OFFICE OF ENVIRONMENTAL HEALTH HAZARD ASSESSMENT

UNITED STEEL, PAPER AND FORESTRY, RUBBER, MANUFACTURING, ENERGY, ALLIED INDUSTRIAL AND SERVICE WORKERS INTERNATIONAL UNION, AFL-CIO, CLC; SIERRA CLUB; ENVIRONMENTAL LAW FOUNDATION; ENVIRONMENT CALIFORNIA; NATURAL RESOURCES DEFENSE COUNCIL; HEALTHY CHILD HEALTHY WORLD; AND CALIFORNIA LABOR FEDERATION, AFL-CIO, Petitioners.

PETITION TO DR. JOAN E. DENTON, DIRECTOR, OFFICE OF ENVIRONMENTAL HEALTH HAZARD ASSESSMENT RE: LISTING OF PERFLUOROOCTANOIC ACID (“PFOA”) AS A REPRODUCTIVE AND DEVELOPMENTAL TOXICANT UNDER PROPOSITION 65 EXPEDITED CONSIDERATION REQUESTED
INTRODUCTION

1. The United Steel, Paper and Forestry, Rubber, Manufacturing, Energy, Allied Industrial and Service Workers International Union, AFL-CIO, CLC; Sierra Club; Environmental Law Foundation; Environment California; Natural Resources Defense Council; Healthy Child Healthy World; and California Labor Federation, AFL-CIO, request that the Office of Environmental Health Hazard Assessment (“OEHHA”) propose perfluorooctanoic acid and its salts (“PFOA”) for consideration and listing by the Developmental and Reproductive Toxicant Identification Committee (“DART Identification Committee”) under Proposition 65 as a chemical that is “known to the state to cause reproductive toxicity.” California Health and Safety Code §25249.8(b); 22 C.C.R. §12305(b)(1).

2. PFOA is ubiquitous in industrial and consumer products and exists in the blood of virtually all humans, including the blood of fetuses and infants, who are more vulnerable to chemical exposure than adults. Epidemiological and animal studies 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. Animal studies of prenatal exposure show increased fetal death, reduced neonatal survival rates, and slowed neonatal weight gain. Exposure during gestation in animal studies also causes a range of anatomical malformations. Given these toxic effects and widespread exposure, California can wait no longer to regulate this toxic substance.

3. Twenty years ago, by an overwhelming vote, the voters of California enacted Proposition 65, the Safe Drinking Water and Toxic Enforcement Act, for a specific and overarching purpose: To enhance their protection from toxic chemicals from which slow moving government agencies had failed to provide protection. As one California appellate court put it: “Proposition 65 clearly reflects the result of public dissatisfaction with the state’s efforts at protecting the people and their water supply from exposure to hazardous chemicals.” AFL-CIO v. Deukmejian, 212 Cal.App.3d 425, 441 (1989).

Proposition 65 mandates publication of a list of chemicals that cause cancer or reproductive toxicity – the threshold and critical step in the statutory scheme – when certain conditions are met. Only through
expeditious listing could be the central purpose of Proposition 65 – allowing people to be told of significant health risks and protect themselves as a matter of personal choice – be accomplished.

4. Specifically, in Proposition 65, the people stated "that hazardous chemicals pose a serious potential threat to their health and well-being, that state government agencies have failed to provide them with adequate protection, and that these failures have been serious enough to lead to investigations by federal agencies of the administration of California’s toxic protection programs." *Id.* at 430 (quoting preamble). To counteract the threat of hazardous chemicals, Proposition 65 declares the following rights of Californians:

"(a) To protect themselves and the water they drink against the chemicals that cause cancer, birth defects, or other reproductive harm.

"(b) To be informed about exposures to chemicals that cause cancer, birth defects, or other reproductive harm.

"(c) To secure strict enforcement of the laws controlling hazardous chemicals and deter actions that threaten public health and safety.

*Id.* at 430-31 (quoting preamble).

5. Those policy goals – and Proposition 65’s mandate to carry them out – remain in full force and effect. The Proposition further requires “a diligent, thorough and continuing search for additional chemicals which evolving scientific knowledge demonstrates are subject to the Act.” *Id.* at 440. Both the scientific evidence and recent actions (and inactions) by government agencies with respect to PFOA conclusively demonstrate why expedited listing of PFOA is required to carry out Proposition 65’s essential purposes. More delay awaiting more studies or until some other governmental entity reaches closure would represent the very result the public intended to remedy by enacting Proposition 65 in 1986.

6. PFOA belongs to a class of chemicals known collectively as the perfluoroalkyl acids (PFAAs). PFOA is a highly controversial substance that, as will be discussed in detail below, has been shown in epidemiological and animal studies to cause developmental and reproductive harm. Moreover, PFOA is environmentally persistent, and has widespread human exposure. PFOA has been detected virtually universally in the blood of adults and children, and in umbilical cord blood.
7. It is against the above background that this petition should be assessed. By acting quickly to list, the debate over the levels of risk presented by PFOA can take place as Proposition 65 intended—with the burden of proof on the company responsible for any exposure to establish that the risks are insignificant and that the public right to know is unnecessary. For that process to be prevented by government delay in the initial listing would defeat the purpose of Proposition 65 and undermine the intent and confidence of California's electorate.

PFOA MEETS THE STANDARD FOR LISTING UNDER PROPOSITION 65

8. PFOA must be listed under Proposition 65 as a reproductive toxicant if it "has been clearly shown through scientifically valid testing according to generally accepted principles to cause . . . reproductive toxicity." California Health and Safety Code §25249.8(b). The DART Identification Committee is charged with listing such chemicals. 22 C.C.R. §12305(b)(1).

9. PFOA is a synthetically-produced fluorochemical compound that has powerful surfactant and water-repelling properties and 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. 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. Because PFOA is not naturally occurring, all PFOA in the environment is attributable to human activity. PFOA is a synthetically-produced fluorochemical compound that has powerful surfactant and water-repelling properties and 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. 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. Because PFOA is not naturally occurring, all PFOA in the environment is attributable to human activity.

10. The United States Environmental Protection Agency ("EPA") first identified the potential reproductive and developmental toxicity effects of PFOA as early as 2002. In light of an initial draft

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4 U.S. EPA, Draft Risk Assessment of the Potential Human Health Effects Associated With (continued...)
hazard assessment of PFOA, and the subsequent receipt of “additional animal toxicity data on PFOA" that suggest a potential for developmental/reproductive toxicity,” in 2002 EPA initiated a “priority review” to determine whether PFOA met the criteria for action under Section 4(f) of the Toxic Substance Control Act. Pursuant to that “priority review,” EPA issued a Draft Risk Assessment in 2005, which describes the evidence that PFOA causes reproductive and developmental effects in animals.

11. EPA has not finalized the 2005 Draft Risk Assessment. 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. 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.

12. 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. The settlement was based on violations involving DuPont’s failure to report information about substantial risk of injury to human health or the

4(...continued)


5 Id.

6 Id. at 8, 60-72.


environment that DuPont obtained about PFOA from as early as 1981 and as recently as 2004. 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. The document revealed that PFOA was transplacental and reported at least two children born with birth defects. 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.” 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.”

EPA has also 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 but no later than 2015, but has taken no other steps to regulate the chemical.

The stable carbon-fluorine bonds that make PFOA such a pervasive industrial and consumer product also result in its persistence. There is no known environmental breakdown mechanism. 

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9 Id.


11 Id.

12 Id. at ¶10-13.

13 Id. at ¶45.

for this chemical. 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.

As a result of its pervasive use in consumer and industrial products, PFOA is virtually universally present in the blood of the general U.S. population, and around the world. Indeed, one study found that approximately 96% of the U.S. children tested had PFOA in their blood. Two studies have found PFOA in donated adult blood from a Los Angeles blood bank and in California’s children. Measurable levels have been documented also in the umbilical cord blood of a very high proportion of...
newborn infants in the United States.\textsuperscript{20} Indeed, a very recent 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.\textsuperscript{21}

16. In general, infants and children are more vulnerable to exposure to environmental toxins than are adults.\textsuperscript{22} Children’s susceptibility results from two primary factors: increased or unique sensitivity to toxic effects of contaminants due to rapid growth and development; and increased exposure because of physical size and behavioral characteristics.\textsuperscript{23}

17. Human data on the developmental toxicity of PFOA are sparse, but disquieting. A study submitted only recently for scientific publication from Johns Hopkins University suggests that exposure in utero of human infants to PFOA is associated with decreased head circumference at birth, decreased birth weight, and possibly increased future risk of obesity and diabetes.\textsuperscript{24}

18. Animal studies show that PFOA is toxic to reproduction and development. Studies described below demonstrate that 1) prenatal exposures are associated with dose-related increased rates of fetal loss [resorption], reduced neonatal survival, and slowed neonatal body-weight gain; 2) prenatal exposures are also associated with abnormalities in mammary gland development in the offspring; 3) exposures during gestation are associated with a range of anatomical malformations in the offspring; and 4) exposures early in gestation appeared to result in the most damaging consequences. Representative studies include:

\begin{itemize}
\item Apelberg et al. 2007, pending publication. Fetal Exposure to Perfluorooctane Sulfonate (PFOS) and Perfluorooctanoate (PFOA) in Relationship to Weight and Size at Birth.
\end{itemize}

- This study by a team from EPA sought to characterize the developmental toxicity of PFOA in the mouse.
- Timed-pregnant CD-1 mice were given 1, 3, 5, 10, 20, or 40 mg/kg PFOA by oral gavage daily from gestational day (GD) 1 to 17; controls received an equivalent volume (10 ml/kg) of water.
- A major finding was that PFOA treatment produced dose-dependent full-litter resorptions (resorptions are the equivalent of spontaneous abortions); all dams in the 40-mg/kg group resorbed their litters. The study also found: 1) the percent of live fetuses was lower only in the 20-mg/kg group (74% vs. 94% in controls), and fetal weight was also significantly lower in this group; 2) the incidence of live birth was significantly lowered by PFOA: approximately 70% for the 10- and 20-mg/kg groups compared to 96% for controls; 3) postnatal survival was severely compromised at 10 or 20 mg/kg, and moderately so at 5 mg/kg; 4) dose-dependent growth deficits were detected in all PFOA-treated litters except the 1-mg/kg group.
- The authors concluded: “These data indicate maternal and developmental toxicity of PFOA in the mouse, leading to early pregnancy loss, compromised postnatal survival, delays in general growth and development, and sex-specific alterations in pubertal maturation.”


- This recent paper by investigators from U.S. EPA and the Center for Disease Control (“CDC”) sought to examine the relative contribution to the reproductive toxicity of PFOA of gestational and lactational exposures.
- Pregnant CD-1 mice were dosed on gestation days (GD) 1-17 with 0, 3, or 5 mg PFOA/kg body weight, and pups were fostered at birth to give seven treatment groups: unexposed controls, pups exposed in utero (3U and 5U), lactationally (3L and 5L), or in utero +
lactationally (3U + L and 5U + L). In the restricted exposure (RE) study, pregnant mice received 5 mg PFOA/kg from GD7-17, 10-17, 13-17, or 15-17 or 20 mg on GD15-17.

- Major findings were that treatment with 5 mg/kg on GD1-17 increased the incidence of whole litter loss, pups in surviving litters had reduced birth weights, and pup survival from birth to weaning was affected in 5U + L litters. In utero exposure (5U), in the absence of lactational exposure, was sufficient to produce postnatal body weight deficits and developmental delay in the pups. All PFOA-exposed pups had deficits in postnatal weight gain, and those exposed on GD7-17 and 10-17 also showed developmental delay in eye opening and hair growth.

- The authors concluded that the postnatal developmental effects of PFOA are due to gestational exposure. Exposure earlier in gestation produced stronger responses.


- This recent report from the University of North Carolina, U.S. EPA, and CDC sought to determine whether developmental effects of PFOA were linked to gestational time of exposure or to subsequent lactational changes.

- Timed-pregnant CD-1 mice were orally dosed with 5 mg PFOA/kg on gestation days (GD) 1-17, 8-17, 12-17, or vehicle on GD 1-17.

- Mean pup birth weights on postnatal day (PND) 1 in all PFOA-exposed groups were significantly reduced and decrements persisted until weaning.

- In addition, mammary glands from lactating dams and female pups on PND 10 and 20 were scored based on differentiation or developmental stages. A significant reduction in mammary differentiation among dams exposed GD 1-17 or 8-17 was evident on PND 10. On PND 20, delays in normal epithelial involution and alterations in milk protein gene expression were observed. All exposed female pups displayed stunted mammary epithelial branching and growth at PND 10 and 20.

In summary, the scientific literature demonstrates that PFOA meets the requirement for listing as a chemical causing reproductive toxicity under California Health and Safety Code §25249.8(b).
20. Unlike many chemicals that come before the DART Identification Committee, 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 widespread and continuing exposure of Californians to this hazardous chemical warrants an abbreviation of the typical prioritization procedures to protect the public health. OEHHA should therefore place PFOA on the agenda of the next scheduled meeting of the DART Identification Committee, according to the abbreviated listing procedure described in OEHHA, Process For Prioritizing Chemicals For Consideration Under Proposition 65 By The “State’s Qualified Experts,” December 2004. Given the potential severity of the health hazards caused by PFOA and the nearly universal exposure of the public, the DART Identification Committee should list PFOA under Proposition 65 as soon as possible.

21. Finally, in addition to acting immediately to list PFOA, OEHHA should examine other members of the class of PF AAs, such as perfluorooctyl sulfonate (“PFOS”), to determine whether to list those other members or, indeed, the entire class.

CONCLUSION

For the reasons stated above, the DART Identification Committee should consider PFOA at its next scheduled meeting and list PFOA under Proposition 65.

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Respectfully submitted,

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