

McKenna Long
& Aldridge^{LLP}
Attorneys at Law

Atlanta

Denver

Los Angeles

Philadelphia

San Diego

San Francisco

Washington, DC

Brussels

101 California Street • 41st Floor • San Francisco, CA 94111
415.267.4000 • Fax: 415.267.4198
www.mckennalong.com

STANLEY W. LANDFAIR
(415) 267-4170

EMAIL ADDRESS
slandfair@mckennalong.com

November 9, 2006

VIA ELECTRONIC MAIL & FEDERAL EXPRESS

Thomas M. Mack, Ph.D., M.P.H., Committee Chairperson
Committee Members
Carcinogen Identification Committee

RE: *DuPont Response To Petition For Expedited Consideration Of PFOA*

Dear Dr. Mack and Committee Members:

On behalf of our client E.I. du Pont de Nemours and Company (“DuPont”), we are writing to provide information regarding the chemical substance known as perfluorooctanoic acid and its salts (referred to herein as “PFOA”) and whether the Office of Environmental Health Hazard Assessment should give “expedited consideration” to the question of whether PFOA should be designated as a carcinogen under Proposition 65. DuPont is informed that this issue will be discussed at the November 16, 2006 meeting of the Carcinogen Identification Committee (“Committee”), and will be pleased to appear at the meeting and participate in that discussion. In the meantime, we have requested that these comments be distributed for the Committee’s preparation and consideration in advance of the meeting.

SUMMARY

The issue for the Committee’s recommendation – whether PFOA should be given “expedited consideration” – arises from a petition filed on February 22, 2006. The petition claims, wrongly, that “animal studies of PFOA show that the substance meets the requirement for listing under Proposition 65.” This is incorrect. As demonstrated below, the animal data do not even approach the standard for listing.

In the words of the statute and its implementing regulations, PFOA has not been “clearly shown through scientifically valid testing according to generally accepted principles to cause

cancer.”¹ Even a cursory review of the carcinogenicity studies in animals and humans shows that PFOA does not come close to meeting the “clearly shown” standard of Proposition 65.

The animal data show that the chemical produces only benign tumors and (even then) only in one sex of one species (the male rat), and that PFOA is not genotoxic. Moreover, the fact that PFOA is a peroxisome proliferator raises questions about the relevance to humans of the benign tumors observed in male rats. Finally, epidemiological studies do not show carcinogenicity in humans, even in worker populations with historically high occupational exposure to PFOA.

The Committee’s “Guidance Criteria for Identifying Chemicals for Listing as ‘Known to the State to Cause Cancer’” states that “if the weight of evidence *clearly shows* that a certain chemical causes *invasive cancer* in humans, or that it causes *invasive cancer* in animals (unless the mechanism of action has been shown not to be relevant to humans), the committee will normally identify that chemical for listing.” (Emphasis added.) The term “invasive cancer” refers to malignant tumors (*i.e.*, tumors that metastasize), as opposed to benign tumors. No study of PFOA has shown any statistically significant increase in malignant tumors. It is inappropriate to recommend an expedited review for a chemical that does not meet the Proposition 65 listing criteria.

No regulatory agency or authoritative body has ever classified PFOA as a carcinogen. OEHHA has advised us in its October 13 letter that one of the reasons the Committee desires to determine whether consideration of PFOA should be expedited is the conclusion of a “majority of the [US EPA] Science Advisory Board Panel . . . that the potential for PFOA was consistent with the ‘likely to be carcinogenic’ descriptor.” In light of this concern, we believe it is important for the Committee to understand that US EPA has not accepted the application of that descriptor to PFOA. Rather, that issue is under consideration, and is expected to be resolved as US EPA finalizes its Draft Risk Assessment.

Under the Committee’s new prioritization procedure, PFOA would not be assigned even a “high” priority. Chemicals that do not pass the epidemiological screen ordinarily are not considered unless there is “very strong evidence from animal studies” that they are carcinogenic. The animal data on PFOA do not meet this standard. In short, a chemical should not receive an *expedited* review if it would not even receive a *high priority* under the prioritization procedure.

The scope of Proposition 65 is limited to cancer and reproductive toxicity, and the Committee’s role in implementing Proposition 65 is limited to identifying carcinogens for listing under the statute. It is not the role of OEHHA or the Committee under Proposition 65 to address generalized concerns about exposure to chemicals that are not carcinogenic. Nevertheless, even in declining to expedite consideration of PFOA for listing, the Committee can take comfort that no significant health risks will be ignored. As the Committee may be aware, the United States Environmental Protection Agency (“US EPA”) is conducting a comprehensive inquiry into *all*

¹ Cal. Health & Safety Code § 25249.8(b); Cal. Code Regs., *tit.* 22, § 12305(a)(1)

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potential health effects of PFOA, including potential carcinogenicity, as a result of its observation in June 2000 that PFOA is present (at very low levels) in blood samples of the general population. As part of that inquiry, US EPA is considering whether PFOA causes cancer. As recently as June 8, 2006, US EPA issued a public statement that ***“EPA has no information linking current levels of PFOA in the blood of the general public to any adverse effects in people.”*** Nothing has changed since that time. Nevertheless, US EPA is taking aggressive measures to reduce the level of emissions of PFOA into the environment, and it appears that those measures are working.

In light of these facts, there is no sound reason to give expedited consideration to the question of whether PFOA should be listed under Proposition 65. The petition is the only basis for expediting consideration of PFOA, and it is clear from the data recited in the petition itself that PFOA does not meet the criteria for listing. To place PFOA ahead of other chemicals that may present a genuine issue of carcinogenicity would only consume OEHHA's and the Committee's resources and time, and divert resources and priority from other chemicals that should, perhaps, be listed.

For these reasons, as discussed more thoroughly below, we believe the most appropriate course for the Committee is to recommend that OEHHA decline to expedite consideration of PFOA. All parties would benefit from the completion of US EPA's thorough, ongoing review, before OEHHA must decide whether or how fast to proceed. We trust you will agree, and look forward to discussing the matter with you further at the November 16 public meeting.

BACKGROUND REGARDING PFOA

What is PFOA? The chemical identity of PFOA is presented fully in the US EPA Draft Risk Assessment, a copy of which has been forwarded by OEHHA to the Committee.² Further elaboration on that issue is not necessary here. It is important, however, that the Committee be informed accurately as to the manner in which PFOA is produced and used.

PFOA (or a form of PFOA) is used as a processing agent to manufacture fluoropolymer resins and dispersions. Fluoropolymers are used to make products such as architectural fabrics, non-stick cookware, chemical processing piping and vessels, automotive fuel systems, telecommunications and electronic wiring insulation and computer chip processing equipment. DuPont uses PFOA as a processing aid in the manufacturing process for its Teflon® brand fluoropolymers. In this regard, it is important to recognize that ***PFOA is not Teflon®.***

PFOA also may be produced as an unintended byproduct in the manufacture of fluorotelomers and telomer-based products. Fluorotelomers are used to make surface protection

² “Draft Risk Assessment of the Potential Human Health Effects Associated with Exposure to Perfluorooctanoic Acid and its Salts,” US Environmental Protection Agency, Office of Pollution Prevention and Toxics Assessment Division, January 4, 2005, at 12.

products, including surfactants and repellents, for applications such as textiles, paper, fire fighting foam, non-wovens, coatings and stone and tile protection.

What are the sources of exposure to PFOA? The Committee undoubtedly is aware that the US EPA has been studying PFOA since approximately 2000, as a result of generalized concerns regarding exposure and bio-persistence. Extensive reports of bio-monitoring studies conducted since 1976 at manufacturing facilities in Minnesota, Alabama, West Virginia and Belgium are summarized in the US EPA Draft Risk Assessment.³ (The material has not been manufactured in California, to our knowledge.)

Extensive study notwithstanding, neither EPA nor manufacturers have determined the source of this exposure.^{4,5} It is clear, however, that there is no exposure to PFOA from the use of Teflon®-coated non-stick cookware. Indeed, US EPA has clearly stated that ***“EPA does not have any indication that the public is being exposed to PFOA through the use of Teflon®-coated cookware or other trademarked nonstick cookware. Teflon® and other trademarked products are not PFOA.”***⁶ A recent study by the US Food & Drug Administration similarly concluded that any migration of PFOA from non-stick cookware into food, even under extreme and abusive test methods not reflective of consumer use, was too small to measure.⁷ Government studies by the Danish Technological Institute, the Chinese State Testing Academy and the European Food Safety Authority all reached similar conclusions.

A study sponsored by DuPont confirms the conclusion that there is no significant exposure to PFOA from the use of articles produced using DuPont fluoropolymers or fluorotelomers that may contain PFOA. This study, conducted by Environ and independently peer-reviewed by a panel moderated by Dr. George Gray, Executive Director of the Harvard Center for Risk Analysis, concluded that ***any exposure to PFOA from the use of Teflon®-coated non-stick cookware, non-woven medical garments and those textiles tested was below***

³ “Draft Risk Assessment of the Potential Human Health Effects Associated with Exposure to Perfluorooctanoic Acid and its Salts,” US Environmental Protection Agency, Office of Pollution Prevention and Toxics Assessment Division, January 4, 2005, at 90-95.

⁴ “EPA does not have a full understanding of how people are exposed to PFOA.” Basic Information on PFOA: How are people exposed to PFOA?, <http://epa.gov/opptintr/pfoa/pubs/pfoainfo.htm>.

⁵ “At present, there are no steps that EPA recommends that consumers take to reduce exposures to PFOA because the sources of PFOA in the environment and the pathways by which people are exposed are unknown.” Basic Information on PFOA: Are there steps that consumers can take to reduce their exposure to PFOA?, <http://epa.gov/opptintr/pfoa/pubs/pfoainfo.htm>.

⁶ Basic Information on PFOA: Are there steps that consumers can take to reduce their exposure to PFOA?, <http://epa.gov/opptintr/pfoa/pubs/pfoainfo.htm>.

⁷ Begley, T., *et al.*, Food Additives and Contaminants, 22 (10), 2005.

detectable levels.⁸ The study shows that the use of these products will not result in measurable levels of PFOA in human blood.

COMMENTS IN OPPOSITION TO EXPEDITED LISTING

OEHHA has confirmed that the limited issue for the Committee's recommendation is *whether to expedite consideration* of PFOA as a candidate for listing under Proposition 65; *i.e.*, the issue is not whether to list PFOA, but whether the Committee and OEHHA should advance PFOA ahead of other chemicals, and give PFOA the most immediate priority for the Committee's review. Nevertheless, we find it difficult to separate the reasons why PFOA should not be expedited for consideration from the reasons why it should not be listed at all, if and when it is considered. Accordingly, we have included below a very brief summary of the carcinogenicity data on PFOA.⁹

PFOA is not a valid candidate for listing. The criteria under Proposition 65 and its implementing regulations for determining that a chemical is "known to the state to cause cancer" are recited above. The body of toxicological data on PFOA is extensive, and it is beyond the beyond the scope of these comments to survey all of the studies here. Nevertheless, it is clear that carcinogenicity studies on PFOA do not support listing.

PFOA has been investigated for carcinogenicity in two separate 2-year feeding studies in Sprague-Dawley rats. In the first study (Sibinski, 1987), PFOA was given to male and female rats at dietary levels of 0, 30, and 300 parts per million ("ppm"). In the second study (Biegel, *et al.*, 2001), PFOA was administered to male rats only at a single dietary level of 300 ppm, and the study included both *ad libitum* and pair-fed controls.

In the Sibinski study, there was an increase in the incidence of benign testicular Leydig cell tumors exposed to PFOA at a dietary dose of 300 ppm, but not at 30 ppm.¹⁰ In the Biegel *et*

⁸ Environmental Science & Technology 2005, 39(11), pp. 3904-3910.

⁹ We trust the Committee is aware that OEHHA has discussed PFOA with DuPont in an extensive meeting on June 28, 2006. OEHHA has provided the Committee with the extensive materials we shared with the Agency, both at our meeting and in a letter dated July 12, 2006. The information that follows includes some of the information provided to OEHHA, as well a further information that has developed since July 12.

¹⁰ There was some initial evidence of an increase in the incidence of mammary fibroadenomas in the female rats; the incidence was originally reported as 22%, 42%, and 48% at 0, 30, and 300 ppm in the diet, respectively. But there was no apparent difference in the incidence, despite dietary levels that were an order of magnitude apart. The study authors concluded that the mammary tumor data did not reflect an effect of PFOA. Unfortunately, the laboratory did not have an adequate historical control database for comparison. However, the historical control data of the supplier and of DuPont's Haskell Laboratories showed an average incidence of mammary fibroadenomas of 41% and 37%, respectively. A subsequent reevaluation of the mammary tissues by an independent Pathology Working Group showed no significant increase in mammary tumors in the Sibinski (1987) study. The details are presented in a later section of this letter.

al. study, there was an increase in benign hepatocellular, Leydig cell, and pancreatic acinar-cell tumors in rats exposed to PFOA at 300 ppm (Table 1). A similar finding on hepatocellular and pancreatic acinar-cell tumors was not observed in the Sibinski study, although a recent (2001) peer review of pancreatic tissues from both studies revealed evidence of acinar-cell hyperplasia in the Sibinski study, but no increase in adenoma.¹¹

Moreover, neither of the rat carcinogenicity studies shows a statistically significant increase in *malignant* tumors of any type. This is important because the Committee's "Guidance Criteria for Identifying Chemicals for Listing as 'Known to the State to Cause Cancer'" states that "if the weight of evidence *clearly shows* that a certain chemical causes *invasive cancer* in humans, or that it causes *invasive cancer* in animals (unless the mechanism of action has been shown not to be relevant to humans), the committee will normally identify that chemical for listing." (Emphasis added.) The term "invasive cancer" refers to malignant tumors. No study of PFOA has shown any statistically significant increase in "invasive cancer." The scientific evidence, therefore, does not support listing PFOA as a chemical that is "known to the State" to cause cancer.

SUMMARY OF HYPERPLASIA AND NEOPLASIA
 IN LIVER, TESTES, AND PANCREAS OF RATS FED PFOA
 (DATA FROM BIEGEL *et al.*, 2001)

ORGAN/LESION	CONTROL (FED AD LIB), INCIDENCE (%)	PAIR-FED CONTROL, INCIDENCE (%)	PFOA (300 PPM), INCIDENCE (%)
Liver			
Adenoma	2/80 (3)	1/79 (1)	10/76 (13) ^b
Carcinoma	0/80 (0)	2/79 (3)	0/76 (0)
Testes, Leydig-cell			
Hyperplasia	11/80 (14)	26/78 (33)	35/76 (46) ^a
Adenoma	0/80 (0)	2/78 (3)	8/76 (11) ^b
Pancreas, Acinar-cell			
Hyperplasia	14/80 (18)	8/79 (10)	30/76 (39) ^b
Adenoma	0/80 (0)	1/79 (1)	7/76 (9) ^b
Carcinoma	0/80 (0)	0/79 (0)	1/76 (1)

^a Significantly different from *ad libitum* control group.

^b Significantly different from pair-fed control group.

¹¹ Kennedy GL et al. (2004) The toxicology of perfluorooctanoate. Crit. Rev. Toxicol. 34(4):351-384.

Furthermore, animal testing data show that PFOA causes “peroxisome proliferation,” to which rats are uniquely susceptible. These studies call into question the relevance of studies showing benign tumors observed in rats, when assessing the effects of PFOA in humans.

As to other indicators of carcinogenic potential, the weight of the evidence from studies evaluating the genotoxicity of PFOA indicates that PFOA is not genotoxic. These studies include evaluations of mutagenicity, clastogenicity, and cell transformation.

Finally, the epidemiological data do not support a finding of carcinogenicity. Occupational epidemiological studies conducted on populations of significant size, including workers with prolonged exposure to PFOA at levels significantly higher than the potential exposures to the general public, did not show an increased risk of cancer.

One of the more extensive studies was a mortality study of approximately 4000 workers at the 3M Cottage Grove, Minnesota manufacturing facility who were exposed to PFOA for approximately 108,000 person-years, in which no significant increase in cancer risk was observed. In total, 3183 male and 809 female workers were followed for vital status from 1947 through 1997. The all-cause mortality (SMR = 0.9, 95% CI 0.8-0.9) and all-cancer mortality (SMR = 0.8, 95% CI 0.7-1.0) ratios for the entire study population regardless of classification, as well as for the exposure sub-cohorts, were less than expected in the general population. Specifically, there was no association between cohort members (employees with a minimum of 1 year employment in a job with definite or probable PFOA exposure) and all-cancer mortality (SMR = 0.9, 95% CI 0.7-1.1), liver cancer (SMR = 0.6, 95% CI 0.0-3.3), pancreatic cancer (SMR = 1.4, 95% CI -0.5-3.1), or prostate cancer (SMR = 1.2, 95% CI 0.4-2.5).

In a 2006 epidemiological study of workers at the DuPont Washington Works, West Virginia facility, about 6000 employees were followed for more than 50 years. No increased mortality risk was found in workers exposed to PFOA. The results showed lower mortality rates than those found in both West Virginia and the U.S. general population. They were also consistent with mortality rates in comparable workers from other DuPont plants, a worker population generally more healthy than the population at large. The study, which was reviewed by an external board of scientists, examined the occupations of 6,027 people who had worked at the Washington Works facility between 1948 until the end of 2002. It also examined the causes of death for those who had died over the 54-year period and compared mortality rates to those found in three groups: other DuPont workers, West Virginia residents, and members of the U.S. general population. Prostate cancer rates among the cases studied were found to be lower than rates in all three reference populations.¹²

¹² This contrasts with a previous non-DuPont study where an increase in prostate cancer initially was reported, but subsequently was discounted when the study was updated. Across the entire study population, there was a slight, but not statistically significant, site-wide increase in the rate of kidney cancer mortality. Only a third of the employees at the facility worked with PFOA, and most of the cases showed little exposure to PFOA; the numbers were too small to allow any conclusions.

PFOA does not merit a high priority for review under the OEHHA Prioritization Process. In considering whether to “expedite” consideration of PFOA, it is useful to consider what level of priority would be assigned to the chemical under OEHHA’s “Process for Prioritizing Chemicals for Consideration Under Proposition 65 by the ‘State’s Qualified Experts” (“Prioritization Process”). As the Committee is aware, the Prioritization Process was established recently, in 2004, at the request of the Committee, in part to avoid inefficiencies and misallocation of resources. Although OEHHA has discretion to vary from the Prioritization Process, it nevertheless establishes valid criteria for considering how the Committee and OEHHA should expend their resources, and which chemicals actually merit review.

Under the Prioritization Process, “all candidate chemicals initially undergo an epidemiological data screen. This involves the identification of those chemicals with epidemiological evidence suggesting they cause cancer” As discussed above, none of the epidemiological evidence suggests that PFOA causes cancer. Chemicals that do not pass the epidemiological screen ordinarily are not considered unless there is “very strong evidence from animal studies,” that they are carcinogenic. Again, the animal data on PFOA do not meet this standard.

PFOA has not been classified as a carcinogen by US EPA or any other authoritative body. OEHHA advised us in an October 13, 2006 letter advising us of the Committee meeting that one of the reasons to determine whether consideration of PFOA should be expedited is the conclusion of a “majority of the [US EPA] Science Advisory Board Panel . . . that the potential for PFOA was consistent with the ‘likely to be carcinogenic’ descriptor.” In light of this concern, we believe it is important for the Committee to understand that US EPA has not accepted the application of that descriptor to PFOA. Rather, that issue is presently under consideration. The background for that issue is set forth below.

US EPA began scrutinizing PFOA in approximately June, 2000. In January, 2005, US EPA released a draft risk assessment that preliminarily categorized PFOA as “suggestive” of carcinogenicity. This opinion was based on the view that human studies on the effects of exposure to PFOA do not support a conclusion that the chemical is carcinogenic, as well as uncertainties on the part of the Agency’s Office of Prevention, Pesticides and Toxic Substances (“OPPTS”) regarding differences between rats and humans, and consequent questions regarding the relevance of tumor data in rats in determining whether the chemical is carcinogenic in humans.

Soon after the 2005 draft risk assessment was released, US EPA established an independent panel of outside scientific experts under the supervision of the Agency’s Scientific Advisory Board (“SAB”) to peer-review the document. This panel, referred to herein as the “Panel,” was charged with ensuring that assumptions used in the Agency’s 2005 draft risk assessment were scientifically sound and could be used effectively to determine potential risks and to identify appropriate risk-management actions. The Panel deliberated on the questions charged to it during meetings on February 22 and 23, 2005. On July 6, 2005, the Panel met by telephone conference call to discuss the text of a draft report. Among the issues for discussion

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were the Panel's interpretation of studies conducted on mammary gland tissues, and which "descriptor" of hazard potential should be used to describe PFOA under the Agency's new 2005 Guidelines for Carcinogen Risk Assessment ("Cancer Guidelines").

Incidences of mammary tumors played an important part in the Panel's deliberation, and were critical in the eventual recommendation of a descriptor. Due to uncertainties, the Panel recommended that the Agency "consider new information that has been verified and peer-reviewed prior to use in their revision of the [2005] Draft Risk Assessment" and that an "independent, appropriately-designed histopathology review of . . . female mammary glands from the [1987] Sibinski study be conducted to re-analyze the resulting tumor incidence data."

As a result, an independent Pathology Working Group (PWG) was convened to reevaluate the mammary tissues collected from the 1987 Sibinski study. Experimental Pathology Laboratories, Inc. ("EPL") was selected to administer the PWG, which was chaired by EPL's president, Dr. Jerry F. Hardisty, Diplomate, A.V.C.P. Dr. Hardisty was responsible for selecting the other members, compiling the findings, and drafting the report for concurrence and signature.¹³ The other PWG panelists were Dr. Gabrielle Wilson, FRC Path., Dr. W. Ray Brown, Diplomate, A.C.V.P., and Dr. Ernest E. McConnell, Diplomate, A.V.C.P., D.A.B.T. The panelists all have extensive experience in the microscopic evaluation and interpretation of lesions observed as a result of chronic toxicity and bioassay studies conducted in rodents.

The PWG conducted its review in accordance with US EPA guidelines (US EPA PR Notice 94-5), and reexamined all mammary gland tissues microscopically, without identifying their treatment groups to the pathologists. Using the diagnostic criteria and nomenclature recommended by the Society of Toxicologic Pathologists, the PWG concluded that there were no statistically-significant increases in incidence of mammary tumors, of total benign neoplasms, or total malignant neoplasms, and no increase in tumor multiplicity. The PWG also concluded that the incidence of mammary gland neoplasms observed in the study was similar to the historical control incidence.

In May 2006, the SAB issued its report and noted a split among Panel members with regard to the hazard descriptor used to indicate the potential for carcinogenicity. To explain, this Panel was the first to apply the Agency's new Cancer Guidelines. Thus, as noted above, there was considerable discussion among the Panel members as to how the new descriptors articulated in the Guidelines should be interpreted and utilized. The SAB reported that one-quarter of the Panel agreed with EPA's January 2005 draft assessment, and the use of the descriptor "suggestive evidence of carcinogenicity, but not sufficient to assess human carcinogenic potential." Three-quarters of the Panel chose the hazard descriptor "likely to be carcinogenic." As a result, the SAB report strongly "urge[d] the Agency to strengthen its risk assessment by considering verified and peer-reviewed new information found to be relevant and critical to the

¹³ Dr. Hardisty is an expert in conducting pathology peer reviews. EPL is the largest independent pathology contract laboratory in the world, providing hazard identification services to governmental agencies and industry in support of human risk assessment programs. Dr. Hardisty advises both EPA and FDA on pathology issues.

assessment.” Thus, although the PWG was convened in response to the Panel’s recommendation, the results of the pathology review were not available in time for the Panel’s consideration. As a result, the recent conclusions of the Panel process do not incorporate these key findings regarding the mammary glands.

Obviously, it is essential that any determination of cancer risk include the most current information. Therefore, US EPA declined to reach any conclusions regarding the SAB Panel report. Referring to the new data and the SAB process on its website, US EPA states that “[s]ome of this new research may impact the Panel’s assessment of PFOA. For this reason, *it is premature to draw any conclusions on the potential risks, including cancer, from PFOA until all of this new testing is complete and the data are integrated into the risk assessment.*” (Emphasis added.)

US EPA Administrator Stephen Johnson similarly stated his concern about the Agency’s need to consider completely the most recent information. In his June 20, 2006 letter to the Panel co-chairs acknowledging receipt of the SAB report, Mr. Johnson stated that “[i]t has been nearly two years since the package of information that the Panel reviewed was compiled, and since that time, a considerable amount of research has been completed . . . or is presently underway.” The Administrator added that the Agency intends to “integrate this new toxicity testing and mechanistic data into the risk assessment as it becomes available.”

As it stands now, US EPA has not adopted the 2005 Draft Risk Assessment or its preliminary cancer classification. The Agency announced in a public PFOA Information Forum on June 8, 2006 that it will incorporate the most recent data into a new (third) draft of the risk assessment. Further, US EPA said that it intends to conduct another SAB review of this draft, presumably completing the risk assessment in the next twelve to twenty-four months in order to inform further regulatory action on PFOA after this most thorough assessment. At this same forum, the Agency also announced that “*EPA has no information linking current levels of PFOA in the blood of the general public to any adverse health effects in people.* Additional study is still needed to understand these persistent chemicals. While information is being developed, EPA is taking the prudent step of seeking to reduce possible sources now, to avoid potentially larger future problems.” (Emphasis added.)

There is no “new information” or “emerging public health issue” related to cancer to support an expedited review. As noted above, OEHHA has discretion to vary from the Prioritization Process. By its own terms, however, the Prioritization Process indicates when that discretion should be exercised. “The Director may abbreviate or otherwise modify the process. For example, the public or a [CIC] committee member may petition the Director to abbreviate the prioritization process to respond to *new information* or an *emerging public health issue*”¹⁴ Obviously, these terms refer to the matters that the Committee is authorized by Proposition 65 and its implementing regulations to address – reviewing test data and

¹⁴ OEHHA (2004) Process for Prioritizing Chemicals for Consideration under Proposition 65 by the “State’s Qualified Experts,” December 2004, p. 6.

establishing processes to determine whether chemical substances cause *cancer*.¹⁵ Thus, expedited review process should be reserved for chemicals where exceptional circumstances, such as a new study, that clearly shows a chemical causes *cancer*.

PFOA does not meet either of these criteria. There is no new information demonstrating that PFOA causes *cancer*. In fact, the two carcinogenicity bioassays of PFOA that are discussed in the petition were publicly available in 1987 and 2001, and have been the subject of continued governmental evaluation since that time. Any “new information” regarding the presence or persistence of the chemical is not related to carcinogenicity, which Proposition 65 is intended to address. Similarly, the absence of any data to support a conclusion that exposure to PFOA causes cancer prevents any conclusion that exposure presents an “emerging public health issue” that Proposition 65 is intended to address.

Exposure to PFOA is being addressed. In opposing an expedited review of PFOA, DuPont does not ignore or discount the evidence showing that PFOA has been shown to be present in human blood, and that the presence of the material at low levels in the general population cannot be fully explained. To the contrary, US EPA, as well as DuPont and other companies that make and use PFOA, are investigating diligently to determine the sources of exposure and to reduce exposure. Moreover, as noted above, scientific evidence gathered thus far does not show any adverse effects in humans attributable to PFOA.

US EPA has confirmed this on several occasions within the past year. Last December, Susan Hazen, Acting Administrator of US EPA’s OPPTS, stated: “The agency has information based on animal and toxic effects in animals, [but] we have ***no information at this point that would lead us to believe there is a significant human health impact.***” On January 25, 2006, US EPA Administrator Johnson announced that “[a]lthough our risk assessment activities are not complete and new data may change the current picture, to date ***EPA is not aware of any studies specifically relating current levels of PFOA exposure to human health effects.***” In its June 8, 2006 PFOA Information Forum, US EPA reiterated: “***EPA has no information linking current levels of PFOA in the blood of the general public to any adverse health effects in people.***” (Emphasis added in all quotations.)

Because of the presence and persistence of PFOA in humans and in the environment, regulatory agencies, most notably US EPA and US FDA, are addressing these issues vigorously, despite the lack of any adverse health effects in people. The petition noted that US EPA has

¹⁵ The “Powers and Duties” of the Committee, “as an advisory body to the Governor and [OEHHHA],” are: (1) to “render an opinion . . . whether specific chemicals may have been clearly shown, through scientifically valid testing according to generally accepted principles, to cause *cancer*,” (2) to “identify bodies which are considered to be authoritative and which have .formally identified as causing *cancer*,” (3) to “identify specific chemicals that are required by state or federal law to have been tested for potential to cause *cancer* . . . ;” (4) to “review or propose standards and procedures for determining *carcinogenicity* of chemicals,” and (5) “upon request by the lead agency,” and to “review or propose standards, procedures and definitions related to the implementation, administration or interpretation of the Act in support of the duties of” the Governor to publish the list of chemicals required under the statute. Cal. Code Regs., tit. 22, § 12305(a)(1)-(5).

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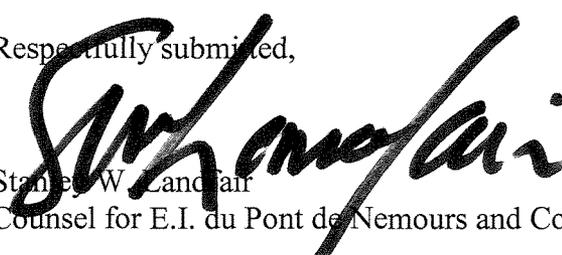
asked companies to agree voluntarily to reduce their PFOA releases. To be more precise, US EPA took aggressive steps to reduce possible sources of human exposure through a voluntary program announced in January 2006. By March, 2006, DuPont and the other seven fluoropolymer and telomer manufacturers agreed to participate in this comprehensive program to reduce exposure to PFOA. For example, all of these companies have committed to achieve, no later than 2010, a 95% reduction, measured from a year 2000 baseline, in both facility emissions to all environmental media and product content levels of PFOA, precursor chemicals that can break down to PFOA, and related higher homologue chemicals.

In short, current levels of exposure to PFOA have not been linked to any adverse health effects in the general population. Nevertheless, an aggressive program is in place to reduce exposure to PFOA in response to issues regarding the presence and persistence of PFOA in humans and the environment. Public health will not be compromised if the Committee chooses to deny an expedited review of PFOA as a chemical "known to the state to cause cancer."

CONCLUSION

For all of the reasons discussed above, we believe it is unnecessary and inappropriate to make PFOA the subject of an expedited review. A chemical should not be designated for an expedited review if it does not even meet the listing criteria. The scientific evidence on PFOA does not support the proposal, because it does not demonstrate that PFOA has been clearly shown to be a carcinogen. Finally, a thorough review of all aspects of exposure to PFOA already has been initiated by US EPA (an "authoritative body" for purposes of Proposition 65) and will address all of the carcinogenicity questions raised by the petition without diverting OEHHA or Committee resources from other compelling issues. All interested parties – including the regulated community, the public, and OEHHA – would benefit from the completion of the US EPA analysis, before OEHHA decides whether, much less how quickly, to proceed.

Respectfully submitted,


Stanley W. Landfair
Counsel for E.I. du Pont de Nemours and Company

cc: Joan Denton, Ph.D., Director, OEHHA
George Alexeeff, Ph.D., Deputy Director, OEHHA
Michael Shepard, Acting Chief Counsel