THE COURT: Besides that. We'll get to that.

9 What about the witness in the courtroom?

10 MR. KENNEDY: That is the doctor.

11 THE COURT: Oh, I thought that was the second one.

12 MR. KENNEDY: Dr. Mastovska. And Dr. Troxell is in  
13 town. He's available. It will be very, very brief. It will  
14 be rebuttal to some errors of fact by Dr. Spingarn.

15 MR. METZGER: This is the first I'm hearing about  
16 Dr. Troxell.

17 Dr. Troxell is an expert who the defense previously  
18 submitted a declaration to the Court.

19 They chose not to designate him, and now they want to  
20 bring him in as a witness even though -- after concealing him  
21 and choosing not to designate him.

22 As a matter of fact, I filed a motion in limine to  
23 preclude all of the defendants' designated experts from  
24 relying on Dr. Troxell's opinions set forth in his report.

25 THE COURT: All right. Briefly with Dr. Troxell, I  
26 understand he's not being called to venture any opinions  
27 whatsoever.

28 The doctor is going to be called to impeach some

1 factual statements made by some witness?

2 MR. KENNEDY: Yes, it will be very brief.

3 If we go beyond true rebuttal, I'm sure your Honor will  
4 shut us down.

5 Frankly, we can hear it in less time.

6 THE COURT: Let's go back to the other witness.

7 MR. KENNEDY: Mr. Schurz is probably in the best  
8 position to answer questions about her.

9 THE COURT: Okay. Any problem with the other witness,  
10 that's Dr. -- what is the name again?

11 MR. SCHURZ: Mastovska, M-A-S-T-O-V-S-K-A.

12 MR. METZGER: Yes, there is a problem, your Honor.

13 As you recall, you directed Mr. Schurz to send me a  
14 communication, a letter, an email advising specifically what  
15 foundational facts that the defense claims are either false or  
16 non-existent Dr. Mastovska would testify to; that is, what  
17 facts relied upon by another expert who testified in this  
18 trial --

19 THE COURT: Yes.

20 MR. METZGER: -- are false or non-existent.

21 THE COURT: Just a second.

22 I just want to confirm that Dr. Mastovska is not going  
23 to render any new opinions, contrary opinions?

24 She's going to testify only as to factual issues  
25 concerning Covance?

26 MR. SCHURZ: Correct.

27 MR. METZGER: So Mr. Schurz sent me nine topics.

28 The first one was validation of the Covance method.

1 All of these are topics that Mr. Sullivan testified  
2 about and that also Dr. Spingarn testified about.

3 None of them are in the nature of -- he did not  
4 identify a single fact that Dr. Spingarn or any other expert  
5 testified about that is false or non-existent.

6 These are entirely opinions that contradict opinions.

7 In fact, most of her opinions that they are proposing  
8 to offer contradict sworn testimony of their designated  
9 expert, Mr. Sullivan.

10 I have prepared and submitted an in limine motion which  
11 lays all of this out. Every one of the nine topics are not in  
12 the nature of a false or non-existent predicate fact.

13 Every one of them is a contradict -- is an opinion that  
14 contradicts testimony offered by Mr. Sullivan and/or  
15 Dr. Spingarn.

16 THE COURT: That may be, but that's defendants'  
17 problem.

18 So Dr. Mastovska is going to offer impeachment  
19 testimony about what, specifically?

20 MR. SCHURZ: She will address eight specifics areas,  
21 your Honor.

22 She will testify that Dr. Spingarn told the incorrect  
23 formula to this Court when calculating the concentration of  
24 acrylamide.

25 You will recall, your Honor, he got up --

26 THE COURT: You don't have to go into more detail.

27 So the formula.

28 Next?

1 MR. SCHURZ: Dr. Spingarn provided and displayed a  
2 table, Exhibit 61939, that is factually incorrect with respect  
3 to the material that was provided there. He's just got it  
4 wrong.

5 It's pretty straightforward.

6 THE COURT: And how much time will be consumed by  
7 Dr. Mastovska's testimony?

8 MR. SCHURZ: Less than an hour.

9 THE COURT: The Court will allow the defendant to call  
10 Dr. Mastovska, again, not to provide any opinions. Just for  
11 the purpose of impeachment of any factual testimony.

12 MR. METZGER: Your Honor, while you're doing this, may  
13 I provide you with a copy of our in limine motion? Because  
14 I'm going to be referring to testimony that she's  
15 contradicting opinions.

16 THE COURT: Well, if she contradicts defendants'  
17 testimony, that's defendants' problem.

18 MR. METZGER: You don't want this?

19 THE COURT: Yes, you can present it.

20 I know you're thinking you were concerned about  
21 defendant impeaching themselves, but that's okay.

22 All right. Dr. Melnick, please step down from the  
23 stand.

24 Mr. Schurz.

25 MR. SCHURZ: Thank you, your Honor.

26 MR. METZGER: Your Honor, do I get to depose this  
27 witness?

28 THE COURT: It is just for impeachment of the

1 plaintiff's witnesses, not impeachment of defendants'  
2 witnesses.

3 MR. METZGER: Foundational facts.

4 THE COURT: Right.

5 MR. SCHURZ: Your Honor, at this time we would call  
6 Katerina Mastovska.

7 THE CLERK: Please raise your right hand.

8  
9 KATERINA MASTOVSKA,  
10 having been called as a witness and sworn testified as  
11 follows:

12 THE WITNESS: Yes, I do.

13 THE CLERK: And can you please state and spell your  
14 name for the record.

15 THE WITNESS: My first name is Katerina,  
16 K-A-T-E-R-I-N-A. And my last name is Mastovska,  
17 M-A-S-T-O-V-S-K-A.

18 THE CLERK: Thank you.

19 THE COURT: Good morning, Dr. Mastovska.  
20 And, Mr. Schurz, you may proceed.

21 MR. SCHURZ: Thank you, your Honor.

22

23 **DIRECT EXAMINATION**

24 BY MR. SCHURZ:

25 Q. Good morning, Dr. Mastovska.

26 A. Good morning.

27 Q. And let me ask you, if you would, to lean into  
28 the microphone that's there in front of you so we can all hear

1 you.

2 Dr. Mastovska, I've provided you with a binder, and to  
3 opposing counsel as well as the Court, with a set of exhibits  
4 that we may be referring to over the course of our brief  
5 discussion. And you should feel free to refer to those as we  
6 discuss them.

7 But with that as an orientation, can I ask you to  
8 describe for the Court your current position at Covance?

9 MR. METZGER: Objection. She doesn't need to be  
10 qualified as an expert. She's not testifying as an expert.

11 THE COURT: Well, let's hear about what she does for a  
12 living.

13 Go ahead, Mr. Schurz.

14 THE WITNESS: I'm associated at Covance Solutions, and  
15 I lead the global chemistry research development and  
16 innovation group.

17 Q. BY MR. SCHURZ: Now, let's turn specifically to  
18 the work that you've done with respect to the analysis of the  
19 defendants' coffee products by Covance.

20 Can you tell this Court when you became involved in  
21 Covance's work relating to the acrylamide testing in  
22 defendants' brewed coffee products?

23 MR. METZGER: Objection, CCP 2034.310(b). This is not  
24 going to a foundational fact.

25 THE COURT: This is just merely background. We'll get  
26 to the testimony as to this case in a moment.

27 THE WITNESS: Should I answer?

28 THE COURT: You may answer the question.

1 THE WITNESS: Okay. I got first involved on July 19th  
2 of this year.

3 Q. BY MR. SCHURZ: And how did you become involved  
4 in the work that Covance performed relating to defendants'  
5 brewed coffee products?

6 A. I was contacted by Julie Lowe who is the  
7 technical leader and by Ben Abel who is the supervisor in the  
8 group who performed the testing.

9 And they asked me to review calculations performed in  
10 this case.

11 Q. And did you, in fact, review the calculations of  
12 the acrylamide concentrations for the defendants' coffee  
13 products in this matter?

14 A. Yes, I did.

15 Q. And as a result of that review, what did you do?

16 MR. METZGER: Objection. This is not going to --

17 THE COURT: Overruled.

18 THE WITNESS: I confirmed that there was a calculation  
19 issue, and I also was involved in the generation of the CAPA,  
20 C-A-P-A. It's the corrective action/preventative action.

21 Q. And directing your attention in the binder to  
22 Exhibit DX 72470, can you identify this document for us.

23 A. Yes, I can. That is the corrective  
24 action/preventative action document.

25 Q. Now, as part of your review of the calculations,  
26 did you check on how the calculation concentration value set  
27 out in the Covance data sheets was developed?

28 A. Yes, I did.

1 Q. All right. And we'll come back to that in a  
2 moment.

3 In addition to your work on the corrective  
4 action/preventative action plan, did you have any other  
5 involvement with respect to the coffee products in this case  
6 following your involvement in July of 2017?

7 A. Yes. I also reviewed a revised supplemental  
8 validation report.

9 Q. And directing your attention to, in the binder,  
10 72484, can you identify this document for us?

11 A. Yes. That's the document which I reviewed and  
12 revised, and my revisions were implemented.

13 I also reviewed the data tables provided in this  
14 report.

15 Q. All right. Thank you.

16 With that context, then, I would like to turn now  
17 specifically to certain statements that were made by Dr. Neil  
18 Spingarn in his testimony.

19 Showing you first Dr. Spingarn's testimony at 164,  
20 lines 16 to 24.

21 And did you review the testimony of Dr. Neil Spingarn  
22 in your preparation for your testimony today?

23 A. Yes, I did review it.

24 Q. Directing your attention to the specific  
25 statement made at page 164, line 16 through 24, it reads as  
26 follows:

27 "Q. All right. And did you review a  
28 series of these?

1 "A. Yes.

2 "Q. And what did you find?

3 "A. I found that they had corrected the  
4 unit issue so that the units in the lower  
5 left box are now micrograms per  
6 milliliter, as they should have been in  
7 the first set. But I also noticed what  
8 appears to be a calculation error in  
9 these sheets."

10 Did you review that testimony?

11 A. Yes, I did.

12 Q. As a factual matter, did Covance make a  
13 calculation error?

14 MR. METZGER: Objection. This is a contrary opinion.  
15 It's not a factual foundation.

16 THE COURT: Overruled.

17 THE WITNESS: No, we did not.

18 Q. BY MR. SCHURZ: Showing you the exhibit that  
19 Dr. Spingarn was relying on, which is Exhibit 61941 -- do you  
20 have that in front of you?

21 A. Yes, I have.

22 Q. Now, Dr. Spingarn started with the premise that  
23 calculating the concentration is a fairly simple calculation  
24 and that everything you need to perform that calculation is  
25 present on this sheet, referencing 61941.

26 MR. METZGER: Objection. I object to Mr. Schurz's  
27 characterization Dr. Spingarn's testimony.

28 He's mischaracterizing it and including it in the

1 question to the witness.

2 THE COURT: Yes. Please avoid doing that.

3 You can ask the witness. And if the witness has  
4 different information about Dr. Spingarn, she can so state.

5 Q. BY MR. SCHURZ: As a factual matter, is  
6 Dr. Spingarn correct that everything he needed to arrive  
7 at --

8 MR. METZGER: Objection.

9 THE COURT: Objection sustained.

10 Please phrase the questions where you do not have the  
11 witness commenting on some other witness's testimony.

12 Q. BY MR. SCHURZ: Did Dr. Spingarn --

13 THE COURT: Again, go to facts, not whether they agree  
14 or disagree with some other witness.

15 MR. SCHURZ: I understand.

16 Q. Did Dr. Spingarn use the correct formula?

17 A. No, he did not.

18 MR. METZGER: Objection. This is contradicting his  
19 opinion.

20 THE COURT: Again, just -- you can ask the witness as  
21 to some specific factual statement that you believe is  
22 inconsistent with the previous testimony but not to argue  
23 about whether the witness agrees or disagrees or whether the  
24 witness is right or wrong.

25 Q. BY MR. SCHURZ: Is all the information necessary  
26 to calculate the acrylamide concentration available on 61941?

27 MR. METZGER: Objection. This is contradicting an  
28 opinion.

1 THE COURT: Overruled.

2 You may answer the question.

3 THE WITNESS: No, it's not.

4 Q. BY MR. SCHURZ: What is missing?

5 A. What's missing is the calculation equation which  
6 is needed to calculate the concentration of acrylamide in the  
7 extracts.

8 Q. Showing you 73517.

9 Can you tell us what this document is?

10 A. This is the calibration which relates to that  
11 batch for the sample which was shown on the previous screen.

12 Q. And where is the relevant formula required for  
13 calculating the acrylamide concentration necessary for the  
14 data sheet 61941?

15 MR. METZGER: Your Honor, this is all beyond the scope  
16 of the --

17 THE COURT: Overruled.

18 THE WITNESS: The formula is in the upper left corner.

19 If you could zoom on it. So you can see it here.

20 That's an integration equation which I won't read, but  
21 this is the equation that is used for calculation in this case  
22 and in this matter.

23 Q. BY MR. SCHURZ: Did Dr. Spingarn use this  
24 formula in calculating the acrylamide concentration values?

25 A. No, he did not.

26 Q. All right. So did Dr. Spingarn use the correct  
27 formula for calculating the concentration values in these data  
28 sheets?

1 MR. METZGER: Objection. He's seeking a contrary  
2 opinion.

3 THE COURT: Overruled.

4 THE WITNESS: No, he did not.

5 Q. BY MR. SCHURZ: All right. Let me turn to the  
6 second issue relating to Dr. Spingarn's testimony.

7 I would show you Exhibit 61939, the Covance modified  
8 protocol demonstrative that Dr. Spingarn provided to this  
9 Court.

10 Have you reviewed this document?

11 A. Yes, I have.

12 Q. Directing your attention to the lower right-hand  
13 corner in which 61939 states with respect to validation as  
14 with respect to the column "Will Be Washed," Dr. Spingarn  
15 states, "None. Ineffective. Retention at start of June 21st  
16 data equals 4.0 minutes. Retention at start of June 22nd data  
17 equals 5.3 minutes."

18 Do you have that in mind?

19 A. Yes, I can see it. I have it in mind.

20 Q. Let's start with the retention times from the  
21 June 22nd that are referenced here.

22 Did you try to look up to determine whether the data  
23 and retention times that are reflected in Dr. Spingarn's  
24 exhibit are accurate with respect to June 22nd?

25 MR. METZGER: Objection. It's a contrary opinion.

26 THE COURT: Overruled.

27 THE WITNESS: Yes, I tried.

28 Q. BY MR. SCHURZ: And what did you find?

1 A. I couldn't find his data.

2 Q. Did you find a retention time of 5.3 minutes, as  
3 stated by Dr. Spingarn in Exhibit 61939?

4 A. No, I did not.

5 Q. Why not?

6 MR. METZGER: Well, objection. Now she's offering an  
7 opinion.

8 THE WITNESS: No, it's not an opinion.

9 THE COURT: Hey, wait. Your job is to answer  
10 questions, not to argue with the lawyer.

11 The objection is overruled.

12 Next question.

13 Q. BY MR. SCHURZ: How is it that you did not find  
14 the 5.3-minute retention times at the start of the June 22nd  
15 data?

16 A. Because no data were required on June 22nd in  
17 this case.

18 Q. How do you know that?

19 A. I reviewed all data. I put it all together and  
20 was trying to find acquisition date on June 22nd.

21 I also confirmed with Mr. Ladd, with Julie Lowe and Ben  
22 Abel.

23 MR. METZGER: Objection. It's hearsay.

24 THE WITNESS: It's not hearsay.

25 THE COURT: Dr. Mastovska, your job is to answer  
26 questions, not to argue.

27 THE WITNESS: I'm sorry.

28 THE COURT: Do you understand that?

1 THE WITNESS: Yes.

2 I apologize.

3 THE COURT: Please repeat the question.

4 Q. BY MR. SCHURZ: Dr. Mastovska, how did you  
5 confirm that there was no testing performed on June 22nd?

6 A. I reviewed the data myself.

7 Q. Thank you.

8 Now, let's turn to Dr. Spingarn's statement with  
9 respect to June 21.

10 He states, "The retention at start of June 21 data  
11 equals 4.0 minutes."

12 Do you see that?

13 A. Yes, I can see that.

14 MR. METZGER: Objection, offering a contrary opinion.

15 THE COURT: Overruled.

16 THE WITNESS: Can you repeat the question, please?

17 Q. BY MR. SCHURZ: Is that a correct statement,  
18 that the retention time at the start of the June 21 data is  
19 4.0 minutes?

20 A. No, it's not a correct statement.

21 Q. And what was the retention time for the testing  
22 that was performed on June 21?

23 A. The retention time for the first injection on  
24 June 21st was 4.5 minutes.

25 Q. And directing your attention in the binder to  
26 Exhibit 72344.065, can you identify this document for us?

27 A. Yes. That's the worksheet. That's the printout  
28 for the -- for the first injection date on June 21st.

1 Q. And what is the retention time for the first  
2 sample on 6/21/2017?

3 A. The retention time for acrylamide is  
4 4.5 minutes.

5 Q. All right. So with respect to this value, if we  
6 could go back Dr. Spingarn's table, 61939, so is it the case,  
7 Dr. Mastovska, that Dr. Spingarn's statement that the  
8 retention time at the start of June 21 data of four minutes is  
9 incorrect?

10 MR. METZGER: Objection. He's expressly seeking a  
11 contrary opinion.

12 THE COURT: Overruled.

13 THE WITNESS: Yes, that's incorrect.

14 Q. BY MR. SCHURZ: All right. Now, so having  
15 determined what the retention time was for the first sample on  
16 June 21st, when was the next testing performed with respect to  
17 the defendants' coffee products at Covance?

18 A. The next testing was performed on June 27th.

19 Q. And what was the retention time for the first  
20 sample tested on June 27th?

21 MR. METZGER: Objection. This is not addressing any  
22 opinion rendered by an expert, any fact testified to by an  
23 expert.

24 THE COURT: I don't know what that is addressing in  
25 terms of any prior correction of somebody else or  
26 contradiction.

27 Objection sustained.

28 Q. BY MR. SCHURZ: All right. So what we have here

1 is a statement in both respects as to the retention time that  
2 are incorrect from Dr. Spingarn, correct?

3 MR. METZGER: Objection. Contrary opinion.

4 THE COURT: Overruled.

5 You may answer.

6 THE WITNESS: Can you repeat the question, please.

7 Q. BY MR. SCHURZ: Yes.

8 Both statements by Dr. Spingarn in Exhibit 61939 with  
9 respect to the retention times are factually incorrect?

10 A. Yes, they are.

11 Q. All right. Now, what was the start time --  
12 strike that.

13 What was the retention time for the next start of  
14 testing following the June 21st data?

15 A. It was 4.38 minutes which was on June 27th.

16 Q. And what is the consequence or significance of  
17 the retention times that you have now testified to with  
18 respect to June 21 and June 27?

19 MR. METZGER: Objection. Now he's asking for an  
20 opinion which has not anything to do with the factual  
21 predicate.

22 THE COURT: The objection is sustained.

23 Q. BY MR. SCHURZ: Was Dr. Spingarn correct in his  
24 statement that the retention times reflected that the  
25 equipment was not operating correctly?

26 MR. METZGER: Objection, seeking a contrary opinion.

27 THE COURT: Sustained.

28 The way the question is phrased, it's sustained.

1 MR. SCHURZ: All right.

2 Q. Let me turn to the retention times and a third  
3 area of Dr. Spingarn's testimony.

4 Here I would direct, Dr. Mastovska, your attention to  
5 page 58, lines 1 through 13, of Dr. Spingarn's testimony.

6 "Q. You mean where temporally?

7 "A. In time.

8 "That is, you have a ten-minute run.  
9 You start your injection. How long does  
10 it take for the peak, the internal  
11 standard, to come out the other end of  
12 the machine. That has to be extremely  
13 consistent also for the system to be in  
14 control.

15 "In these two cases, the June 21st  
16 and June 22nd, the June 21st peaks came  
17 out at 5.3 minutes. The June 22nd came  
18 out at 4.0 minutes.

19 "We have a tremendous change in the  
20 retention time. That means the  
21 chromatography isn't working. That's the  
22 first half of the machine."

23 Do you have that testimony in mind?

24 A. Yes, I have.

25 Q. Did you examine any retention time data to  
26 determine if the chromatography method was working during the  
27 time period being referenced here by Dr. Spingarn?

28 MR. METZGER: Objection, contrary opinion.

1 THE COURT: Overruled.

2 THE WITNESS: Yes.

3 In addition to those you just mentioned, I also  
4 reviewed the retention times for both the acrylamide peak and  
5 the internal standard peak across all batches.

6 Q. BY MR. SCHURZ: And what did you find?

7 MR. METZGER: Objection. This is not a factual  
8 predicate. She is now testifying as to what she found.

9 THE COURT: Objection sustained.

10 Q. BY MR. SCHURZ: And in looking in your analysis  
11 with respect to the change that was purportedly found by  
12 Dr. Spingarn, what did you find?

13 MR. METZGER: Same objection.

14 THE COURT: Objection sustained.

15 Q. BY MR. SCHURZ: Now, did you find that there was  
16 a tremendous change in the retention time from the period of  
17 June 21st to the next actual testing of samples in this case?

18 MR. METZGER: Objection.

19 THE COURT: Objection sustained.

20 MR. SCHURZ: All right.

21 Q. So let me turn to another area of testimony of  
22 Dr. Spingarn.

23 Now I would like to show you the second of his two  
24 demonstratives that he provided which you will find in your  
25 binder at 16940. It's the second tab.

26 Do you have that in front of you?

27 A. Yes, I have.

28 Q. Is the factual foundation upon which this

1 demonstrative rests correct?

2 MR. METZGER: Objection. That's asking for an opinion.

3 THE COURT: Objection sustained.

4 Q. BY MR. SCHURZ: Are the facts upon which  
5 Dr. Spingarn relies in depicting on Exhibit 61940 correct?

6 MR. METZGER: Objection, vague as to what facts.

7 THE COURT: Objection sustained.

8 Q. BY MR. SCHURZ: Let's try it this way.

9 Do you see the title "Internal Standard Response  
10 Factors"?

11 A. Yes, I see it.

12 Q. Does this Exhibit 61940 accurately depict the  
13 internal standard response factors?

14 MR. METZGER: Objection.

15 THE COURT: I think we'll take the recess, and I would  
16 ask counsel to discuss this over lunch.

17 I think we have some miscommunication here. The Court  
18 allowed the witness to testify for impeachment purposes only.

19 That is, if a witness came in and said that  
20 January 15th, 1965, was a Thursday and gave some opinion based  
21 on that, then a witness could come in and say, no,  
22 January 15th is not a Thursday. It was a Wednesday.

23 That is an underlying foundational fact that is in  
24 dispute but the purpose was not to call a witness to render a  
25 whole bunch of additional opinions to bootstrap positions of  
26 the parties.

27 So think about what a foundational fact is to support  
28 some other witness as opposed to opinion.

1 We'll be in recess until 1:30.

2 (At 12:00 noon a recess was taken until.

3 1:30 p.m. of the same day.)

4

5

6

7

8

9

10

11

12

13

14

15

16

17

18

19

20

21

22

23

24

25

26

27

28

1 SUPERIOR COURT OF THE STATE OF CALIFORNIA

2 FOR THE COUNTY OF LOS ANGELES

3 DEPARTMENT 323

HON. ELIHU M. BERLE, JUDGE

4

5 CERT, )  
6 )  
7 ) Plaintiff, )  
8 )  
9 ) vs. )  
10 )  
11 ) STARBUCKS CORP, ET AL., )  
12 )  
13 ) Defendants. )  
14 )  
15 )  
16 )  
17 )  
18 )  
19 )  
20 )  
21 )  
22 )

---

SUPERIOR COURT  
CASE NO. BC 435759  
BC 461182

12 I, DAVID A. SALYER, Official Pro Tem Reporter of the  
13 Superior Court of the State of California, for the County of  
14 Los Angeles, do hereby certify that the foregoing pages, 1  
15 through 94, inclusive, comprise a true and correct transcript  
16 of the proceedings taken in the above-entitled matter reported  
17 by me on October 3, 2017.

18 DATED: October 3, 2017.

22 \_\_\_\_\_  
23 DAVID A. SALYER, CSR, RMR, CRR  
24 Official Pro Tem Court Reporter  
25 CSR No. 4410  
26  
27  
28

# **EXHIBIT “F”**

## CURRICULUM VITAE – Ronald L. Melnick

**Address:** [REDACTED] [REDACTED]  
[REDACTED], UT [REDACTED] [REDACTED], CT [REDACTED]

**Phone:** Home: 435-[REDACTED] 203-[REDACTED]  
Mobile: 919-[REDACTED]

**Email:** ron.melnick@gmail.com

**Date & Place of Birth:** May 19, 1943, New York, NY

**Citizenship:** United States

### Education:

- 1965 B.S. (Food Science) Rutgers University, New Brunswick, NJ
- 1967 M.S. (Food Science/Biochemistry) University of Massachusetts, Amherst, MA. Thesis: *A Study of Bound and Solubilized Lactate Dehydrogenase in Skeletal Muscle.*
- 1970 Ph.D. (Food Science/Biochemistry) University of Massachusetts, Amherst, MA. Thesis: *Cellular Organization. A Study of Glycolytic Enzymes in Skeletal Muscle.*

### Chronology of Employment:

- 1970 - 1973 Postdoctoral fellow, Department of Physiology-Anatomy, University of California, Berkeley, CA.
- 1973 - 1980 Assistant Professor of Life Sciences, Polytechnic Institute of New York, Brooklyn, NY.
- 1980 – 1990 Toxicologist, National Toxicology Program, National Institute of Environmental Health Sciences, Research Triangle Park, NC. Study scientist for NTP toxicology/carcinogenesis studies on 1,3-butadiene, bromoform, chloroacetophenone, chloroprene, chlorpheniramine, 2,4-dichlorophenol, diethanolamine, diphenhydramine, isoprene, melamine, phthalate esters, succinic anhydride.
- 1981 – 1990 Project Officer, National Toxicology Program, NIEHS:

- 1) Twelve toxicology/ carcinogenicity studies at Physiological Research Labs., Minneapolis, MN;  
 2) Development and use of an in vitro system for the study of toxicity in renal tubules from several mammalian species, SRI International, Menlo Park, CA
- 1985 - 1989      Manager, Experimental Toxicology Unit, Carcinogenesis and Toxicology Evaluation Branch, NIEHS
- 1990 – 1993      Toxicologist, Division of Biometry and Risk Assessment, NIEHS
- 1993 – 1995      Toxicologist, Environmental Carcinogenesis Program, NIEHS,
- 1995 - 1996      Agency Representative in the White House Office of Science and Technology Policy (Environment Division), Washington, DC.
- 1995 – 2000      Group Leader, Toxicokinetics and Biochemical Modeling Group, Laboratory of Computational Biology and Risk Analysis, Environmental Toxicology Program, NIEHS
- 2001- 2008      Director of Special Programs, Environmental Toxicology Program, NIEHS. Identification and characterization of the potential health effects of cell phone radiofrequency radiation, perfluorinated chemicals, and drinking water disinfection byproducts.
- 2001-2008      Project Officer, NTP/NIEHS: 1) Interagency Agreement with the National Institute of Standards and Technology (Boulder, CO) on “Determining the potential hazards of exposures to radio frequencies generated during the use of cellular phones “  
 2) IIT Research Institute (Chicago, IL) on “Studies to evaluate the toxic and carcinogenic potential of cell phone radio frequency radiation in laboratory animals for the National Toxicology Program”
- 2001-2002      Consultant to the Attorney General of the State of California, concerning cancer risk of di(2-ethylhexyl)phthalate.
- 2006-2009      Consultant to the Attorney General of the State of California on cancer risks associated with dietary exposure to acrylamide.
- 2009-              Independent Consultant, Ron Melnick Consulting LLC

**Awards and Honors:**

Tuition Scholarship from the New York Division of the Institute of Food Technologists,

1964-1965

Sigma Xi, Phi Kappa Phi, Alpha Zeta, Phi Tau Sigma

NIEHS representative in the U.S.-Japan Non-Energy Research and Development Program, 1985

Cited in: American Men and Women of Science

Selected for a one-year appointment to work on risk assessment issues at the White House Office of Science and Technology Policy, 1995-1996

Elected to the Council of Fellows of the Collegium Ramazzini, 1996

Commendations for Sustained High Quality Work Performance, NIEHS, numerous dates

NIH Merit Award for outstanding accomplishments as a member of the NIEHS/NTP Review Committee for the Report on Carcinogens, 2000

Cited in: Who's Who in America, 58<sup>th</sup> Edition, 2003

NIH Plain Language Award 2005, for the NCI/NIEHS brochure "Cancer and the Environment: What You Need to Know, What You Can Do"

2007 David P. Rall Award for Advocacy in Public Health from the American Public Health Association

2008 NIH Merit Award

## **Professional Activities:**

### ***NIEHS Committees:***

Toxicokinetics Faculty, Chairman 1996-2000

Review group for the NTP Report on Carcinogens, Chairman 2005-2008

Chemical Nominations Committee

Project Design and Evaluation teams for NTP chemicals or toxicological issues

NTP Project Review Committee

Committee on Promotions II

Committee for the Development of the NTP Vision

### ***Journal Reviewer:***

Cancer Research

Carcinogenesis

Critical Reviews in Toxicology

Environmental Health Perspectives (Editorial Board 1991-1997)

Environmental Health (Editorial Board)

Environmental and Molecular Mutagenesis  
Fundamental and Applied Toxicology/Toxicological Sciences  
International Journal of Occupational and Environmental Health (Editorial Board)  
Journal of the National Cancer Institute  
Toxicology  
Toxicology and Applied Pharmacology  
Toxicology and Industrial Health

***Symposium/Workshop Organizer:***

International Symposium on the "Toxicology, Carcinogenesis, and Human Health Aspects of 1,3 Butadiene," Research Triangle Park, NC, April 12-13, 1988. Editor of the symposium proceedings published in Environ. Health Perspect. 86: 1-171, 1990.

International Symposium on "Cell Proliferation and Chemical Carcinogenesis," Research Triangle Park, NC, January 14-16, 1992. Editor of the symposium proceedings published in Environ. Health Perspect. 101 (suppl. 5): 1-285, 1993.

Workshop on "Colorectal Cancer: Trihalomethanes and other Environmental Factors," Research Triangle Park, NC, September 14, 1993. Workshop report published in Environ. Health Perspect. 102: 586-588, 1994.

Workshop on "Characterizing the Effects of Endocrine Disruptors on Human Health at Environmental Exposure Levels," Raleigh, NC, May 11-13, 1998. Editor of the workshop proceedings published in Environ. Health Perspect. 107 (suppl. 4): 601-649, 1999.

Co-chair of session on Use of Mechanistic Data in Risk Assessment: Human Variability and Susceptibility in Risk Assessment, conference on "Toxicology and Risk Assessment Approaches in the 21<sup>st</sup> Century," King's Island, OH, April 10-13, 2000.

Organizing Committee for the international symposium: on "Evaluation of Butadiene, Isoprene and Chloroprene Health Risks," London, September 2000.

Organizing Committee (Chair) for the NTP/NIEHS Endocrine Disruptors Low-Dose Peer Review, Research Triangle Park, NC, October 2000.

***Invited Member of Scientific Review/Advisory Panels:***

Working group of the International Agency for Research on Cancer that prepared the "IARC Monograph on the Evaluation of Carcinogenic Risks to Humans: Volume 54: Strong Acid Mists and Some Other Industrial Exposures," Lyon, France, October 1991.

Working group of the International Program on Chemical Safety that prepared the IPCS Environmental Health Criteria document titled "Scientific Principles for Assessment of Human Health Risks Associated with Exposures to Chemicals," Surrey, England, March 1992.

Butadiene subgroup of the Health Effects Institute Workshop on Mobile Air Toxics that prepared the HEI Communications document "Research Priorities for Mobile Air Toxics," Monterey, CA, December 1992.

International Symposium on Health Hazards of Butadiene and Styrene, Espoo, Finland, April, 1993: Editorial Board for the Symposium Proceedings published in the IARC Scientific Publications Series, No. 127, 1993; Rapporteur for session on Dose Estimation

National Toxicology Program Workshop on Mechanism-Based Toxicology in Cancer Risk Assessment: Implications for Research, Regulation, and Legislation. Working group: Mechanism-based toxicology for species extrapolation, Chapel Hill, NC, January 1995.

Working group of the International Agency for Research on Cancer that prepared the "IARC Monograph on the Evaluation of Carcinogenic Risks to Humans, on Dry Cleaning, Some Chlorinated Solvents and Other Industrial Chemicals," Volume 63, Lyon, France, February 1995.

Risk Assessment Advisory Committee of the Office of Environmental Health Hazard Assessment's Science Advisory Board, California Environmental Protection Agency. The committee's report "A Review of the California Environmental Protection Agency's Risk Assessment Practices, Policies, and Guidelines" was completed in 1996.

Interagency Task Force for the Assessment of the Health Effects of Oxygenated Fuels for the White House Office of Science and Technology Policy and the preparation of the National Science and Technology Council's report "Interagency Assessment of Potential Health Risks Associated with Oxygenated Gasoline" 1996.

Endocrine Disruptor Working Group of the National Science and Technology Council's Committee on Environment and Natural Resources that prepared the documents "The Health and Ecological Effects of Endocrine Disrupting Chemicals: A Framework for Planning," "Endocrine Disruptors: Research Needs and Priorities, 1998" and that created the Federal Endocrine Disruptor Inventory.

Working group of the International Agency for Research on Cancer that prepared the Consensus Report "Species Differences in Thyroid, Kidney and Urinary Bladder Carcinogenesis," IARC Scientific Publication No. 147. Lyon, France, November 1997.

ILSI Expert Panel's Evaluation of EPA's Proposed Guidelines for Carcinogen Risk Assessment Using Chloroform and Dichloroacetate as Case Studies Canadian Environmental Health Assessment on 1,3-Butadiene, September 1997.

Consultant to the Science Advisory Board Environmental Health Committee's review of EPA's health risk assessment of 1,3-butadiene. Washington, DC, April 1998.

Member of the National Occupational Research Agenda (NORA) subgroup on Cancer Research Methods that is charged with identifying research needs that will address occupational cancer risks and lead to improved worker safety, 1998-2002.

Invited technical consultant to EPA's Federal Advisory Committee on Cancer Health Effects of Disinfection Byproducts (DBPs). Presented a "Perspective on Toxicology Data and DBP Cancer Health Risk." Washington, DC. May 1999.

Toxicology and Risk Assessment working group for the NIOSH workshop on Future Research for Improving Risk Assessment Methods. Aspen CO. August 16-18, 2000.

Member of the National Drinking Water Advisory Council Working Group on Drinking Water Research. This group will assist EPA in identifying and prioritizing drinking water research needs to support drinking water regulatory activities. 2000.

Member of the NCI and NIEHS group of scientists (2001-2003) that prepared the public information booklet "Cancer and the Environment: What you need to know and what you can do". US DHHS, NIH Publication No. 03-2039, 2003.

Reviewer for EPA's proposed research program on "Evaluation and prioritization of genetic and molecular events as biomarkers of carcinogenicity and comparison of the molecular biology of cancer in humans and laboratory animals." Cincinnati, OH, November 2001.

Member of EPA's Science Advisory Board to review the document "Trichloroethylene Health Risk Assessment: Synthesis and Characterization." Washington, DC, 2002

Member of the International Advisory Committee and the Research Coordination Committee of WHO's International Electromagnetic Fields Project. Geneva, SW, June 2003.

Participant in the WaterReuse Foundation's Water Reuse Research Needs Workshop. San Diego, CA, February 2004.

Participant/consultant for the UAW-Ford Peer Review of cohort mortality and leukemia case-control studies of workers in metal stamping and transmission plants. Detroit, MI, February 2004.

Member of the North Carolina Science Advisory Board on Toxic Air Pollutants. North Carolina Department of Environment and Natural Resources. 2004-2006.

External peer reviewer of EPA's Revised Technical Review of Diisononyl Phthalate. 2004.

External peer reviewer of grant proposals on non-ionizing radiation submitted to the Danish Research Agency, Copenhagen, Denmark. 2004 and 2005.

Member of EPA's Science Advisory Board for review of "Draft Risk Assessment of the Potential Human Health Effects Associated with Exposure to Perfluorooctanoic Acid and its Salts." Washington, DC, 2005.

Member of Planning Committee and Participant in ILSI-Health and Environmental Sciences Institute Workshop on "Improving the use of quantitative pharmacokinetic methods to determine dosimetry for evaluating human health risks." Research Triangle Park, NC, 2005.

Member of the Federal Interagency Working Group on "Pharmaceuticals in the Environment." Lead for the chapter on potential human health effects, 2005 – 2007.

Working group member (chair of the section on mechanistic considerations) of the International Agency for Research on Cancer expert panel that prepared the "IARC Monograph on the Evaluation of Carcinogenic Risks to Humans, on 1,3-Butadiene, Ethylene Oxide and Vinyl Halides (Vinyl Fluoride, Vinyl Chloride and Vinyl Bromide)" Volume 97, Lyon, France, June 2007.

Reviewer of the report Science and Decisions: Advancing Risk Assessment from the National Research Council's Board on Environmental Studies and Toxicology. The report was written by the Committee on Improving Risk Analysis Approaches Used by the US EPA. March 2008.

Member of the expert advisory panel for California Chemicals Policy and Breast Cancer Project. 2009. "Pathways to breast cancer: A case study for innovation in chemical safety evaluation."

Member of the expert review panel for the Health Risk Assessment of Methyl Iodide for the California Environmental Protection Agency, Department of Pesticide Regulation, 2009-2010.

Working group member (co-chair of the section on mechanistic considerations) of the International Agency for Research on Cancer for the preparation of volume 100 of the IARC Monograph on the Evaluation of Carcinogenic Risks to Humans on Chemical Agents and Related Occupations. Lyon, France, 2009.

Reviewer of the National Research Council's report Review of EPA's Draft IRIS Assessment on Tetrachloroethylene written by the NRC's Board on Environmental Studies and Toxicology, October 2009.

Member of the Integrated Risk Information System (IRIS) external peer review panel of US EPA's "Toxicological Review of Trichloroacetic Acid." Washington, DC, 2009.

Member of the IRIS external peer review panel of US EPA's "Toxicological Review of Chloroprene." Washington, DC, 2010.

Member of the External Advisory Board of the European Commission-supported project: Sound Exposure and Risk Assessment of Wireless Network Devices, 2009-2012.

Reviewer for the European Commission's Seventh Framework Programme: "Network for Environmental Chemical Toxicants Affecting Reproduction" 2010.

Working group member of the International Agency for Research on Cancer for the preparation of volume 101 of the IARC Monograph on the Evaluation of Carcinogenic Risks to Humans: Some Chemicals in Industrial and Consumer Products, Food Contaminants and Flavourings, and Water Chlorination By-Products. Lyon, France, 2011.

Working group member (chair of the section on exposure) of the International Agency for Research on Cancer for the preparation of volume 102 of the IARC Monograph on the Evaluation of Carcinogenic Risks to Humans: Non-Ionizing Radiation, Part II: Radiofrequency Electromagnetic Fields [includes mobile telephones]. Lyon, France, 2011.

Member of the IRIS external peer review panel of US EPA's "Toxicological Review of 1,4-Dioxane." 2011-12.

Invited participant to the International Agency for Research on Cancer workshops on "Tumor Concordance and Mechanisms of Carcinogenesis." Lyon, France, April and November, 2012.

Working group member (chair of the section on cancer studies in experimental animals) of the International Agency for Research on Cancer for the preparation of volume 106 of the IARC Monograph on the Evaluation of Carcinogenic Risks to Humans: Trichloroethylene and other chlorinated agents. Lyon, France, 2012.

Reviewer for the European Commission's Seventh Framework Programme: "Closing gaps of knowledge and reducing exposure to electromagnetic fields (EMF)". Brussels, Belgium, 2013.

Member of the external review panel of US EPA's "TSCA Workplan Chemical Risk Assessment for Trichloroethylene." 2013.

Consultant to the project "Protecting Human Health from Cumulative Effects of Exposure to Multiple Fumigant Pesticides." Funded by the Clarence E. Heller Charitable Foundation: Environmental and Health Program, 2015-2016.

Working group member of the International Agency for Research on Cancer for the preparation of volume 115 of the IARC Monograph on the Evaluation of Carcinogenic Risks to Humans: Some Industrial Chemicals. Lyon, France, 2016.

### **Invited lectures since joining NIEHS in 1980:**

Melnick, R.L. (1981). Mitochondrial toxicity of phthalate esters. National Toxicology Program/Interagency Regulatory Liaison Group Conference on Phthalates. Washington, D.C.

Melnick, R.L. (1983). Toxicity of ethylene glycol and ethylene glycol monoethyl ether in F344 rats and B6C3F1 mice. NIOSH Symposium on Toxic Effects of Glycol Ethers. Cincinnati, OH.

Melnick, R.L. (1984). NTP toxicological and carcinogenic studies of 1,3-butadiene. 76th Meeting of the Interagency Collaborative Group on Environmental Carcinogenesis. Bethesda, MD.

Melnick, R.L. (1985). Toxicity and carcinogenicity of 1,3-butadiene. National Institute of Hygienic Sciences. Tokyo, Japan.

Melnick, R.L., Morrissey, R.E., and Tomaszewski, K.E. (1986). National Toxicology Program studies on di(2-ethylhexyl)phthalate (DEHP). CMA Symposium on Recent Advances in Phthalate Esters Research. Washington, DC.

Melnick, R., Roycroft, J., Chou, B., and Miller, R. (1988). Inhalation toxicology and carcinogenicity of 1,3-butadiene in B6C3F1 mice. International Symposium on the Toxicology, Carcinogenesis, and Human Health Aspects of 1,3-Butadiene. Research Triangle Park, NC.

Melnick, R., Roycroft, J., Chou, B., Ragan, H., and Miller, R. (1988). Inhalation toxicology of isoprene in F344 rats and B6C3F1 mice. International Symposium on the Toxicology, Carcinogenesis, and Human Health Aspects of 1,3-Butadiene. Research Triangle Park, NC.

Toxicology and carcinogenicity of 1,3-butadiene. International Life Sciences Institute (ILSI) Symposium on Assessment of Inhalation Hazards: Integration and Extrapolation Using Diverse Data. Hannover, Federal Republic of Germany, 1989.

Recent studies on 1,3-butadiene and other high volume chemicals used in the rubber industry. United Rubber Workers Joint Labor/ Management Health and Safety Symposium. Daytona Beach, FL, 1989.

Overview on the toxicity and carcinogenicity of 1,3-butadiene in mice. Testimony for the OSHA Hearing on the Proposed Occupational Standard for 1,3-Butadiene. Department of Labor, Washington, DC, 1991.

Is chemically induced hepatocyte proliferation a predictor of liver carcinogenesis? International Life Sciences Institute (ILSI) Workshop on Mouse Liver Tumors. Washington, DC, 1992.

Alternative hypothesis on the role of alpha<sub>2</sub>-globulin in gasoline-caused kidney cancers. Collegium Ramazzini Symposium. Carpi, Italy, 1993.

Butadiene induced cancer in experimental animals. International Symposium on Health Hazards of Butadiene and Styrene. Espoo, Finland, 1993.

Carcinogenicity of 1,3-butadiene. International Conference on Motor Gasolines and Additives: Methyl-Tertiary Butyl Ether. Washington, DC, 1995.

Inhalation toxicity and carcinogenicity of isoprene in rats and mice: Comparisons with 1,3-butadiene. International Symposium: Evaluation of Butadiene & Isoprene Health Risks. Blaine, WA, 1995.

Role of the Office of Science and Technology Policy in federal risk assessment activities. US Department of Agriculture. Washington, DC, 1996.

CENR endocrine disruptor inventory: human health effects. Committee on Environment and Natural Resources (CENR) Meeting on Endocrine Disruptor Research. Washington, DC, 1996.

Carcinogenicity of trihalomethanes in female B6C3F<sub>1</sub> mice: Relationships among hepatotoxicity, regenerative hyperplasia and replicative DNA synthesis. Collegium Ramazzini Symposium. Carpi, Italy, 1997.

Possible mechanisms of induction of renal tubular cell neoplasms in rats associated with  $\alpha_2$ -globulin: role of protein accumulation versus ligand delivery to the kidney. IARC Meeting on "Mechanisms of Carcinogenesis thought to be Species-Specific." Lyon, France, 1997.

Endocrine disruptors. California's Emerging Environmental Challenges: A workshop to identify future issues for Cal/EPA. Sacramento, CA, 1998.

Dose-response analyses of experimental cancer data. Arkansas Toxicology Symposium Honoring David P. Rall. Little Rock, AR, 1998.

Perspective on chloroform cancer risk assessment. The Toxicology Forum. Washington, DC, 1999.

Chloroform cancer risk and dose-response relationships. University of Florida Symposium on Drinking Water and Health. Sarasota, Fla, 1999.

Melnick, R.L. Overview on the use of mechanistic data in risk assessment: Conference on Toxicology and Risk Assessment Approaches for the 21<sup>st</sup> Century. Kings Island, OH, 2000.

Melnick, R.L., Sills, R., Roycroft, J., Chou, and Miller, R.A. Comparative carcinogenicity of butadiene, isoprene, and chloroprene in rats and mice. International Symposium: Evaluation of Butadiene, Isoprene, and Chloroprene Health Risks. London, UK., 2000.

Role of the National Toxicology Program in drinking water research. EPA's Federal/State Toxicology and Risk Analysis Committee Biannual Meeting, Durham, NC, 2000.

Summary of the NTP/NIEHS endocrine disruptors low-dose peer review. International Symposium on Environmental Endocrine Disruptors 2000, Pacifico Yokohama, Japan, 2000.

NTP's Drinking Water Research Program. North Carolina Chapter of the Society of Toxicology, Chapel Hill, NC 2001.

Carcinogenicity of epoxides and epoxide-forming chemicals. New York Academy of Sciences Conference commemorating the Lifework of Cesare Maltoni. Chairman of Session On National Toxicology Program's Carcinogenesis Bioassays: Legacy of David P. Rall. New York, NY. 2002.

Studies on Drinking Water Disinfection Byproducts by The National Toxicology Program. ISEA/ISEE Conference, Vancouver, BC. 2002.

Carcinogenic responses in experimental animals after long-term inhalation exposures to dusts and particulates. Ramazzini International Conference on Carcinogenicity of Non-fibrous, Poorly Soluble Particulates, Carpi, Italy, 2002.

Endocrine disruption – what can work with laboratory animals tell us? 2<sup>nd</sup> Copenhagen Workshop on Endocrine Disruptors, Copenhagen, Denmark, 2002.

NTP research program on health effects of cell phone radio frequency radiation. The Toxicology Forum. Washington, DC, 2003.

Health effects of cell phone radiofrequency radiation: National Toxicology Program's carcinogenicity studies in rats and mice. Special symposium (Session Co-chair) of the Bioelectromagnetics Society 25<sup>th</sup> Annual Meeting. Maui, Hawaii, 2003.

Feasibility and design of rodent carcinogenicity studies on cell phone radio frequency radiation in reverberation chambers. Asia-Pacific EMF Conference on Electromagnetic Fields, Research, Health Effects, and Standard Harmonization. Bangkok, Thailand, 2004.

The hormesis thesis. Integrity in Science Conference sponsored by Center for Science in the Public Interest. Washington, DC, 2004.

Use and misuse of mechanistic data in risk assessment. Ramazzini International Conference: Framing the Future in Light of the Past: Living in a Chemical World. Bologna, Italy, 2005.

Induction of peroxisome proliferation by trichloroethylene and perchloroethylene: implications for risk assessment. Ramazzini International Conference: Framing the Future in Light of the Past: Living in a Chemical World. Bologna, Italy, 2005.

Determining disease causality from experimental toxicology studies. Science for Judges VII. Brooklyn Law School, Brooklyn, NY, 2006.

Experimental design and evaluation as sources of conflicting views in science. Project on Scientific Knowledge & Public Policy, Coronado Conference III: Truth and Advocacy: the Quality and Nature of Litigation and Regulatory Science. San Diego, CA, 2006.

Hormesis in public health decisions: Who benefits? EOHSI Days, Rutgers University, Piscataway, NJ, 2006.

National Toxicology Program's research on emerging and priority disinfection by-products. Gordon Research Conference on Drinking Water Disinfection By-products. Mount Holyoke College, South Hadley, MA, 2006.

Judicial Gatekeeping: Commentary by Scientists. Judicial Symposium on Scientific Evidence in the Courts. AEI-Brookings Joint Center for Regulatory Studies. Georgetown University Law Center, Washington, DC, 2007.

In vitro studies of PFAAs (perfluoroalkyl acids). PFAA Days Workshop, US EPA, Research Triangle Park, NC, 2008

Risk evaluations and governance. Health Risk from Exposure to Wireless Network Devices. EMF & Health Risk Research Workshop, Ascona, Switzerland, 2012.

A Framework for Considering the CYP2F2 MOA Hypothesis & Relevance of Mouse Lung Tumors to Humans. Co-chair of Session 3: Biological Mechanisms. US EPA Mouse Lung Tumor Workshop, Research Triangle Park, NC, 2014.

Radiofrequency radiation: A possible human carcinogen? Co-chair of Basic Science working group. IIAS/EHT Conference on Wireless Radiation and Health: Expert Forum on Environmental Health Research and Policy Priorities. Hebrew University, Jerusalem, Israel, 2017.

## Publications

### *Scientific Journals*

1. Melnick, R.L. and Hultin, H.O. Solubilization of bound lactate dehydrogenase by NADH in homogenates of trout skeletal muscle as a function of tissue concentration. *Biochem. Biophys. Res. Commun.* 33: 863-868, 1968.
2. Melnick, R.L. and Hultin, H.O. Factors affecting the distribution of lactate dehydrogenase between particulate and soluble phases of homogenized trout skeletal muscle. *J. Food Sci.* 35: 67-72, 1970.
3. Melnick, R.L. and Packer, L. Freeze fracture faces of inner and outer membranes of mitochondria. *Biochim. Biophys. Acta* 253: 503-508, 1971.
4. Hultin, H.O., Ehman, J.D., and Melnick, R.L. Modification of kinetic properties of muscle lactate dehydrogenase by subcellular associations and possible role in the control of glycolysis. *J. Food Sci.* 37: 269-273, 1972.
5. Melnick, R.L. and Hultin, H.O. Studies on the nature of the subcellular localization of lactate dehydrogenase and glyceraldehyde-3-phosphate dehydrogenase in chicken skeletal muscle. *J. Cell. Physiol.* 81: 139-148, 1973.
6. Melnick, R.L., Tinberg, H.M., Maguire, J., and Packer, L. Studies on mitochondrial proteins. I. Separation and characterization by polyacrylamide gel electrophoresis. *Biochim. Biophys. Acta* 311: 230-241, 1973.
7. Melnick, R.L. and Hultin, H.O. On the existence of a complex of glycolytic enzymes. *J. Bioenergetics* 5: 107-117, 1974.
8. Tinberg, H.M., Melnick, R.L., Maguire, J., and Packer, L. Studies on mitochondrial proteins. II. Localization of components in the inner membrane. Labeling with diazobenzenesulfonate, a non-penetrating probe. *Biochim. Biophys. Acta* 345: 118-128, 1974.
9. Tinberg, H.M., Melnick, R.L., Maguire, J., and Packer, L. Interaction of mitochondrial inner membranes with bifunctional alkylating agents. *BBA Library* 13: 539-541, 1974.
10. Melnick, R.L., Tavares de Sousa, J., Maguire, J., and Packer, L. Action of the adenosine triphosphate analog, adenylyl imidodiphosphate, in mitochondria. *Arch. Biochem. Biophys.* 166: 139-144, 1975.
11. Melnick, R.L. and Donohue, T. Use of an adenosine triphosphate analog, adenylyl imidodiphosphate, to evaluate adenosine triphosphate dependent reactions in mitochondria. *Arch. Biochem. Biophys.* 173: 231-236, 1976.

12. Melnick, R.L., Monti, L.G., and Motzkin, S.M. Uncoupling of mitochondrial oxidative phosphorylation by thallium. *Biochem. Biophys. Res. Commun.* 69: 68-73, 1976.
13. Melnick, R.L., Hanson, R.M., and Morris, H.P. Membranous effects on adenosine triphosphatase activities of mitochondria from rat liver and Morris Hepatoma 3924A. *Cancer Res.* 37: 4395-4399, 1977.
14. Melnick, R.L., Rubenstein, C.P., and Motzkin, S.M. Measurement of mitochondrial oxidative phosphorylation: Selective inhibition of adenylate kinase activity by P<sub>1</sub>,P<sub>5</sub>-(adenosine-5')-pentaphosphate. *Anal. Biochem.* 96: 7-11, 1979.
15. Melnick, R.L., Haspel, H.C., Goldenberg, M., Greenbaum, L.M., and Weinstein, S. Use of fluorescent probes that form intramolecular excimers to monitor structural changes in model and biological membranes. *Biophys. J.* 34: 499-515, 1981.
16. Melnick, R.L., Rubenstein, C.P., and Birenbaum, L. Effects of millimeter wave irradiation on ATP synthesis and calcium transport in mitochondria. *Radiat. Res.* 89: 348-360, 1981.
17. Melnick, R.L. and Schiller, C.M. Mitochondrial toxicity of phthalate esters. *Environ. Health Perspect.* 45: 51-56, 1982.
18. Melnick, R.L., Huff, J., Haseman, J.K., Dieter, M.P., Grieshaber, C.K., Wyand, D.S., Russfield, A.B., Murthy, A.S.K., Fleischman, R.M. and Lilja, H.S. Chronic effects of agar, guar gum, gum arabic, locust bean gum, or tara gum in F344 rats and B6C3F1 mice. *Food Chem. Toxicol.* 21: 305-311, 1983.
19. Melnick, R.L., Boorman, G.A., Haseman, J.K., Montali, R.J., and Huff, J. Urolithiasis and bladder carcinogenicity of melamine in rodents. *Toxicol. Appl. Pharmacol.* 72: 292-303, 1984.
20. Melnick, R.L. Toxicities of ethylene glycol and ethylene glycol monoethyl ether in Fischer 344/N rats and B6C3F1 mice. *Environ. Health Perspect.* 57: 147-155, 1984.
21. Melnick, R.L. and Schiller, C.M. Effect of phthalate esters on energy coupling and succinate oxidation in rat liver mitochondria. *Toxicology* 34: 13-27, 1985.
22. Huff, J.E., Melnick, R.L., Solleveld, H.A., Haseman, J.K., Powers, M. and Miller, R.A. Multiple organ carcinogenicity of 1,3-butadiene in B6C3F1 mice after 60 weeks of inhalation exposure. *Science* 227: 548-549, 1985.
23. Tomaszewski, K.E., Agarwal, D.K., and Melnick, R.L. In vitro steady-state levels of hydrogen peroxide after exposure of male F344 rats and female B6C3F1 mice to hepatic peroxisome proliferators. *Carcinogenesis* 7: 1871-1876, 1986.

24. Melnick, R.L., Jameson, C.W., Goehl, T.J., and Kuhn, G.O. Application of microencapsulation for toxicology studies. 1. Principles and stabilization of trichloroethylene in gelatin-sorbitol microcapsules. *Fundam. Appl. Toxicol.* 8: 425-431, 1987.
25. Melnick, R.L., Jameson, C.W., Goehl, T.J., Maronpot, R.R., Collins, B.J., Greenwell, A., Harrington, F.W., Wilson, R.E., Tomaszewski, K.E., and Agarwal, D.K. Application of microencapsulation for toxicology studies. 2. Toxicity of microencapsulated trichloroethylene in Fischer 344 rats. *Fundam. Appl. Toxicol.* 8: 432-442, 1987.
26. Melnick, R.L., Morrissey, R.E., and Tomaszewski, K.E. Studies by the National Toxicology Program on di(2-ethylhexyl)phthalate. *Toxicol. Indus. Health* 3: 99-116, 1987.
27. Tomaszewski, K.E., Derks, M.C., and Melnick, R.L. Acyl CoA oxidase is the most suitable marker for hepatic peroxisomal changes caused by treatment of F344 rats with di(2-ethylhexyl) phthalate. *Toxicol. Lett.* 37: 203-212, 1987.
28. Greenwell, A., Tomaszewski, K.E., and Melnick, R.L. A biochemical basis for 1,2-dibromo-3-chloropropane - induced male infertility: Inhibition of sperm mitochondrial electron transport activity. *Toxicol. Appl. Pharmacol.* 91: 274-280, 1987.
29. Melnick, R.L., Huff, J.E., Haseman, J.K., and McConnell, E.E. Chronic toxicity results and ongoing studies of 1,3-butadiene by the National Toxicology Program. *Ann. NY Acad. Sci.* 534: 648-662, 1988.
30. Tomaszewski, K.E., Montgomery, C., and Melnick, R.L. Modulation of 2,3,7,8-tetrachlorodibenzo-p-dioxin toxicity in F344 rats by di(2-ethylhexyl)phthalate. *Chem. Biol. Interactions* 65: 205-222, 1988.
31. Tice, R.R., Boucher, R., Luke, C.A., Paquette, D.E., Melnick, R.L., and Shelby, M.D. Chloroprene and isoprene: Cytogenetic studies in mice. *Mutagenesis* 3: 141-146, 1988.
32. Bond, J.A., Martin, O.S., Birnbaum, L.S., Dahl, A.R., Melnick, R.L., and Henderson, R.F. Metabolism of 1,3-butadiene by lung and liver microsomes of rats and mice repeatedly exposed by inhalation to 1,3-butadiene. *Toxicol. Lett.* 44: 143-151, 1988.
33. Kralovanszky, J., Jenkins, W.L., Greenwell, A., and Melnick, R.L. Metabolic processes in isolated rat small intestine villus cells: Effects of cis-diamminedichloro-platinum (II). *Res. Commun. Chem. Pathol. Pharmacol.* 64: 299-316, 1989.
34. Miller, R.A., Melnick, R.L., and Boorman, G.A. Neoplastic lesions induced by 1,3-butadiene in B6C3F1 mice. *Exptl. Pathol.* 37: 136-146, 1989.
35. Melnick, R.L., Huff, J.E., Bird, M.G., and Acquavella, J.F. Symposium overview: Toxicology, carcinogenesis, and human health aspects of 1,3-butadiene. *Environ. Health Perspect.* 86: 3-5, 1990.

36. Melnick, R.L., Huff, J.E., Roycroft, J.H., Chou, B.J., and Miller, R.A. Inhalation toxicology and carcinogenicity of 1,3-butadiene in B6C3F1 mice following 65 weeks exposure. *Environ. Health Perspect.* 86: 27-36, 1990.
37. Melnick, R.L., Roycroft, J.H., Chou, B.J., Ragan, H.A., and Miller, R.A. Inhalation toxicology of isoprene in F344 rats and B6C3F1 mice following 2-week exposures. *Environ. Health Perspect.* 86: 93-98, 1990.
38. Tyson, C.A., Dabbs, J.E., Cohen, P.M., Green, C.E., and Melnick, R.L. Studies of nephrotoxic agents in an improved renal proximal tubule system. *Toxic. In Vitro* 4: 403-408, 1990.
39. Kralovanszky, J., Harrington, F., W.L., Greenwell, A., and Melnick, R.L. Isolation of viable intestinal epithelial cells and their use for in vitro toxicity studies. *In Vivo* 4: 201-204, 1990.
40. Melnick, R.L., Huff, J., Chou, B.J., and Miller, R.A. Carcinogenicity of 1,3-butadiene in C57BL/6 x C3H F1 mice at low exposure concentrations. *Cancer Res.* 50: 6592-6599, 1990.
41. Tomaszewski, K.E., Heindel, S.W., Jenkins, W.L., and Melnick, R.L. Induction of peroxisomal acyl CoA oxidase activity and lipid peroxidation in primary rat hepatocyte cultures. *Toxicology* 65: 49-60, 1990.
42. Melnick, R.L. and Huff, J. 1,3-Butadiene: Toxicity and carcinogenicity in laboratory animals and in humans. *Rev. Environ. Contam. Toxicol.* 124: 111-144, 1992.
43. Melnick, R.L. Does chemically induced hepatocyte proliferation predict liver carcinogenesis? *FASEB J.* 6: 2698-2706, 1992.
44. Melnick, R.L. An alternative hypothesis on the role of chemically induced protein droplet ( $\alpha$ 2u-globulin) nephropathy in renal carcinogenesis. *Regulatory Toxicol. Pharmacol.* 16: 111-125, 1992.
45. Melnick, R.L. Mechanistic data in scientific public health decisions. *Regulatory Toxicol. Pharmacol.* 16: 109-110, 1992. Reprinted by Collegium Ramazzini in *Ramazzini Newsletter* 3.1992: 45-46 (1992).
46. Melnick, R.L., Huff, J., and Matanoski, G.M. Carcinogenicity of 1,3-butadiene. *The Lancet* 340: 724-725, 1992.
47. Melnick, R.L. and Huff, J. Chemicals and human cancer. *The Lancet* 340: 1409, 1992.
48. Huff, J. and Melnick, R. Identifying carcinogens. *Issues Sci. Technol.* 9: 14-15, 1993.

49. Kohn, M.C. and Melnick, R.L. Species differences in the production and clearance of butadiene metabolites: A mechanistic model indicates predominantly physiological, not biochemical, control. *Carcinogenesis* 14: 619-628, 1993.
50. Dunnick, J.K. and Melnick, R.L. Assessment of the carcinogenic potential of chlorinated water: Experimental studies of chlorine, chloramine, and trihalomethanes. *J. Natl. Cancer Inst.* 85: 817-822, 1993.
51. Melnick, R.L. and Huff, J.E. Liver carcinogenesis is not a predicted outcome of chemically induced hepatocyte proliferation. *Toxicol. Indus. Health* 9: 415-438, 1993.
52. Melnick, R.L., Shackelford, C.C., and Huff, J. Carcinogenicity of 1,3-butadiene. *Environ. Health Perspect.* 100: 227-236, 1993.
53. Melnick, R.L., Huff, J., Barrett, J.C., Maronpot, R.R., Lucier, G., and Portier, C.J. Cell proliferation and chemical carcinogenesis: Symposium overview. *Mol. Carcinogen.* 7: 135-138 and in *Environ. Health Perspect.* 101 (Suppl. 5): 3-8, 1993.
54. Bucher, J.R., Melnick, R.L., and Hildebrandt, P.K. Lack of carcinogenicity in mice exposed once to high concentrations of 1,3-butadiene. *J. Natl. Cancer Inst.* 85: 1866-1867, 1993.
55. Melnick, R.L. Critique does not validate assumptions in the model on  $\alpha$ 2u-globulin and renal carcinogenesis. *Regulatory Toxicol. Pharmacol.* 18: 365-368, 1993.
56. Melnick, R.L., Mahler, J., Bucher, J.R., Thompson, M., Hejtmancik, M., Ryan, M.J., and Mezza, L.E. Toxicity of diethanolamine. 1. Topical application and drinking water exposures in F344 rats. *J. Appl. Toxicol.* 14: 1-9, 1994.
57. Melnick, R.L., Mahler, J., Bucher, J.R., Hejtmancik, M., Singer, A., and Persing, R.L. Toxicity of diethanolamine. 2. Topical application and drinking water exposures in B6C3F1 mice. *J. Appl. Toxicol.* 14: 11-19, 1994.
58. Tomaszewski, K.E. and Melnick, R.L. Involvement of CoA thioesters in peroxisome proliferation and hypolipidemia. *Biochim. Biophys. Acta* 1120: 118-124, 1994.
59. Melnick, R.L., Dunnick, J.K., Sandler, D.P., Elwell, M.R., and Barrett, J.C. Trihalomethanes and other environmental factors that contribute to colorectal cancer. *Environ. Health Perspect.* 102: 586-588, 1994.
60. Melnick, R.L., Sills, R.C., Roycroft, J.H., Chou, B.J., Ragan, H.A., and Miller, R.A. Isoprene, an endogenous hydrocarbon and industrial chemical, induces multiple organ neoplasia in rodents after 26 weeks of inhalation exposure. *Cancer Res.* 54: 5333-5339, 1994.
61. Melnick, R.L. and Kohn, M.C. Mechanistic data indicate that 1,3-butadiene is a human carcinogen. *Carcinogenesis* 16: 157-163, 1995.

62. Melnick, R.L., Elwell, M.R., Roycroft, J.H., Chou, B.J., Ragan, H.A., and Miller, R.A. Toxicity of inhaled chloroprene (2-chloro-1,3-butadiene) in F344 rats and B6C3F<sub>1</sub> mice. *Toxicology* 108: 79-91, 1996.
63. Melnick, R.L., Kohn, M.C., and Portier, C.J. Implications for risk assessment of suggested nongenotoxic mechanisms of chemical carcinogenesis: *Environ. Health Perspect.* 104 (Suppl. 1): 123-134, 1996.
64. Melnick, R.L., Sills, R.C., Roycroft, J.H., Chou, B.J., Ragan, H.A., and Miller, R.A. Inhalation toxicity and carcinogenicity of isoprene in rats and mice: comparisons with 1,3-butadiene. *Toxicology* 113: 247-252, 1996.
65. Kohn, M.C. and Melnick, R.L. Effects of the structure of a toxicokinetic model of butadiene inhalation exposure on computed production of carcinogenic intermediates. *Toxicology* 113: 31-39, 1996.
66. Hong, H.L., Devereux, T.R., Melnick, R.L., Eldridge, S.R., Greenwell, A., Haseman, J., Boorman, G.A., and Sills, R.C. Both K-ras and H-ras protooncogene mutations are associated with harderian gland tumorigenesis in B6C3F<sub>1</sub> mice exposed to isoprene for 26 weeks. *Carcinogenesis* 18: 783-789, 1997.
67. Buchanan, J.R., Burka, L.T., and Melnick, R.L. Purpose and guidelines for toxicokinetic studies within the National Toxicology Program. *Environ. Health Perspect.* 105: 468-471, 1997.
68. Melnick, R.L., Boorman, G.A., and Dellarco, V. Water chlorination, 3-Chloro-4-(dichloromethyl)-5-hydroxy-2(5H)-furanone (MX), and potential cancer risk. *J. Natl. Cancer Inst.* 89: 832-833, 1997.
69. Melnick, R.L., Kohn, M.C., and Huff, J. Weight-of-evidence versus weight-of-speculation to evaluate the  $\alpha$ 2u-globulin hypothesis. *Environ. Health Perspect.* 105: 904-906, 1997.
70. Melnick, R.L., Kohn, M.C., Dunnick, J., and Leininger, J.R. Regenerative hyperplasia is not required for liver tumor induction in female B6C3F<sub>1</sub> mice exposed to trihalomethanes. *Toxicol. Appl. Pharmacol* 148: 137-147, 1998.
71. Reiter, L.W., DeRosa, C., Kavlock, R.J., Lucier, G., Mac, M.J., Melillo, J., Melnick, R.L., Sinks, T., and Walton, B.T. The US federal framework for research on endocrine disruptors and an analysis of the supported research programs supported during fiscal year 1996. *Environ. Health Perspect.* 106: 105-113, 1998
72. Melnick, R.L. and Kohn, M.C. Response to letter from ILSI panel on liver tumor induction by trihalomethanes. *Toxicol. Appl. Pharmacol* 153: 135-136, 1998.

73. Melnick, R.L., Kohn, M.C., Dunnick, J., and Leininger, J.R. Cell proliferation is not predictive of liver tumor induction by trihalomethanes in mice. *Eur. J. Oncology* 3: 413-417, 1998.
74. Melnick, R.L. and Lucier, G. Endocrine disruptors. In: *Proceedings of the Workshop on California's Emerging Environmental Challenges*, California Environmental Protection Agency, Sacramento, CA, pp. 4.11-4.17. 1999.
75. Melnick, R.L. Introduction to the workshop on characterizing the effects of endocrine disruptors on human health at environmental exposure levels. *Environ. Health Perspect.* 107 (Suppl. 4): 603-604 1999.
76. Melnick, R.L., Sills, R.C., Portier, C.J., Roycroft, J.H., Chou, B.J., Grumbein, S.L., and Miller, R.A. Multiple organ carcinogenicity of inhaled chloroprene (2-chloro-1,3-butadiene) in F344/N rats and B6C3F<sub>1</sub> mice and comparison of dose-response with 1,3-butadiene in mice. *Carcinogenesis* 20: 867-878, 1999.
77. Sills, R.C., Hong, H.L., Melnick, R.L., Boorman, G.A., and Devereux, T.R. High frequency of codon 61 *K-ras* A→T transversions in lung and Harderian gland neoplasms of B6C3F<sub>1</sub> mice exposed to chloroprene (2-chloro-1,3-butadiene) for 2 years and comparisons with the structurally related chemicals isoprene and 1,3-butadiene. *Carcinogenesis* 20: 657-662, 1999.
78. Kohn, M.C. and Melnick, R.L. A physiological model for ligand-induced accumulation of  $\alpha$ 2u-globulin in male rat kidney: Roles of protein synthesis and lysosomal degradation in the renal dosimetry of 2,4,4-trimethyl-2-pentanol. *Toxicology* 136: 89-105, 1999.
79. Sills, R.C., Hailey, J.R., Neal, J., Boorman, G.A., Haseman, J.K., and Melnick, R.L. Examination of low-incidence brain tumor responses in F344 rats following chemical exposures in National Toxicology Program carcinogenicity studies. *Toxicol. Pathol.* 27: 589-599, 1999.
80. Melnick, R.L. and Kohn, M.C. Dose-response analyses of experimental cancer data. *Drug Metab. Rev.* 32: 193-209, 2000.
81. Kohn, M.C. and Melnick, R.L. The privileged access model of 1,3-butadiene disposition. *Environ. Health Perspect.* 108 (Suppl. 5): 911-917, 2000.
82. Hong, H-H.L., Devereux, T.R., Melnick, R.L., Moomaw, C. R., Boorman, G.A., and Sills, R.C. Mutations of *ras* protooncogenes and *p53* tumor suppressor gene in cardiac hemangiosarcomas from B6C3F<sub>1</sub> mice exposed to 1,3-butadiene for 2-years. *Toxicol. Pathol.* 28: 529-534, 2000.
83. Tomatis, L., Melnick, R.L., Haseman, J., Barrett, J.C., and Huff, J. Alleged "misconceptions" distort perceptions of environmental cancer risks. *FASEB J.* 15: 195-203, 2001.

84. Melnick, R.L. Is peroxisome proliferation an obligatory precursor step in the carcinogenicity of di(2-ethylhexyl)phthalate (DEHP)? *Environ. Health Perspect.* 109: 437-442, 2001.
85. Haseman, J., Melnick R., Tomatis, L., and Huff, J. Carcinogenesis bioassays: study duration and biological relevance. *Food Chem. Toxicol* 39: 739-744, 2001.
86. Melnick, R.L. and Sills, R.C. Comparative carcinogenicity of 1,3-butadiene, isoprene, and chloroprene in rats and mice. *Chem. Biol. Interact.* 135: 27-42, 2001.
87. Kohn, M.C. and Melnick, R.L. Physiological modeling of butadiene disposition in mice and rats. *Chem. Biol. Interact.* 135: 285-301, 2001.
88. Sills, R.C., Hong, H.L., Boorman, G.A., Devereux, T.R., and Melnick, R.L. Point mutations of K-ras and H-ras genes in forestomach neoplasms from control B6C3F1 mice and following exposure to 1,3-butadiene, isoprene or chloroprene for up to 2-years. *Chem. Biol. Interact.* 135: 373-386, 2001.
89. Melnick, R. Peroxisome proliferators: response. *Environ. Health Perspect.* 109: A463-A464, 2001.
90. Willems, B.A.T., Melnick, R.L., Kohn, M.C., and Portier, C.J. A physiologically based pharmacokinetic model for inhalation of naphthalene in rats and mice. *Toxicol. Appl Pharmacol.* 176: 81-91, 2001.
91. Melnick, R., Lucier, G., Wolfe, M., Hall, R., Stancel, G., Prins, G., Gallo, M., Reuhl, K., Ho, S.M., Brown, T., Moore, J., Leakey, J., Haseman, J., and Kohn, M. Summary of the National Toxicology Program's report of the endocrine disruptors low-dose peer review. *Environ. Health Perspect.* 110: 427-431, 2002.
92. Kohn, M.C., Melnick, R.L., Ye, F., Portier, C. J. Pharmacokinetics of sodium nitrite-induced methemoglobinemia in the rat. *Drug Metab. Dispos.* 30; 676-683, 2002..
93. Kohn, M.C. and Melnick, R.L. Biochemical origins of non-monotonic receptor-mediated dose-response. *J. Molec. Endocrin.* 29; 113-123, 2002.
94. Melnick R.L. The IARC evaluation of di(2-ethylhexyl)phthalate (DEHP): A flawed decision based on an untested hypothesis. *Int. J. Occup. Environ Health* 8; 284-286, 2002.
95. Melnick R.L. Carcinogenicity and mechanistic insights on the behavior of epoxides and epoxide-forming chemicals. *Ann. NY Acad. Sci.* 982; 177-189, 2002.
96. Huff, J., Castleman, B., LaDou, J., Epstein, S.S., Frank, F.L., Greenberg, M., Hooper, K., Infante, P., Melnick, R., Sass, J.B., Teitelbaum, D., Tomatis, L. Primary prevention of cancer. *The Scientist* 16; 10-11, 2002.

97. Toraason, M., Anderson, M., Bogdanffy, M.S., Dankovic, D., Faustman, E., Foster, P., Frederick, C., Haber, L., Kimmel, C.A., Lewis, S., McClellan, R., Melnick, R., Mirer, F., Morgan, K., Schaeffer, V., Silbergeld, E., Slikker, W., Swenberg, J., and Vainio, H. Improving risk assessment: toxicological research needs. *Hum. Ecol. Risk Assess.* 8; 1405-1419, 2002.
98. Axelson, O., Castleman, B., Epstein, S., Franco, G., Giannasi, F., Grandjean, P., Greenberg, M., Hooper, K., Huff, J., Jacobson, M., Joshi, T.K., Kulkarni, G.K., LaDou, J., Mazaheri, M., Mekonnen, Y., Melnick, R., Mirabelli, D., Ofrin, R., Partanen, T., Pott, F., Sass, J., Soskolne, C.L., Suplido, M.L., Terracini, B., Tomatis, L., Ungvary, G., Watterson, A., Wesseling, C., and Yassi, A. Re: Implementation of WHO Guidelines on Disclosure of Interest by members of WHO Expert Panels. *Int. J. Occup. Environ. Health* 8: 271-273, 2002.
99. Ward, E.M., Schulte, P.A., Bayard, S., Blair, A., Brandt-Rauf, P., Butler, M.A., Dankovic, D., Hubbs, A.F., Jones, C., Karstadt, M., Kedderis, G.L., Melnick, R., Redlich, C., Rothman, N., Savage, R.E., Sprinker, M., Toraason, and Weston, A. Priorities for development of research methods in occupational cancer. *Environ. Health Perspect.* 111; 1-12, 2003.
100. Melnick, R.L., Kamel, F., and Huff, J. Declaring chemicals “not carcinogenic to humans” requires validation not speculation. *Environ. Health Perspect.* 111; A203-A204, 2003.
101. Melnick, R.L. Suppression of crucial information in the IARC evaluation DEHP. *Int. J. Occup. Environ. Health* 9: 84-85, 2003.
102. Melnick, R.L., Brody, C., DiGangi, J., and Huff J. The IARC evaluation of DEHP excludes key papers demonstrating carcinogenic effects. *Intl. J. Occup. Environ. Health* 9: 400-402, 2003.
103. Melnick, R.L., Bucher J.R., Roycroft, J.H., Hailey, J.R., and Huff, J. Carcinogenic and toxic effects on inhaled non-fibrous poorly soluble particulates in rats and mice contradict threshold lung cancer hypotheses that are dependent on chronic pulmonary inflammation. *European J. Oncology* 8; 177-186, 2003.
104. Huff, J., Melnick, R., Tomatis, L., LaDou, J., and Teitelbaum, D. Trichloroethylene and cancers in humans. *Toxicology* 197: 185-187, 2004.
105. Schechter, A., Lucier, G.W., Cunningham, M.L., Abdo, K.M., Blumenthal, G., Silver, A.G., Melnick, R., Portier, C., Barr, D.B., Barr, J.R., Stanfill, S.B., Patterson, D.G., Needham, L.L., Stopford, W., Masten, S., Mignogna, J., and Tung, K.C. Human consumption of methyleugenol and its elimination from serum. *Environ. Health Perspect.* 112; 678-680, 2004.
106. Melnick R.L. and Huff, J. Testing toxic pesticides in humans: Health risks with no health benefits. *Environ. Health Perspect.* 112; A459-461, 2004.
107. Melnick, R.L. A Daubert motion: A legal strategy to exclude essential scientific evidence in toxic tort litigation. *Am. J. Public Health* 95; S30-S34, 2005.

108. Kim, Y., Hong, H-H.L., Lachat, Y., Clayton, N.P., Devereux, T.R., Melnick, R.L., Hegi, M.E., and Sills, R.C. Genetic alterations in brain tumors following 1,3-butadiene exposure in B6C3F1 mice. *Toxicol. Pathol.* 33; 307-312, 2005.
109. Thayer, K.A., Melnick, R., Burns, K., Davis, D., and Huff, J. Fundamental flaws of hormesis for public health decisions. *Environ. Health Perspect.* 113; 1271-1276, 2005.
110. Ton, T.V., Hong, H.H., Devereux, T.R., Melnick, R.L., Sills, R.C., and Kim, Y. Evaluation of genetic alterations in cancer-related genes in lung and brain tumors from B6C3F1 mice exposed to 1,3-butadiene or chloroprene. *Chem. Biol. Interact.* 166; 112-120, 2007.
111. Melnick R. and Bucher, J. Determining disease causality from experimental toxicology studies. *J. Law and Policy* 107; 113-133, 2005.
112. Melnick, R.L., Nyska, A., Foster, P.M., Roycroft, J.H., and Kissling, G.E. Toxicity and carcinogenicity of the water disinfection byproduct, dibromoacetic acid, in rats and mice. *Toxicology* 230; 126-136, 2007.
113. Melnick, R., Thayer, K., and Bucher J. Conflicting views on chemical carcinogenesis arising from the design and evaluation of rodent carcinogenicity studies. *Environ. Health Perspect.* 116; 130-135, 2008.
114. Vineis, P. and Melnick R. A Darwinian perspective: right questions, questionable conclusions: Commentary on Niall Shanks and Rebecca Pyles' evolution and medicine: the long reach of "Dr. Darwin". *Philos. Ethics Humanit Med* Feb 12; 3-6, 2008.
115. Slotkin, T.A., MacKillop, E.A., Melnick, R.L., Thayer, K.A., and Seidler, F.J. Developmental neurotoxicity of perfluorinated chemicals modeled in vitro. *Environ. Health Perspect.* 116; 716-722, 2008.
116. Stout, M.D., Herbert, R.A., Kissling, G.E., Collins, B.J., Travlos, G.S., Witt, K.L., Melnick, R.L., Malarkey, D.E., and Hooth, M.J. Hexavalent chromium is carcinogenic to F344/N rats and B6C3F1 mice after chronic oral exposure. *Environ. Health Perspect.* 117; 716-722, 2009.
117. Baan, R., Grosse, Y., Straif, K., et al. (including WHO International Agency for Research on Cancer Monograph Working Group). A review of human carcinogens – Part F: chemical agents and related occupations. *Lancet Oncol* 10: 1143-1144, 2009.
118. Matthews' J.L., Schultz I.R., Easterling, M.R., and Melnick' R.L. Physiologically based pharmacokinetic modeling of dibromoacetic acid in F344 rats. *Toxicol. Appl. Pharmacol.* 244; 196-207, 2010.

119. Collins, B.J., Stout, M.D., Levine, K.E., Kissling, G.E., Melnick, R.L., Fennell, T.R., Walden, R., Pritchard, J.B., Fernando, R.A., Burka, L.T., and Hooth, M.J. Exposure to hexavalent chromium resulted in significantly higher tissue chromium burden compared with trivalent chromium following similar oral doses to male F344/N rats and female B6C3F1 mice. *Toxicol. Sci.* 118; 368-379, 2010.
120. Huff, J., Chan P.C. and Melnick, R. Clarifying carcinogenicity of ethylbenzene. *Regulatory Toxicol. Pharmacol.* 58:167-169, 2010.
121. Melnick, R.L. and Huff, J. Lorenzo Tomatis and primary prevention of environmental cancer. *Environ. Health* 10 (Suppl 1), S14, 2011.
122. Grosse, Y., Baan, R., et al. (including WHO International Agency for Research on Cancer Monograph Working Group). Carcinogenicity of chemicals in industrial and consumer products, food contaminants and flavourings, and water chlorinations byproducts. *Lancet Oncol.* 12: 328-329, 2011.
123. Baan, R., Grosse, Y., et al., (including WHO International Agency for Research on Cancer Monograph Working Group). Carcinogenicity of radiofrequency electromagnetic fields. *Lancet Oncol.* 12: 624-626, 2011.
124. Huff, J. and Melnick, R. Environmental justice and primary prevention of cancer: The Odyssey and legacy of Lorenzo Tomatis. *New Solut.* 22:7-17, 2012.
125. Burns, K.M. and Melnick, R.L. MTBE: Recent carcinogenicity studies. *Int. J. Occup. Environ. Health* 18:66-68, 2012
126. Melnick, R.L., Burns, K.M., Ward, J.M., Huff, J. Chemically exacerbated chronic progressive nephropathy not associated with renal tubule tumor induction in rats: An evaluation based on 60 carcinogenicity studies by the National Toxicology Program. *Toxicol. Sci.* 128:346-356, 2012.
127. Wallace, K.B., Kissling, G.E., Melnick, R.L., and Blystone, C.R. Structure-activity relationships for perfluoroalkane-induced *in vitro* interference with rat liver mitochondrial respiration. *Toxicol. Lett.* 222:257-264, 2013.
128. Melnick, R.L., Ward, J.M., and Huff, J. War on carcinogens: Industry disputes human relevance of chemicals causing cancer in laboratory animals based on unproven hypothesis, using kidney tumors as an example. *Int. J. Occup. Environ. Health* 19:255-260, 2013.
129. Capstick, M., Kuster, N., Kuhn, S. Berdinas-Torres, V., Gong, Y., Wilson, P., Ladbury, J., Koepke, G., McCormick, D., Gauger, J., and Melnick R. A radio frequency radiation reverberation chamber exposure system for rodents. *IEEE Trans Electromagn Compat.* 59:1041-1052, 2017.

130. Gong, Y., Capstick, M., McCormick, D.L., Gauger, J.R. Horn, T., Wilson, R., Melnick, R.L., and Kuster, N. Life time dosimetric assessment for mice and rats exposed to cell phone radiation. *IEEE Trans Electromagn Compat.* DOI: [10.1109/TEMC.2017.2665039](https://doi.org/10.1109/TEMC.2017.2665039), 2017

### *Book Chapters*

1. Melnick, R.L., Huff, J.E., and Miller, R.A. Toxicology and carcinogenicity of 1,3-butadiene. In "Assessment of Inhalation Hazards". Mohr, U., Bates, D.V., Dungworth, D.L., Lee, P.N., McCellan, R.O., and Roe, F.J.C. (eds). Springer-Verlag, New York, pp. 177-188, 1989.
2. Melnick, R.L. and Tomaszewski, K.E. Ethanolamine. In Ethel Browning's Toxicity and Metabolism of Industrial Solvents, 2nd edition, vol II: Nitrogen and Phosphorus Solvents. Ed., D.R. Buhler and D.J. Reed. Elsevier Biomedical Press, Amsterdam, Netherlands. pp. 423-430, 1990.
3. Melnick, R.L. and Tomaszewski, K.E. Diethanolamine. In Ethel Browning's Toxicity and Metabolism of Industrial Solvents, 2nd edition, vol II: Nitrogen and Phosphorus Solvents. Ed., D.R. Buhler and D.J. Reed. Elsevier Biomedical Press, Amsterdam, Netherlands. pp. 401-410, 1990.
4. Melnick, R.L. and Tomaszewski, K.E. Triethanolamine. In Ethel Browning's Toxicity and Metabolism of Industrial Solvents, 2nd edition, vol II: Nitrogen and Phosphorus Solvents. Ed., D.R. Buhler and D.J. Reed. Elsevier Biomedical Press, Amsterdam, Netherlands. pp. 441-450, 1990.
5. Melnick, R.L. and Huff, J. 1,3-Butadiene-induces cancer in experimental animals at all exposure concentrations ranging from 6.25 to 8000 parts per million. In "Butadiene and Styrene: Assessment of Health Hazards". Sorsa, M., Peltonen, K., Vainio, H., and Hemminiki, K. (eds). IARC Sci. Publ. No. 127. International Agency for Research on Cancer, Lyon, France. pp 309-322, 1993.
6. Melnick, R.L., White, M.C., Davis, J.M., Hartle, R.W., Ghanayem, B., Ashley, D.L., Harry, G.J., Zeiger, E., Shelby, M., and Ris, C.H. Potential health effects of oxygenated gasoline. In: Interagency Assessment of Oxygenated Fuels. National Science and Technology Council, Washington, DC, pp. 4.1-4.38, 1997.

7. Melnick, R.L. 1,3-Butadiene. *In*: Encyclopaedia of Occupational Health and Safety, Fourth Edition, Rubber Industry. Beliczky, L.S. and Fajen, J. (eds.). International Labour Office, Geneva. p. 80.9, 1998.
8. Melnick, R.L. and Kohn, M.C. Possible mechanisms of induction of renal tubular cell neoplasms in rats associated with  $\alpha_{2u}$ -globulin: role of protein accumulation versus ligand delivery to the kidney. In "Species Differences in Thyroid, Kidney and Urinary Bladder Carcinogenesis", Capen, C.C., Dybing, E., Rice, J.M., and Wilbourn, J.D. (eds). IARC Scientific Publication No. 147. International Agency for Research on Cancer, Lyon, France, pp 119-137, 1999.
9. Aggarwal G, Kohn MC, and Melnick RL. Development of a physiologically based pharmacokinetic model for isoprene, In Toxicology and Carcinogenesis Studies of Isoprene (CAS No. 78-79-5) in F344/N Rats an (Inhalation Studies). NTP Technical Report 486, NIH Publication 97-3976. National Toxicology Program, Research Triangle Park, NC, 1999.
10. Melnick R.L. Chloroform: Different viewpoints on the cancer risk assessment. The Toxicology Forum – Winter 1999. The Toxicology Forum, Washington, DC, pp. 135-141, 2000.
11. Melnick, R.L. Occupational Chemical Carcinogenesis. In "Patty's Industrial Hygiene and Toxicology", 5th Edition. Bingham, E, Cohrssen, B., Powell, C.H. (eds). John Wiley & Sons, Inc., New York. Vol. 1, pp. 117-167, 2001.
12. Melnick, R.L. Summary of the NTP/NIEHS endocrine disrupters low-dose peer review, In International Symposium on Environmental Endocrine Disrupters 2000 Report. Environment Agency, Pacifico Yokohama, Japan, pp. 459-468, 2001.
13. Mehlman, M.A., Bingham, E., Landrigan, P., Soffritti, M., Belpoggi, F., and Melnick, R.L., Eds. Carcinogenesis Bioassays and Protecting Public Health. Vol. 982. New York Academy of Sciences, New York, NY, 2002.
14. Melnick, R.L. Derivation and modeling of mechanistic data for use in risk assessment. In "Toxicokinetics and Risk Assessment," J.C. Lipscomb and E.V. Ohanian (Eds). Informa Healthcare, New York, pp. 47-68, 2006.
15. Melnick, R.L. and Kissling G.E. Cancer risk evaluation from animal studies. In "Encyclopedia of Quantitative Risk Analysis and Assessment," E. Melnick and B. Everitt (Eds). John Wiley & Sons, UK, Vol 4, pp. 179-180, 2008.
16. Melnick, R.L. and Hooth, M.J. Carcinogenicity of disinfection byproducts in laboratory animals. In "Encyclopedia of Environmental Health," J.O. Nriagu (Ed), Elsevier Ltd., UK, Vol. 1, pp. 516-523, 2011.
17. Bond, J. and Melnick, R.L. Electrophilic compounds. In "Tumour concordance and mechanisms of carcinogenesis". IARC Scientific Publication (in press).

18. Caldwell, J.C., Melnick, R.L., and Zeise, L. Host susceptibility: Factors influencing tumour site concordance between humans and experimental animals exposed to environmental carcinogens. In "Tumour concordance and mechanisms of carcinogenesis". IARC Scientific Publication (in press).

19. Krewski, D., Rice, J.M., Bird, M., .....Melnick, R. Concordance between sites of tumor development in humans and in experimental animals for 111 agents that are carcinogenic to humans. In "Tumour concordance and mechanisms of carcinogenesis". IARC Scientific Publication (in press).

### *Technical Reports*

1. Carcinogenesis Bioassay of Agar (CAS No. 9002-18-0) in F344 Rats and B6C3F<sub>1</sub> Mice (Feed Study). NTP-TR-230. NIH Publication No. 82-1786, 1982.
2. Carcinogenesis Bioassay of Melamine (CAS No. 108-78-1) in F344/N Rats and B6C3F<sub>1</sub> Mice (Feed Study). NTP-TR-245. NIH Publication No. 83-2501, 1983.
3. Toxicology and Carcinogenesis Studies of Chlorpheniramine Maleate (CAS No. 113-92-8) in F344/N Rats and B6C3F<sub>1</sub> Mice (Gavage Studies). NTP-TR-317. NIH Publication No. 86-2573, 1986.
4. Toxicology and Carcinogenesis Studies of Tribromomethane (Bromoform) (CAS No. 75-25-2) in F344/N Rats and B6C3F<sub>1</sub> Mice (Gavage Studies). NTP-TR-350. NIH Publication No. 88-2805, 1988.
5. Toxicology and Carcinogenesis Studies of 2,4-Dichlorophenol (CAS No. 120-83-2) in F344/N Rats and B6C3F<sub>1</sub> Mice (Feed Studies). NTP-TR-353. NIH Publication No. 88-2808, 1988.
6. Toxicology and Carcinogenesis Studies of Diphenhydramine Hydrochloride (CAS No. 147-24-0) in F344/N Rats and B6C3F<sub>1</sub> Mice (Feed Studies). NTP-TR-355. NIH Publication No. 89-2810, 1989.
7. Toxicology and Carcinogenesis Studies of Succinic Anhydride (CAS No. 108-30-5) in F344/N Rats and B6C3F<sub>1</sub> Mice (Gavage Studies). NTP-TR-373. NIH Publication No. 89-2828, 1990.
8. Toxicology and Carcinogenesis Studies of 2-Chloroacetophenone (CAS No. 532-27-4) in F344/N Rats and B6C3F<sub>1</sub> Mice (Inhalation Studies). NTP-TR-379. NIH Publication No. 89-2834, 1990.
9. Toxicology and Carcinogenesis Studies of 1,3-Butadiene (CAS No. 106-99-0) in B6C3F<sub>1</sub> Mice (Inhalation Studies). NTP-TR-434. NIH Publication No. 93-3165, 1993.

10. Toxicity Studies of Diethanolamine (CAS No. 111-42-2) Administered Topically and in Drinking Water to F344/N Rats and B6C3F<sub>1</sub> Mice NTP-TOX-20. NIH Publication No. 92-3343, 1992.
11. Toxicity Studies of Isoprene (CAS No. 78-79-5) Administered by Inhalation to F344/N Rats and B6C3F<sub>1</sub> Mice. NTP-TOX-31. NIH Publication No. 95-3354 (1995).
12. Interagency Assessment of Potential Health Risks Associated with Oxygenated Gasoline. National Science and Technology Council, 1996.
13. Toxicology and Carcinogenesis Studies of Chloroprene (CAS No. 126-99-8) in F344/N Rats and B6C3F<sub>1</sub> Mice (Inhalation Studies). NTP-TR-467. NIH Publication No. 96-3957, 1998.
14. Toxicology and Carcinogenesis Studies of Isoprene (CAS No. 78-79-5) in F344/N Rats (Inhalation Studies). NTP-TR-486. NIH Publication No. 97-3976, 1999.
15. Toxicology and Carcinogenesis Studies of Bromodichloromethane (CAS No. 75-27-4) in Male F344/N Rats and Female B6C3F<sub>1</sub> Mice (Drinking Water Studies). NTP-TR-532. NIH Publication No. 05-4468, 2006.
16. Toxicology and Carcinogenesis Studies of Dibromoacetic Acid (CAS No. 631-64-1) in F344/N Rats and B6C3F<sub>1</sub> Mice (Drinking Water Studies). NTP-TR-537. NIH Publication No. 05-4475, 2007.
17. Toxicology and Carcinogenesis Studies of Bromochloroacetic Acid (CAS No. 5589-96-8) in F344/N Rats and B6C3F<sub>1</sub> Mice (Drinking Water Studies). NTP-TR-549. NIH Publication No. 09-5890, 2009.
18. Toxicology and Carcinogenesis Studies of Dibromoacetonitrile (CAS No. 3252-43-5) in F344/N Rats and B6C3F<sub>1</sub> Mice (Drinking Water Studies). NTP-TR-544. NIH Publication No. 10-5886, 2010.
19. Exposure and Interaction: the Potential Health Impacts of Using Multiple Pesticides. Zaunbrecher V, Hattis D, Melnick R, Kegley S, Malloy T, and Froines J. UCLA Sustainable Technology & Policy Program, 2016.