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May 5, 2009

Via E-Mail and Overnight Mail

Cynthia Oshita
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Proposition 65 Implementation
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Re: Comments on the Presentation of Perfluorooctanoic Acid and Its Salts for
“Consultation” to the CIC

Dear Ms. Oshita:

More than three years ago, on February 22, 2006, a coalition of labor, environmental, and other concerned groups including the United Steel, Paper and Forestry, Rubber, Manufacturing, Energy, Allied Industrial and Service Workers International Union, AFL-CIO, CLC (“USW”); the Sierra Club; the Environmental Law Foundation; the Environmental Working Group; the U.S. Public Interest Research Group; Environment California; and the Natural Resources Defense Council (and later joined by the California Labor Federation) submitted a petition proposing perfluorooctanoic acid and its salts (“PFOA”) for consideration and listing by the Carcinogen Identification Committee (“CIC”) under Proposition 65 as a chemical “known to the state to cause cancer.” California Health and Safety Code §25249.8(b); 22 C.C.R. §12305(a)(1). The Petition presented scientific evidence demonstrating that PFOA has pervasive and widespread exposure in California, and met the standard for listing as a carcinogen. OEHHA denied the Petition on December 6, 2006, even though four members of the CIC voted at its November meeting to expedite consideration of the chemical for listing.

OEHHA has taken no further action with respect to the chemical until March 2009 – when it announced that PFOA was among a group of 38 chemicals that had passed two “screens”

in its Prioritization Process. Although the recent decision to present 38 chemicals for the “advice on the prioritization of these chemicals for *possible* preparation of hazard identification materials” is movement in the right direction, the glacial pace at which OEHHA has proceeded with respect to its mandatory duty to list chemicals as carcinogens under Proposition 65 is wholly inconsistent with the Act. Since the implementation of this “Prioritization Process” in 2004, despite the *hundreds* of chemicals in OEHHA’s tracking database, OEHHA has presented only three chemicals to the CIC for consideration for listing. The presentation of only three chemicals over the past five years is inconsistent with the mandate imposed by Proposition 65.

PFOA met the standard for listing as a chemical known to the state to cause cancer when the initial Petition was filed in early 2006. It still meets that standard today. OEHHA and the CIC’s extraordinary and unnecessary delay in listing chemicals – including PFOA – that meet the Proposition 65 standard for listing as carcinogens must end.

I. PFOA Meets the Proposition 65 Standard for Listing as a Carcinogen

PFOA must be listed under Proposition 65 if it “has been clearly shown through scientifically valid testing according to generally accepted principles to cause cancer.” California Health and Safety Code §25249.8(b). The CIC is charged with listing such chemicals. 22 C.C.R. §12305(1). In particular, under the governing regulations, a chemical is to be listed as causing cancer if “studies in experimental animals indicate that there is an increased incidence of malignant tumors or combined malignant and benign tumors in multiple species or strains, in multiple experiments (e.g., with different routes of administration or using different dose levels), or, to an unusual degree, in a single experiment with regard to high incidence, site, or type of tumor, or age at onset.” 22 C.C.R. §12306(e)(2).

PFOA is a synthetically-produced fluorochemical compound that is ubiquitous in modern consumer and industrial products. PFOA is used to create non-stick and stain-resistant surfaces on consumer products including cookware. PFOA also has numerous and varied industrial uses, in almost all industry segments, including the aerospace, automotive, building/construction, chemical processing, electrical and electronics, semiconductor, and textile industries.¹ PFOA is not only used in the manufacture of consumer and industrial products, but can be released into the atmosphere during their use, such as in the heating of non-stick cookware.² Because PFOA is not naturally occurring, all PFOA in the environment is attributable to human activity.³

¹ U.S. Environmental Protection Agency, “Basic Information on PFOA,” available at <http://www.epa.gov/opptintr/pfoa/pfoainfo.htm>.

² Environmental Working Group, “PFCs: Global Contaminants, available at <http://www.ewg.org/reports/pfcworld>.

³ U.S. Environmental Protection Agency, “Perfluorooctanoic Acid (PFOA), Fluorinated Telomers; Request for Comment, Solicitation of Interested Parties for Enforceable Consent

In 2005, the EPA reached a settlement with DuPont that imposes the largest civil administrative penalty in EPA's history, \$16.5 million, against DuPont for violations of reporting provisions of the federal Toxic Substances Control Act ("TSCA") and the Resource Conservation and Recovery Act ("RCRA") with respect to PFOA.⁴ The settlement was based on violations involving DuPont's failure to report information about substantial risk of injury to human health or the environment that DuPont obtained about PFOA from as early as 1981 and as recently as 2004.⁵ EPA's TSCA claim was based in large part on the discovery of a 1981 DuPont document that revealed the results of DuPont's testing of the blood of pregnant women and infants, and in one case, umbilical blood, at one of DuPont's PFOA manufacturing facilities.⁶ The document revealed that PFOA was transplacental and reported at least two children born with birth defects.⁷ Among the allegations in EPA's Complaint relevant to the TSCA claim were: "PFOA is biopersistent in animals and humans," "PFOA is bioaccumulative in humans," "PFOA is associated with developmental effects in animals," and "PFOA is in the blood of the general population in all geographic regions of the U.S."⁸ EPA also alleged that "EPA's efforts to characterize effects of PFOA might have been more expeditious had the data on transplacental movement of the chemical in humans been submitted immediately by DuPont when DuPont obtained the information in 1981."⁹

In 2002 the EPA initiated a "priority review" of PFOA in light of evidence of the chemical's toxic effects. In 2005, the EPA issued a Draft Risk Assessment, which concludes that there is evidence that PFOA is carcinogenic in animals.¹⁰ On February 15, 2006, EPA's Science

Agreement, Development, and Notice of Public Meeting," 68 Fed. Reg. 18626-01 (April 16, 2003).

⁴ U.S. EPA, News Release, "EPA Settles PFOA Case Against DuPont for Largest Environmental Administrative Penalty in Agency History" (December 14, 2005), available at: <http://www.epa.gov/cgi-bin/epaprintonly.cgi>.

⁵ *Id.*

⁶ U.S. EPA, Complaint and Notice of Opportunity for Hearing, Docket Nos. TSCA-HQ-2004-0016 and RCRA-HG-2004-0016, at ¶¶34-46, available at <http://www.epa.gov/compliance/resources/complaints/civil/mm/dupont-pfoa-complaint.pdf>.

⁷ *Id.*

⁸ *Id.* at ¶¶10-13.

⁹ *Id.* at ¶45.

¹⁰ U.S. EPA, Draft Risk Assessment of the Potential Human Health Effects Associated With Exposure to Perfluorooctanoic Acid And Its Salts, Office of Pollution Prevention and

Advisory Board, which consists of non-government scientific experts drawn from academia and industry, voted to approve a recommendation that the EPA increase its categorization of PFOA in the Draft Risk Assessment from “suggestive evidence of carcinogenicity” to “likely to be carcinogenic” in humans.¹¹ On June 20, 2006, EPA announced that it would continue to analyze research that had become available since the 2005 report and would resubmit a report to the EPA’s Science Advisory Board upon completion of that revision at some unspecified date in the future.¹² Thus, almost five years after EPA announced its “priority review” of PFOA, EPA has no plans to issue a final report on the potential human health effects of the chemical in the near future.

In the meantime, EPA has asked eight companies that manufacture PFOA, use PFOA in the manufacture of fluoropolymers, or use chemicals that break down into PFOA to agree voluntarily to reduce their PFOA releases and its presence in products by 95 percent by no later than 2010 and to work toward eliminating these sources of exposure five years after that and no later than 2015, but has taken no other steps to regulate the chemical.¹³

The bio-accumulation of PFOA is a very serious concern in light of the body of evidence that demonstrates that PFOA causes cancer. Multiple studies have documented that PFOA causes liver, pancreatic, and Leydig cell (testicular) cancer in animals.

- In one study, groups of rats were fed diets containing 0, 30, or 300 ppm of a PFOA salt (APFO) for two years, with an average consumption per day of 14.2 mg/kg-day for male rats and 16.1 mg/kg-day for female rats.¹⁴ Postmortem studies were conducted on all rats that died throughout the study, as well as on a group selected at the one year interim, and all remaining rats at the two-year termination of the experiment. The study found a statistically significant, dose-related increase in Leydig cell adenomas in male rats (4% and 14% in the low- and high-dose groups, compared to 0% in the control group and .82% in historical

Toxics, Risk Assessment Division (January 4, 2005), at 8.

¹¹ U.S. EPA, Science Advisory Board, Draft Report (January 20, 2006), available at http://www.epa.gov/sab/pdf/2006_0120_final_draft_pfoa_report.pdf.

¹² U.S. EPA, Letter of June 20, 2006 from Administrator Stephen Johnson to EPA SAB Co-Chairs, available at http://www.epa.gov/sab/pdf/sab-06-006_response_06-20-06.pdf.

¹³ Information on U.S. EPA’s “2010/15 PFOA Stewardship Program” is available at <http://www.epa.gov/oppt/pfoa/pubs/pfoastewardship.htm>.

¹⁴ Sibinski, L. J. 1987. Two-Year oral (diet) toxicity/carcinogenicity study of fluorochemical FC-143 (perfluorooctane ammonium carboxylate) in rats. Riker Laboratories, Inc., Experiment No. 0281CR0012, available at U.S. EPA Public Docket AR-226-0437.

controls). This study also found an increase in the incidence of mammary fibroadenomas in female rats (at a rate of 43% in the high-dose group, compared to 21% in the control).¹⁵

- That PFOA causes Leydig-cell tumors was confirmed in a later study of PFOA toxicity in male rats.¹⁶ This study fed PFOA to the animals by gavage at 300 ppm for 2 years, and analyzed the animals at 1, 3, 6, 9, 12, 15, 18, and 21 months. The scientists found a statistically significant increase in Leydig-cell tumors in the treated rats (11%, compared to 0% in the control group).
- The second study found liver and pancreatic tumors as well. The treated rats exhibited significantly increased hepatic β -oxidation activity and increased incidence of hepatocellular adenomas (at a rate of 13%, compared to 3% in the control group). The study also found a statistically significant incidence of pancreatic acinar cell adenomas and carcinomas (at a rate of 11%, compared to the control rate of 0%).
- Other studies have also demonstrated that PFOA acts as a promoter of liver tumors in rats when combined with other cancer initiators.¹⁷

In light of these studies, PFOA meets (and has long met) the requirement for listing as a chemical causing cancer under the standard set forth in 22 C.C.R. §12306(e)(2). We note that

¹⁵ According to the EPA, this study improperly concluded that the increased rates of mammary fibroadenomas were not statistically significant, based on an improper historical control rate from an earlier study. U.S. EPA, Draft Risk Assessment of the Potential Human Health Effects Associated With Exposure to Perfluorooctanoic Acid And Its Salts, Office of Pollution Prevention and Toxics, Risk Assessment Division (January 4, 2005), at 57. The EPA report concludes that the increase shown in the study is statistically significant when compared to the historical control incidence for mammary fibroadenomas of 19% that has been used in 17 carcinogenicity studies. *Id.*

¹⁶ Biegel, L. B., Hurtt, M. E., Frame, S. R., O'Connor, J. C. and Cook, J. C. 2001. Mechanisms of Extrahepatic Tumor Induction by Peroxisome Proliferators in Male CD Rats. *Toxicol. Sci.* 60: 44-55; Cook, J.C., Hurtt, M.E., Frame, S.R., and Biegel, L.B. 1994. Mechanisms of extrahepatic tumor induction by peroxisome proliferators in Crl:CD BR (CD) rats. *Toxicologist* 14:301 (abstract #1169).

¹⁷ Abdellatif, A.G., Preat, V., Taper, H.S., and Roberfroid, M. 1991. The modulation of rat liver carcinogenesis by perfluorooctanoic acid, a peroxisome proliferator. *Toxicology and Applied Pharmacology.* 111(3): 530-7; Nilsson, R., Beije, B., Preat, V., Erxon, K., and Ramel, C. 1991. On the mechanism of the hepatocarcinogenicity of peroxisome proliferators. *Chem. Biol. Interact.* 78: 235-250.

your March 2009 document announcing the consideration of PFOA by the CIC categorizes PFOA as “One study and second with benign tumors only” rather than “Two or more studies.” That characterization is inconsistent with the above evidence.

II. Every Californian Has Been and Continues to Be Exposed to PFOA

Unlike many chemicals that come before the CIC, the vast majority of California residents likely have been exposed to this chemical, and actually have some amount of this chemical in their blood.

The stable carbon-fluorine bonds that make PFOA such a pervasive and successful industrial and consumer product also result in its persistence. *There is no known environmental breakdown mechanism for this chemical.*¹⁸ As a result of the chemical’s stability and pervasive use, the concentrations of PFOA have rapidly increased in the soil, water, and air, and in biological systems, including humans and animals.

Numerous studies have shown that non-occupational exposure to PFOA occurs *daily*, in people of all ages, from infants to the elderly, and that the chemicals may persist in human blood *for years*.¹⁹ For decades, the public has been exposed to PFOA from countless consumer products ranging from cookware, food packaging, carpets and upholstery, automotive products and even cosmetics. As a result of its pervasive use in consumer and industrial products, PFOA exists in the blood of the general U.S. population.²⁰ Indeed, one study found that approximately

¹⁸ Burris, J.M., Lundberg, J.K., Olsen, G., Simpson, C., and Mandel, J. 2002. Determination of Serum Half-Lives of Several Fluorochemicals (Interim Report No. 2), 3M Company, St. Paul, MN, available at USEPA Public Docket AR-226; Corsolini, S. and Kannan, K. 2004. Perfluorooctanesulfonate and related fluorochemicals in several organisms including humans from Italy. *Organohalogen Compounds* 66:4079-4085.

¹⁹ Burris (2002).

²⁰ U.S. Environmental Protection Agency, “Perfluorooctanoic Acid (PFOA), Fluorinated Telomers; Request for Comment, Solicitation of Interested Parties for Enforceable Consent Agreement, Development, and Notice of Public Meeting,” 68 Fed. Reg. 18626-01 (April 16, 2003). Examples of studies reporting the prevalence of PFOA in human blood include the following: Olsen, G.W., Church, T.R., Miller, J.P., Burris, J.M., Hansen, K.J., Lundberg, J.K., Armitage, J.M., Herron, R.M., Medhdizadehkashi, Z., Nobiletti, J.B., O’Neill, E.M., Mandel, J.H., and Zobel, L.R. 2003. Perfluorooctanesulfonate and other fluorochemicals in the serum of American Red Cross adult blood donors. *Environ. Health Perspect.* 111(16):1892–1901; Olsen, G.W., Hansen, K.J., Stevenson, L.A., Burris, J.M., and Mandel, J.H. 2003. Human donor liver and serum concentrations of perfluorooctanesulfonate and other perfluorochemicals. *Environ. Sci. Technol.* 37: 888–891; Olsen, G.W., Church, T.R., Larson, E.B., van Belle, G., Lundberg, J.K., Hansen, K.J., Burris, J.M., Mandel, J.H., and Zobel, L.R. 2004. Serum concentrations of

96% of the U.S. children tested had PFOA in their blood.²¹ Two studies have found PFOA in donated adult blood from a Los Angeles blood bank and in California's children.²² Measurable levels have been documented also in the umbilical cord blood of a very high proportion of newborn infants in the United States.²³ A study of Baltimore infants detected PFOA in 100% of the 299 umbilical cords tested, with no demographic or socioeconomic differences in concentration, leading the authors to conclude PFOA is ubiquitous in babies born in Baltimore.²⁴

PFOA's harms are not limited to consumer exposure. In 2006, the USW tested the blood of union members employed by DuPont and retirees in New Jersey who were exposed to PFOA and related chemicals. The tests revealed exposure rates hundreds of times higher than the general public and were later confirmed by DuPont's own serum tests submitted to the EPA in 2007.²⁵ In addition, in April 2009, DuPont revealed that workers at two Delaware locations also have PFOA in their blood at levels far higher than the general public.²⁶ Early case studies of workers exposed to PFOA revealed increased levels of prostate cancer among other health

perfluorooctanesulfonate and other fluorochemicals in an elderly population from Seattle, Washington. *Chemosphere* 54:1599–1611; Olsen, G.W., Church, T.R., Hansen, K.J., Burris, J.M., Butenhoff, J.L., Mandel, J.H., and Zobel, L.R. 2004. Quantitative evaluation of perfluorooctanesulfonate (PFOS) and other fluorochemicals in the serum of children. *J. Children's Health* 2:1–24; Kannan, K., Corsolini, S., Falandysz, J., Fillmann, G., Kumar, K.S., Loganathan, B.G., Mohd, M.A., Olivero, J., Van Wouwe, N., Yang, J.H., and Aldoust, K.M. 2004. Perfluorooctanesulfonate and related fluorochemicals in human blood from several countries. *Environ. Sci. Technol.* 38(17): 4489-95.

²¹ Olsen, G.W., Burris, J.M., Lundberg, J.K., Hansen, K.J., Mandel, J.H., and Zobel, L.R. 2002. Identification of Fluorochemicals in Human Sera: III. Pediatric Participants in a Group A Streptococci Clinical Trial Investigation (3M Company, Medical Department, Epidemiology, St. Paul, MN), U.S. EPA Public Docket AR-226-1085.

²² Olsen (2003) *Environ. Health Perspect.* 111:1892–1901; Olsen (2002).

²³ Apelberg et al. 2007. Determinants of Fetal Exposure to Polyfluoroalkyl Compounds in Baltimore, Maryland. *Environ. Sci. Technol.* 41: 3891-3897.

²⁴ Apelberg et al. 2007. Determinants of Fetal Exposure to Polyfluoroalkyl Compounds in Baltimore, Maryland. *Environ. Sci. Technol.* 41: 3891-3897.

²⁵ Perfluorooctanoic Acid (PFOA) Updated Occupational Serum Sampling, Chambers Works Facility, Deepwater, New Jersey; submitted to EPA June 4, 2008 by DuPont Corporation.

²⁶ Eder, A. PFOA levels in DuPont's Delaware workers are elevated. *Delaware News Journal* (April 1, 2009).

effects.²⁷

The evidence of PFOA's harmful health effects is accumulating. A recent study published by the UCLA School of Public Health in January 2009 found that women with higher blood levels of PFOA and a related chemical (PFOS) took longer to become pregnant.²⁸ Earlier epidemiological and animal studies also demonstrate that PFOA causes developmental and reproductive harm. Researchers from both government and industry have acknowledged these effects in published studies. In utero exposure of human infants to PFOA has been shown to cause decreased head circumference at birth, decreased birth weight, and possibly increased future risk of obesity and diabetes.²⁹ Animal studies of prenatal exposure show increased fetal death, reduced neonatal survival rates, and slowed neonatal weight gain.³⁰ Exposure during gestation in animal studies also causes a range of anatomical malformations.

The widespread and continuing exposure of Californians to this hazardous chemical warrants consideration of this chemical for listing as soon as possible.

III. Any Further Delay In Listing Chemicals Is Inconsistent with Proposition 65's Mandate

In the five years since OEHHA implemented the Prioritization Process in 2004, only three of the hundreds of candidate chemicals in OEHHA's tracking database have been presented to the CIC for listing, even though other chemicals meet the standard. The presentation of 38 chemicals to the CIC for "consultation" regarding the "possible" preparation of the hazard identification document OEHHA creates in advance of presenting a chemical to the CIC for listing consideration constitutes some forward movement but is far from sufficient to satisfy

²⁷ Gilliland, FD and Mandel. 1993. Mortality among employees of a perfluorooctanoic acid production plant. *J. Occup. Med.* 35(9): 950-4; Environmental Working Group, "PFCs: Global Contaminants, available at <http://www.ewg.org/reports/pfcworld>.

²⁸ Fei et al. 2009. Maternal Levels of perfluorinated chemicals and subfecundity. *Human Reproduction* Vol.1:1.

²⁹ Apelberg et al. 2007. Fetal Exposure to Perfluorooctane Sulfonate (PFOS) and Perfluorooctanoate (PFOA) in Relationship to Weight and Size at Birth. *Environ Health Perspect.* 2007 November; 115(11): 1670-1676.

³⁰ Lau et al. 2006. Effects of Perfluorooctanoic Acid Exposure During Pregnancy in the Mouse. *Toxicological Sciences* 90(2):510-518; Wolf et al. 2007. Developmental Toxicity of Perfluorooctanoic Acid (PFOA) in the CD-1 Mouse after Cross Foster and Restricted Gestational Exposures. *Toxicological Sciences* 95(2):462-473; White SS et al. 2007. Gestational PFOA exposure of mice is associated with altered mammary gland development in dams and female offspring. *Toxicol Sci* 96(1):133-44.

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OEHHA and the CIC's duties under the Act. The mandatory duty imposed by Proposition 65 requires nothing less than OEHHA and the CIC must list – without further delay – *every* chemical that meets the Proposition 65 standard for listing as a carcinogen. OEHHA cannot continue to take *years* to decide which chemicals to present to the CIC for “possible” consideration and then additional years to prepare the scientific summaries that the CIC will use to decide whether a chemical should be listed.

PFOA – and every other chemical in the group of 38 or in the tracking database in general that has been shown to cause cancer in human or animal studies – should be presented to the CIC for listing consideration immediately. Proposition 65 was enacted by the voters in direct response to concerns that state agencies had failed to provide adequate protection with respect to toxic chemicals. OEHHA and the CIC are not fulfilling this mandate, and must do more than in the past, and with greater urgency, over the course of the coming *months* rather than years, to ensure that these carcinogens are listed.

Finally, PFOA should also be listed as a chemical known to cause reproductive and developmental harm. On July 10, 2007, the petitioning labor and environmental groups also filed a petition with OEHHA and the Developmental and Reproductive Toxicant Identification Committee (“DART Identification Committee”) requesting that PFOA be listed as a reproductive toxicant on the basis that both epidemiological and animal studies demonstrate that PFOA causes developmental and reproductive harm. OEHHA denied that Petition and refused to present PFOA to the DART Identification Committee in light of its assessment that the epidemiological studies on PFOA did not pass OEHHA's screening mechanism. That decision must be reconsidered in light of the recent UCLA School of Public Health study that found that women with higher blood levels of PFOA and a related chemical (PFOS) took longer to become pregnant.³¹ OEHHA should present PFOA for consideration to the DART Identification Committee for listing as soon as possible.

Thank you for consideration of this important matter.

Sincerely,



Danielle Leonard

³¹ Fei et al. 2009. Maternal Levels of perfluorinated chemicals and subfecundity. Human Reproduction Vol.1:1.

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cc. Joan Denton, Director, OEHHA
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United Steel, Paper and Forestry, Rubber, Manufacturing, Energy, Allied Industrial and
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The Sierra Club
Environmental Law Foundation
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U.S. Public Interest Research Group
Environment California
Natural Resources Defense Council
California Labor Federation