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10 Rubber, Manufacturing, Energy, Allied Industrial and Service
Workers International Union, AFL-CIO, CLC; Sierra Club;
11 Environmental Law Foundation; Environment California;
Natural Resources Defense Council; Healthy Child
12 Healthy World; and California Labor Federation, AFL-CIO

13 OFFICE OF ENVIRONMENTAL HEALTH HAZARD ASSESSMENT

14 UNITED STEEL, PAPER AND FORESTRY,)
15 RUBBER, MANUFACTURING, ENERGY,)
ALLIED INDUSTRIAL AND SERVICE)
16 WORKERS INTERNATIONAL UNION, AFL-)
CIO, CLC; SIERRA CLUB;)
17 ENVIRONMENTAL LAW FOUNDATION;)
ENVIRONMENT CALIFORNIA; NATURAL)
18 RESOURCES DEFENSE COUNCIL;)
HEALTHY CHILD HEALTHY WORLD; AND)
19 CALIFORNIA LABOR)
FEDERATION, AFL-CIO,)
20)
Petitioners.)
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**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

1 INTRODUCTION

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

10 2. PFOA is ubiquitous in industrial and consumer products and exists in the blood of
11 virtually all humans, including the blood of fetuses and infants, who are more vulnerable to chemical
12 exposure than adults. Epidemiological and animal studies demonstrate that PFOA causes developmental
13 and reproductive harm. Researchers from both government and industry have acknowledged these
14 effects in published studies. In utero exposure of human infants to PFOA has been shown to cause
15 decreased head circumference at birth, decreased birth weight, and possibly increased future risk of
16 obesity and diabetes. Animal studies of prenatal exposure show increased fetal death, reduced neonatal
17 survival rates, and slowed neonatal weight gain. Exposure during gestation in animal studies also causes
18 a range of anatomical malformations. Given these toxic effects and widespread exposure, California can
19 wait no longer to regulate this toxic substance.

20 3. Twenty years ago, by an overwhelming vote, the voters of California enacted Proposition
21 65, the Safe Drinking Water and Toxic Enforcement Act, for a specific and overarching purpose: To
22 enhance their protection from toxic chemicals from which slow moving government agencies had failed
23 to provide protection. As one California appellate court put it: “Proposition 65 clearly reflects the result
24 of public dissatisfaction with the state’s efforts at protecting the people and their water supply from
25 exposure to hazardous chemicals.” *AFL-CIO v. Deukmejian*, 212 Cal.App.3d 425, 441 (1989).
26 Proposition 65 mandates publication of a list of chemicals that cause cancer or reproductive toxicity –
27 the threshold and critical step in the statutory scheme – when certain conditions are met. Only through
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1 expeditious listing could the central purpose of Proposition 65 – allowing people to be told of significant
2 health risks and protect themselves as a matter of personal choice – be accomplished.

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

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

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

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

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

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

24 6. PFOA belongs to a class of chemicals known collectively as the perfluoroalkyl acids
25 (PFAAs). PFOA is a highly controversial substance that, as will be discussed in detail below, has been
26 shown in epidemiological and animal studies to cause developmental and reproductive harm. Moreover,
27 PFOA is environmentally persistent, and has widespread human exposure. PFOA has been detected
28 virtually universally in the blood of adults and children, and in umbilical cord blood.

1 hazard assessment of PFOA, and the subsequent receipt of “additional animal toxicity data on [PFOA]
2 that suggest a potential for developmental/reproductive toxicity,” in 2002 EPA initiated a “priority
3 review” to determine whether PFOA met the criteria for action under Section 4(f) of the Toxic
4 Substance Control Act.⁵ Pursuant to that “priority review,” EPA issued a Draft Risk Assessment in
5 2005, which describes the evidence that PFOA causes reproductive and developmental effects in
6 animals.⁶

7 11. EPA has not finalized the 2005 Draft Risk Assessment. On June 20, 2006, EPA
8 announced that it would continue to analyze research that had become available since the 2005 report
9 and would resubmit a report to the EPA’s Science Advisory Board upon completion of that revision at
10 some unspecified date in the future.⁷ Thus, almost five years after EPA announced its “priority review”
11 of PFOA, EPA has no plans to issue a final report on the potential human health effects of the chemical
12 in the near future.

13 12. In 2005, the EPA reached a settlement with DuPont that imposes the largest civil
14 administrative penalty in EPA’s history, \$16.5 million, against DuPont for violations of reporting
15 provisions of the federal Toxic Substances Control Act (“TSCA”) and the Resource Conservation and
16 Recovery Act (“RCRA”) with respect to PFOA.⁸ The settlement was based on violations involving
17 DuPont’s failure to report information about substantial risk of injury to human health or the
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21 ⁴(...continued)
22 Exposure to Perfluorooctanoic Acid And Its Salts, Office of Pollution Prevention and Toxics, Risk
23 Assessment Division (January 4, 2005), at 11, available at
<http://www.epa.gov/opptintr/pfoa/pubs/pfoarisk.pdf>.

24 ⁵ *Id.*

25 ⁶ *Id.* at 8, 60-72.

26 ⁷ U.S. EPA, Letter of June 20, 2006 from Administrator Stephen Johnson to EPA SAB Co-
27 Chairs, available at http://www.epa.gov/sab/pdf/sab-06-006_response_06-20-06.pdf.

28 ⁸ 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>.

1 environment that DuPont obtained about PFOA from as early as 1981 and as recently as 2004.⁹ EPA's
2 TSCA claim was based in large part on the discovery of a 1981 DuPont document that revealed the
3 results of DuPont's testing of the blood of pregnant women and infants, and in one case, umbilical
4 blood, at one of DuPont's PFOA manufacturing facilities.¹⁰ The document revealed that PFOA was
5 transplacental and reported at least two children born with birth defects.¹¹ Among the allegations in
6 EPA's Complaint relevant to the TSCA claim were: "PFOA is biopersistent in animals and humans,"
7 "PFOA is bioaccumulative in humans," "PFOA is associated with developmental effects in animals,"
8 and "PFOA is in the blood of the general population in all geographic regions of the U.S."¹² EPA also
9 alleged that "EPA's efforts to characterize effects of PFOA might have been more expeditious had the
10 data on transplacental movement of the chemical in humans been submitted immediately by DuPont
11 when DuPont obtained the information in 1981."¹³

12 13. EPA has also asked eight companies that manufacture PFOA, use PFOA in the
13 manufacture of fluoropolymers, or use chemicals that break down into PFOA to agree voluntarily to
14 reduce their PFOA releases and its presence in products by 95 percent by no later than 2010 and to work
15 toward eliminating these sources of exposure five years after that but no later than 2015, but has taken
16 no other steps to regulate the chemical.¹⁴

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

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22 ⁹ *Id.*

23 ¹⁰ U.S. EPA, Complaint and Notice of Opportunity for Hearing, Docket Nos. TSCA-HQ-2004-
24 0016 and RCRA-HG-2004-0016, at ¶¶34- 46, available at
<http://www.epa.gov/compliance/resources/complaints/civil/mm/dupont-pfoa-complaint.pdf>.

25 ¹¹ *Id.*

26 ¹² *Id.* at ¶¶10-13.

27 ¹³ *Id.* at ¶45.

28 ¹⁴ Information on U.S. EPA's "2010/15 PFOA Stewardship Program" is available at
<http://www.epa.gov/oppt/pfoa/pubs/pfoastewardship.htm>.

1 *for this chemical.*¹⁵ As a result of the chemical's stability and pervasive use, the concentrations of PFOA
2 have rapidly increased in the soil, water, and air, and in biological systems, including humans and
3 animals. Numerous studies have shown that non-occupational exposure to PFOA occurs *daily*, in people
4 of all ages, from infants to the elderly, and that the chemicals may persist in human blood *for years.*¹⁶

5 15. As a result of its pervasive use in consumer and industrial products, PFOA is virtually
6 universally present in the blood of the general U.S. population, and around the world.¹⁷ Indeed, one
7 study found that approximately 96% of the U.S. children tested had PFOA in their blood.¹⁸ Two studies
8 have found PFOA in donated adult blood from a Los Angeles blood bank and in California's children.¹⁹
9 Measurable levels have been documented also in the umbilical cord blood of a very high proportion of
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12 ¹⁵ Burris, J.M., Lundberg, J.K., Olsen, G., Simpson, C., and Mandel, J. 2002. Determination of
13 Serum Half-Lives of Several Fluorochemicals (Interim Report No. 2), 3M Company, St. Paul, MN,
14 available at USEPA Public Docket AR-226; Corsolini, S. and Kannan, K. 2004.
15 Perfluorooctanesulfonate and related fluorochemicals in several organisms including humans from Italy.
16 *Organohalogen Compounds* 66:4079-4085.

17 ¹⁶ Burris (2002).

18 ¹⁷ U.S. EPA, "Perfluorooctanoic Acid (PFOA), Fluorinated Telomers; Request for Comment,
19 Solicitation of Interested Parties for Enforceable Consent Agreement, Development, and Notice of
20 Public Meeting," 68 Fed. Reg. 18626-01 (April 16, 2003). Examples of studies reporting the prevalence
21 of PFOA in human blood include the following: Olsen, G.W., Church, T.R., Miller, J.P., Burris, J.M.,
22 Hansen, K.J., Lundberg, J.K., Armitage, J.M., Herron, R.M., Medhdizadehkashi, Z., Nobiletti, J.B.,
23 O'Neill, E.M., Mandel, J.H., and Zobel, L.R. 2003. Perfluorooctanesulfonate and other fluorochemicals
24 in the serum of American Red Cross adult blood donors. *Environ. Health Perspect.* 111(16):1892-1901;
25 Olsen, G.W., Hansen, K.J., Stevenson, L.A., Burris, J.M., and Mandel, J.H. 2003. Human donor liver
26 and serum concentrations of perfluorooctanesulfonate and other perfluorochemicals. *Environ. Sci.*
27 *Technol.* 37: 888-891; Olsen, G.W., Church, T.R., Larson, E.B., van Belle, G., Lundberg, J.K., Hansen,
28 K.J., Burris, J.M., Mandel, J.H., and Zobel, L.R. 2004. Serum concentrations of
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.

¹⁸ 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.

¹⁹ Olsen (2003) *Environ. Health Perspect.* 111:1892-1901; Olsen (2002).

1 newborn infants in the United States.²⁰ Indeed, a very recent study of Baltimore infants detected PFOA
2 in 100% of the 299 umbilical cords tested, with no demographic or socioeconomic differences in
3 concentration, leading the authors to conclude PFOA is ubiquitous in babies born in Baltimore.²¹

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

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

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

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21 ²⁰ Apelberg et al. 2007. Determinants of Fetal Exposure to Polyfluoroalkyl Compounds in
Baltimore, Maryland. *Environ. Sci. Technol.* 41: 3891-3897.

22 ²¹ Apelberg et al. 2007. Determinants of Fetal Exposure to Polyfluoroalkyl Compounds in
23 Baltimore, Maryland. *Environ. Sci. Technol.* 41: 3891-3897.

24 ²² Landrigan et al. 2002. Environmental Pollutants and Disease in American Children: Estimates
25 of Morbidity, Mortality, and Costs for Lead Poisoning, Asthma, Cancer, and Developmental Disabilities.
Environ. Health Perspect. 110(7):721-728.

26 ²³ *Id.*; see also U.S. EPA Toxicity and Exposure Assessment for Children's Health (TEACH), at
27 <http://www.epa.gov/teach/teachintro.html>; U.S. EPA. 2002. Child-specific exposure factors handbook,
available at <http://fn.cfs.purdue.edu/fsq/WhatsNew/KidEPA.pdf>.

28 ²⁴ Apelberg et al. 2007, pending publication. Fetal Exposure to Perfluorooctane Sulfonate (PFOS)
and Perfluorooctanoate (PFOA) in Relationship to Weight and Size at Birth.

1 Lau et al. 2006. Effects of Perfluorooctanoic Acid Exposure During Pregnancy in the Mouse.
2 Toxicological Sciences 90(2):510-518.

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

21 Wolf et al. 2007. Developmental Toxicity of Perfluorooctanoic Acid (PFOA) in the CD-1 Mouse
22 after Cross Foster and Restricted Gestational Exposures. Toxicological Sciences 95(2):462-473.

- 23 • This recent paper by investigators from U.S. EPA and the Center for Disease Control
24 ("CDC") sought to examine the relative contribution to the reproductive toxicity of PFOA
25 of gestational and lactational exposures.
- 26 • Pregnant CD-1 mice were dosed on gestation days (GD) 1-17 with 0, 3, or 5 mg PFOA/kg
27 body weight, and pups were fostered at birth to give seven treatment groups: unexposed
28 controls, pups exposed in utero (3U and 5U), lactationally (3L and 5L), or in utero +

1 lactationally (3U + L and 5U + L). In the restricted exposure (RE) study, pregnant mice
2 received 5 mg PFOA/kg from GD7-17, 10-17, 13-17, or 15-17 or 20 mg on GD15-17.

- 3 • Major findings were that treatment with 5 mg/kg on GD1-17 increased the incidence of
4 whole litter loss, pups in surviving litters had reduced birth weights, and pup survival
5 from birth to weaning was affected in 5U + L litters. In utero exposure (5U), in the
6 absence of lactational exposure, was sufficient to produce postnatal body weight deficits
7 and developmental delay in the pups. All PFOA-exposed pups had deficits in postnatal
8 weight gain, and those exposed on GD7-17 and 10-17 also showed developmental delay
9 in eye opening and hair growth.
- 10 • The authors concluded that the postnatal developmental effects of PFOA are due to
11 gestational exposure. Exposure earlier in gestation produced stronger responses.

12 White SS et al. 2007. Gestational PFOA exposure of mice is associated with altered mammary
13 gland development in dams and female offspring. Toxicol Sci 96(1):133-44.

- 14 • This recent report from the University of North Carolina, U.S. EPA, and CDC sought to
15 determine whether developmental effects of PFOA were linked to gestational time of
16 exposure or to subsequent lactational changes.
- 17 • Timed-pregnant CD-1 mice were orally dosed with 5 mg PFOA/kg on gestation days
18 (GD) 1-17, 8-17, 12-17, or vehicle on GD 1-17.
- 19 • Mean pup birth weights on postnatal day (PND) 1 in all PFOA-exposed groups were
20 significantly reduced and decrements persisted until weaning.
- 21 • In addition, mammary glands from lactating dams and female pups on PND 10 and 20
22 were scored based on differentiation or developmental stages. A significant reduction in
23 mammary differentiation among dams exposed GD 1-17 or 8-17 was evident on PND 10.
24 On PND 20, delays in normal epithelial involution and alterations in milk protein gene
25 expression were observed. All exposed female pups displayed stunted mammary
26 epithelial branching and growth at PND 10 and 20.

27 19. In sum, the scientific literature demonstrates that PFOA meets the requirement for listing
28 as a chemical causing reproductive toxicity under California Health and Safety Code §25249.8(b).

