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October 25, 2002

TELECOPY AND REGULAR U.S. MAIL

Perry D. McDaniel, Esq.
West Virginia Department of
Environmental Protection
Office of Legal Services
1356 Hansford Street
Charleston, WV 25301

Re: Response To WVDEP's September 13, 2002, Letter Regarding CAT Team Activities Involving C-8

Dear Perry:

This letter responds to the letter from your office dated September 13, 2002. As you are aware, we did not receive a copy of that letter until October 10, 2002 - the day we reviewed files made available to us at WVDEP's offices and noticed copies of the letter in those files. Based on our review of WVDEP's files on October 10, 2002, which included several earlier drafts of the September 13, 2002, letter in the files of Dr. Dee Ann Staats, we understand that Dr. Staats drafted the letter for your signature to respond to questions raised in our letter dated August 19, 2002. As explained below, WVDEP's response confirms that WVDEP inexplicably deviated from the express requirements of the Consent Order entered between the State and E.I. duPont de Nemours and Company (the "Consent Order") that set up the process that the C-8 Assessment of Toxicity Team ("CAT Team") was supposed to follow in its assessment of C-8 toxicity issues.

The Consent Order clearly and expressly provided for the CAT Team to perform its activities in three basic "Phases" as set forth on Pages C-4-C-9 of the Consent Order. Phase I involved public meetings to discuss the CAT Team process. During Phase II, WVDEP's consultant, TERA, was to assume primary responsibility for each of the following tasks:

1. Review of the toxicological information regarding C-8 provided by WVDEP and development of a proposal for selecting provisional reference doses, screening levels, and a risk assessment for C-8;

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2. Consultation with the other toxicologists on the CAT Team, as coordinated by Dr. Staats, regarding TERA's proposed approach for developing provisional reference doses, screening levels, and risk assessment activities for C-8;

3. Development of provisional reference doses for C-8 for the oral, inhalation, and dermal (if possible) routes of exposure;

4. Calculation of screening levels for C-8 in water, soil, and air based on the risk factors estimated by TERA;

5. Performance of one general risk assessment involving comparison of exposure concentrations to screening levels (using data obtained from other ongoing C-8 efforts such as WVDEP's and DuPont's air modeling) for the Letart Landfill, Dry Run Landfill, Local Landfill, Washington Works Plant, and Lubeck Public Service District water supply, that focuses on current risk to human health, including workers and residents, with such risk assessment to include:

   A. Identification of reasonably anticipated land use, surface water and groundwater use;

   B. Identification of receptors;

   C. Identification of exposure pathways;

   D. Identification of exposure concentrations; and

   E. Comparison of exposure concentrations to appropriate screening levels.

6. Review of available information to determine whether sufficient studies have been performed and data have been collected to develop C-8 screening criteria for protection of ecological health, particularly aquatic health; and

7. Preparation of a draft and final document that discusses the results of TERA's efforts and summarizes the data utilized from other C-8 efforts.

(Consent Order, at C-4 through C-5.) Under the Consent Order, Phase III of the CAT Team's work is to involve a "second public meeting" designed to "present to the citizenry the results of the efforts of the GIST and CAT Teams including C-8 concentrations in groundwater from residential wells and public wells the screening levels and the general risk assessment. Air

modeling results of the efforts of WVDEP and DuPont will be discussed also. The WVDEP will address any further actions that may be necessary." (*Id.* at C-5.)

It is now clear from the Final CAT Team Report and the September 13, 2002, letter from your office that tasks to be accomplished by TERA as part of the "Phase II" activities of the CAT Team were not performed by TERA, as required under the Consent Order. More specifically, WVDEP confirmed in the September 13, 2002, letter that TERA did not perform the general risk assessment it was supposed to perform under the Consent Order, and that the one page "Comparison of Screening Levels to Site-Related Data" that now appears as Section 3 of the Final CAT Team Report was, in fact, drafted by WVDEP and not by any representatives of TERA. In fact, it has now been confirmed that "TERA did not have the information necessary to draft" the information set out in Section 3 of the Report. (WVDEP's September 13, 2002, letter at 1.) No explanation has been provided as to why TERA was not provided the information necessary to complete the actual risk assessment activity it was supposed to perform as part of the Phase II CAT Team work, or why WVDEP unilaterally took over the task of completing that entire Section of the Final CAT Team Report. In addition, no explanation has been provided as to why the risk assessment information prepared solely by WVDEP inexplicably omits the information required under the Consent Order relating to: identification of reasonably anticipated land use, surface water, and groundwater use; identification of receptors; identification of exposure pathways; identification of all exposure concentrations; and actual exposure concentrations at the three DuPont landfills specified in the Consent Order. WVDEP's one page "risk assessment" also says nothing about the cumulative risk posed to those living in the communities surrounding DuPont's Washington Works facility who are being exposed to C-8 in both air and water, and possibly soils. It is not clear whether WVDEP now even intends to collect any soil data.

In addition, despite the express requirements of the Consent Order that TERA should have been provided data obtained from the ongoing DuPont and WVDEP air modeling of C-8 emissions, (*see* Consent Order, at C-5), WVDEP's September 13, 2002, letter confirms that WVDEP did not share that information with the other members of the CAT Team, including TERA. (*See* WVDEP September 13, 2002, letter, at 3 (WVDEP confirms that, although Dr. Staats and WVDEP were aware of the level of DuPont's C-8 air emissions, "the other members of the [CAT Team] toxicology panel were not provided with information on the air modeling.").)

The lack of any involvement by TERA or any of the other members of the CAT Team in any of the actual risk assessment activities required under the Consent Order beyond selection of screening levels is further confirmed by the lack of any reference to those additional risk assessment activities or evaluations in any of the meeting notes from the CAT Team members or in the final CAT Team Report itself, except for the one page "Comparison of Screening Levels to Site Related Data" that we now understand was prepared solely by WVDEP. It is noted that the

"final" CAT Team Report also inexplicably omits any information with respect to the development of screening criteria for protection of ecological health, as expressly required under the Consent Order, other than a reference at the beginning of the CAT Team Report that such information may be forthcoming in "an addendum to [the final CAT Team] Report . . . to be released in fall 2002." It is not clear why WVDEP chose to ignore the express requirements of the Consent Order to include the results of all of the risk assessment activities required under the Consent Order, along with the screening criteria for protection of ecological health, in one final CAT Team Report before the report was released to the public. It is also unclear as to why WVDEP apparently chose not to complete "Phase III" of the CAT Team work, which requires a second public meeting to present to the citizenry the results of the "general risk assessment" activities required under the Consent Order, and "air modeling results of the efforts of WVDEP and DuPont." (*See* Consent Order, at C-5.)

Of particular concern to our clients is WVDEP's failure to explain why WVDEP has said nothing publically with respect to whether DuPont is exceeding the screening levels selected by the CAT Team for C-8 in air. Although WVDEP wasted no time releasing to the public assurances that DuPont was not exceeding the CAT Team's screening level for C-8 in water within only a matter of days after the CAT Team selected that screening level, it has now been several months since the CAT Team selected its screening levels for C-8 in air but there has been no comment from WVDEP with respect to whether WVDEP's and DuPont's air emissions data indicate that DuPont has exceeded the appropriate air levels in either West Virginia or Ohio. The lack of comment from WVDEP on this issue is puzzling, given that the Year 2000 air modeling data contained within WVDEP's own public files confirms and has confirmed for several months that those air emissions exceed the lower end of the CAT Team's air screening level range (from 0.3 ug/m3 to 10 ug/m3) for C-8 in the local community, and indisputably exceed the "median value" air screening level of 1 ug/m3 at the Washington Works property line.

Although the Consent Order expressly provides WVDEP the authority to promptly issue a notice to DuPont that "DuPont's operations have resulted in C-8 exposures above the screening levels," which would trigger DuPont's obligation to prepare a plan to reduce its C-8 air emissions, we are unaware of WVDEP having taken any steps to send such a notice to DuPont. Instead, we understand that, rather than relying upon the year 2000 air modeling data generated by DuPont and WVDEP to determine DuPont's compliance with the C-8 air screening levels, the generation of which was required under the terms of the Consent Order, WVDEP apparently is now providing DuPont the opportunity to try to generate new, different air emissions data. This point was confirmed on page 46 of the final CAT Team Report where WVDEP notes that "the air modeling effort continues and is currently focusing on determining the results of air emissions reduction efforts by DuPont required in the Consent Order . . . by the end of 2003." (Final CAT Team Report, at 46.)

Consequently, we understand that, rather than making a determination as to whether DuPont is exceeding the CAT Team's screening level for C-8 in air using the Year 2000 modeling data required under the Consent Order that would confirm such exceedances, WVDEP is holding off making any such determination until after DuPont has been given a chance to try to generate new, and different air emissions data. WVDEP should explain promptly whether DuPont has exceeded the air screening range for C-8 established by the CAT Team in either West Virginia or Ohio using the Year 2000 modeling data already generated and possessed by both DuPont and WVDEP, or at least explain how long DuPont will be given to try to generate new, different data and why.

The comment in your September 13, 2002 letter that DEP has not received on behalf of our clients "any substantive comments about the process used by the agency in the evaluation of the scientific studies" by the CAT Team is disingenuous, at best. Numerous letters have been submitted to WVDEP setting out specific objections to and concerns with the methodology and results of the CAT Team process. In particular, we forwarded to your agency, along with each of the members of the CAT Team, including Dr. Staats, a letter dated June 10, 2002, providing very detailed substantive comments relating to our clients' concerns with respect to the process used by WVDEP and the CAT Team in evaluating C-8, and the CAT Team's evaluation of the scientific studies relating to C-8. That letter identified numerous problems inherent in the methodology used by CAT Team and the CAT Team's results. As pointed out in that letter, these problems resulted in the generation of C-8 screening levels over 150 times higher than the levels that should have been selected after reviewing the relevant studies according to the appropriate agency guidance and methodology. This point is confirmed by WVDEP's own C-8 screening level calculations from the summer of 2001, which were based on an evaluation of all of the exact same studies reviewed by the CAT Team, with the exception of a later two-generation rat reproduction study that was not finalized until the Spring of 2002.. (*See* Attachment A (copy of WVDEP's C-8 screening level calculations from the summer of 2001, which were retrieved from the computer files that had been deleted by Dr. Staats).) As confirmed in the "Revised Draft Hazard Assessment of PFOA and Its Salts" released to the public on October 17, 2002, by the United States Environmental Protection Agency ("USEPA's C-8 Report"), that later two-generation rat reproduction study confirmed the existence of certain adverse effects that require an even more conservative approach to protecting public health – not less conservative. (*See* USEPA's C-8 Report, at 5-6, 64-73.)

Now that the Final CAT Team Report has been released, we note that WVDEP has failed to explain the extent to which its screening level calculations are based on exposures in adults versus children. As confirmed in one of the earlier draft screening level charts sent by TERA only to Dr. Staats, the screening level numbers can vary dramatically depending on whether the exposure group is assumed to be adults or children. (*See* Attachment B (also a document retrieved from computer files that had been deleted by Dr. Staats).) Screening levels for children

would be less than half the screening levels derived for adults, according to TERA's calculations. (*See id.*) Although we presume, for example, that the 150 ppb water screening level is based on adult exposures, there is no explanation as to how the lower screening levels for children have been handled in selecting the final screening levels set forth in the CAT Team Report.

WVDEP's September 13, 2002, letter also now confirms that WVDEP derived its screening levels for C-8 through a process that was designed to identify those screening levels that could be agreed to by a "majority" of a committee that included DuPont. Thus, rather than follow standard agency procedures for selecting the screening levels that are the most protective of human health justifiable under the existing toxicological data, the CAT Team's screening levels represent only those levels that could be agreed to through a majority voting process for a group that included DuPont. The practical effect of such a process is highlighted in WVDEP's description of the manner in which the screening level for C-8 in air was selected. Although it is undisputed that members of the CAT Team believed that a screening level for C-8 in air of 0.3 ug/m3 was justifiable under the existing toxicological data for C-8, WVDEP has confirmed that that particular screening level was not selected, because some of the other members of the CAT Team preferred to rely on toxicological data that could support an air screening level for C-8 as high as 10 ug/m3. Yet, rather than select the more conservative C-8 screening level for air of 0.3 mg/m3 in order to be the most protective of public heath, the CAT Team selected a "median" value of 1 ug/m3 as its final screening level for C-8 in air as a compromise. There is, therefore, no doubt that the CAT Team did not select the most conservative screening level for C-8 in air that was supportable by the available toxicological data.

Our clients look forward to prompt confirmation from WVDEP as to when a CAT Team report will be released containing the results of all of the ecological screening levels and risk assessment activities that are required under the Consent Order. Once all of that information is finalized and available, we would like to proceed with completing the deposition of Dr. Staats. We also look forward to prompt confirmation from WVDEP as to what actions will be taken by WVDEP to immediately enforce DuPont's obligations under the Consent Order to ensure that its C-8 emissions do not exceed applicable C-8 screening levels in the surrounding West Virginia and Ohio communities. Thank you.

Very truly yours,

Robert A. Bilott

RAB/mdm
Attachments

cc: R. Edison Hill, Esq. (w/ attachments)
Larry A. Winter, Esq. (w/ attachments)
Heather Heiskell Jones, Esq. (w/ attachments)
John R. McGhee, Jr., Esq. (w/ attachments)
Richard A. Hayhurst, Esq. (w/ attachments)
Greg Smith, Esq. (Ohio EPA, Legal) (w/ attachments)
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Janet Sharke, Esq. (USEPA, Region 3) (w/ attachments)
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DRAFT: DEVELOPMENT OF PROVISIONAL ORAL AND INHALATION REFERENCE DOSES AND PRELIMINARY SCREENING LEVELS FOR AMMONIUM PERFLUOROOCTANOATE

A Provisional Oral Reference Dose (PRfDo) of 3 x 10-5E or 0.00003 mg/kg/day for Ammonium Perfluorooctanoate (C8) and a Provisional Inhalation Reference Concentration (PRfCi) of 0.02 $\mu g/m^3$ have been developed by WVDEP. Subsequently, a Preliminary Screening Level (PSLw) for groundwater of 1 ppb was calculated based on this PRfDo and on the model for the water ingestion exposure pathway with default parameters commonly used by USEPA and WVDEP. The PRfCi of 0.02 $\mu g/m^3$ would serve as the Preliminary Screening Level (PSLi) for air. The scientific rationale used to develop the PRfCi and the PRfDo, and to calculate the PSLw is described below.

Development of the Provisional Oral Reference Dose:

Ammonium perfluorooctanoate (C8 or APFO) is a potent synthetic surfactant. In biologic media, the ammonium quickly dissociates. C8, as perfluorooctanoic acid (PFOA), comprises 93 – 98% of FC-143 FLUORAD Brand Fluorochemical Surfactant. Toxicity studies have been conducted on APFO, PFOA, and FC-143. The USEPA has conducted a literature search and review of toxicological data regarding PFOA; their findings are summarized in the Draft Hazard Assessment of PFOA (in preparation). This document, including supporting references, and information provided to WVDEP by DuPont, and the data contained on 7 compact discs as part of the TSCA submittal by 3M were the major sources of toxicological information utilized in the development of the reference doses.

The first step in the development of a reference dose is to identify appropriate exposure studies. Chronic studies utilizing the appropriate route of exposure and animal model are most highly desirable. However if such studies are not available, then subchronic studies utilizing other routes of exposure may be employed for reference dose development by including additional uncertainty factors. Ideally a NOAEL, No Observable Adverse Effect Level, was determined in the study; however, if a NOAEL was not determined, then a LOAEL, Lowest Observable Adverse Effect Level, may be employed in reference dose development by including an additional uncertaintly factor.

The only chronic oral exposure study available was conducted in male and female rats fed FC-143 over a two-year period (3M, 1987). A LOAEL of 300 ppm (14.2 mg/kg/day for male, and 16.1 mg/kg/day female) was determined in rats based on decreased body weight gains; increased liver and kidney weight; and toxicity in the hematological and hepatic systems. However, a LOAEL for female rates of 30 ppm (1.6 mg/kg/day) was determined based on incidence of ataxia and reversible ovarian tubular hyperplasia.

To date, four 90-day subchronic oral exposure animal studies have been conducted - two in monkeys and two in rats. A LOAEL of 30 ppm (1.72 mg/kg/day) was determined by Goldenthal (1978a) in male rats exposed to a diet including FC-143 based

on increased liver weight; increased blood glucose; and decreased red cell counts. Palazzolo (1993) found a NOAEL of 30 ppm (1.44 mg/kg/day) and a LOAEL of 100 ppm (4.97 mg/kd/day) based on decreased body weight and body weight gain, and on increased absolute and relative liver weights with hepatocellular hypertrophy in male rats exposed to PFOA in the diet for 13 weeks.

Goldenthal (1978b) determined an oral NOAEL of 3 mg/kg/day in rhesus monkeys. However, this dose group occasionally had soft stools or moderate to marked diarrhea, and frothy emesis. Also, there were trends toward increased glucose levels, and decreased alkaline phosphatase levels in this dose group.

Butenhoff, et al., (2001) found an oral LOEL of 3 mg/kg/day based on increased liver weight in cynomolgus monkeys, which occurred at serum concentrations that overlapped those observed in some workers with high exposure; therefore the liver enlargement was considered to be a significant effect by the authors. A NOEL was not determined in this study. Because the monkey is most physiologically similar to humans, as evidenced by the long half-life of C8 in humans (1 – 3.5 years) and monkeys. This LOEL was utilized to estimate an the PRfDo as decribed below:

Calculation of the Oral Provisional Reference Dose:

$$PRfDo = \frac{LOEL}{(UFH)\,(UFA)\,(UFS)\,(UFL)\,(MF)}$$

where:
PRfDo = Provisional Oral Reference Dose (mg/kg/day);
LOEL = Lowest Observable Effect Level (mg/kg/day) = 3;
UF = Uncertainty Factors (unitless);
H = intrahuman variability accounts for variation in sensitivity among the human population =10;
A = animal to human extrapolation = 10;
S = extrapolation from subchronic exposure to chronic exposure = 10;
L = extrapolation from a LOEL to a NOEL = 10;
D = insufficiency in the toxicological database = 3;
MF = Modifying Factor (unitless) = 3
A modifying factor of 3 was used because of the following characteristics of C8:

- Long half-life in humans (approximately 1 – 3.5 years);
- Potential for bioaccumulation;
- Potential for biopersistence;
- Unusual physical properties such as solubility and partition coefficient.

Therefore, the PRfDo equals 3 x 10E-5.

Calculation of the Preliminary Screening Level for C8 in Water:

The PSL of 1 µg/L or ppb was calculated using a Hazard Quotient of 1 and the following equation and default parameters:

$$GW = \frac{(PRfDo)(BWa)(CF)}{(IRWa)}$$

where:

GW = concentration in Groundwater (µg/L);
PRfDo = Provisional Oral Reference Dose (mg/kg/day) = 3 x 10E-5;
BWa = adult body weight (kg) = 70;
CF = conversion factor (from mg to µg) = 1000;
IRWa = Ingestion Rate of Water for an adult (L/day) =2.

Development of Provision Inhalation Reference Concentration:

No monkey inhalation exposure studies have been conducted for PFOA. However, two two-week (6 hr/day; five days/week) inhalation exposure studies were conducted in rats by DuPont (1994). In the first study, a LOAEL of 11 mg/m^3 was found based on decreased body weight and hepatic injury. In the second study, a NOAEL of 1 mg/m^3 was determined. This NOAEL agreed with a NOAEL of 1 mg/m3 found by Staples et al. (1984) in female rats during a developmental toxicity study of PFOA. Inhalation reference doses were calculated for the NOAEL and the LOAEL as described below.

Based on the LOAEL:

Conversion from intermittent exposure to continuous exposure:

LOAEL = E x D (h/24h) x W (days/7 days)

Where:
LOAEL = 11 mg/m^3;
E = exposure level in mg/m^3;
D = hours of exposure (6);
W = days of exposure (5);

Thus the continuous exposure LOAELc = 1.95 mg/m^3;

$$PRfCi = \frac{LOAELc}{(UFH)(UFA)(UFS)(UFL)(MF)}$$

where:
PRfCi = Provisional Inhalation Reference Concentration (mg/m^3);
LOAELc = Lowest Observable Effect Level (mg/m^3) = 11;
UF = Uncertainty Factors (unitless);
H = intrahuman variability accounts for variation in sensitivity among the human population =10;
A = animal to human extrapolation = 10;
S = extrapolation from subchronic exposure to chronic exposure = 10;
L = extrapolation from a LOEL to a NOEL = 10;
D = insufficiency in the toxicological database = 3;
MF = Modifying Factor (unitless) = 3

A modifying factor of 3 was used because of the following characteristics of C8:

- Long half-life in humans (approximately 1 – 3.5 years);
- Potential for bioaccumulation;
- Potential for biopersistence;
- Unusual physical properties such as solubility and partition coefficient.

Therefore, the PRfCi equals $0.022 \mu g/m^3$ based on the LOAEL of 11 mg/m^3.

Based on the NOAEL:

Conversion from intermittent exposure to continuous exposure:

NOAEL = E x D (h/24h) x W (days/7 days)

Where:
NOAEL = 1 mg/m^3;
E = exposure level in mg/m^3;
D = hours of exposure (6);
W = days of exposure (5);

Thus the continuous exposure NOAELc = 0.18 mg/m^3;

$$PRfCi = \frac{NOAELc}{(UFH)\,(UFA)\,(UFS)\,(UFL)\,(MF)}$$

where:
PRfCi = Provisional Inhalation Reference Concentration (mg/m^3);
NOAELc = No Observable Effect Level (mg/m^3) = 1;
UF = Uncertainty Factors (unitless);
H = intrahuman variability accounts for variation in sensitivity among the human population =10;
A = animal to human extrapolation = 10;
S = extrapolation from subchronic exposure to chronic exposure = 10;
D = insufficiency in the toxicological database = 3;

MF = Modifying Factor (unitless) = 3

A modifying factor of 3 was used because of the following characteristics of C8:

- Long half-life in humans (approximately 1 – 3.5 years);
- Potential for bioaccumulation;
- Potential for biopersistence;
- Unusual physical properties such as solubility and partition coefficient.

Therefore, the PRfCi equals 0.02$\mu g/m^3$ based on the NOAEL of 1 mg/m^3. The PRfCi estimated using the NOAEL and the LOAEL are approximately equal.

References:

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Noncancer Screening Level Values for C8									
Reference	Critical Effect Level	Composite UF	RfD/ RfC	Water Adult (ug/L)	Water Child (ug/L)	Soil Adult (mg/kg)	Soil Child (mg/kg)	Air (ug/m^3)	
Oral Studies									
Palazzolo et al. (1993)	0.47 (NOAEL in males)	100	0.005	183	78	3650	391		
York et al. (2002)	1 (LOAEL in males)	300	0.003	110	47	2190	235		
	0.42 (BMDL in males)	100	0.004	146	63	2920	313		
3M (1983)	1.6 (LOAEL in females)	1000	0.002	73	31	1460	156		
	0.73 (BMDL in males)	100	0.007	256	110	5110	547		
Thomford et al. (2001)	3 (NOAEL in males)	1000	0.003	110	47	2190	235		
Inhalation Studies									
Kennedy et al. (1986)a	0.6 (NOAEL - (HEC_R in males)	3000	0.0002					.21	
	0.04 (NOAEL - HEC_T in males)	3000	0.00001					.01	
Dermal Studies									

EXHIBIT 2

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Kennedy et al. (1985)a	LOAEL (LOAEL in males)	3000	0.001						

a. These studies are not adequate for derivation of an IRIS quality RfD/RfC of even low confidence. The values shown would represent only a provisional value, and not recommended for use as the basis of a screening level. Derivation of the RfC or RfD via route-to-route extrapolation is not supported by the available toxicokinetic data.

Calculations for Screening Levels:

Default Exposure Parameters for calculations:

Target Hazard Quotient (THQ):	1
Body Weight (BW):	70 kg adult; 15 kg child
Averaging Time (AT):	10950 days (ED x 365)
Exposure Frequency(EF):	350 days/year
Exposure Duration (ED):	30 years
Water Ingestion Rate (IRW):	2 L/day adult; 1 L/day child
Soil Ingestion Rate (IRS):	100 mg/day adult; 200 mg/day child

General Assumptions:

- C8's low vapor pressure and Henry's Law constant suggest that volatilization of C8 from water and soil is unlikely; therefore parameters estimating volatilization were not included in the screening level equations.
- Inadequate data exist to develop a dermal toxicity value either from dermal data or by route-to-route extrapolation; therefore parameters estimating contribution from dermal exposure to water or soil were not included in the screening level equations.

Water:

$$RBC\ (ug/L) = RfD \times \frac{THQ * BW * AT * 1000\ ug/mg}{EF * ED * IRW}$$

For adult exposure $RfD \times \frac{1 * 70 * 10950 * 1000}{350 * 30 * 2}$

= **RfD x 36500**

For child exposure $RfD \times \frac{1 * 15 * 10950 * 1000}{350 * 30 * 1}$

= **RfD x 15643**

Soil:

RBC (mg/kg) $= RfD \times \frac{THQ * BW * AT}{EF * ED * IRS * 10^{-6}}$ mg/kg

For adult exposure $= RfD \times \frac{1 * 70 * 10950}{350 * 30 * 100 * 10^{-6}}$

= **RfD x 730,000**

For child exposure $= RfD \times \frac{1 * 15 * 10950}{350 * 30 * 200 * 10^{-6}}$

= **RfD x 78,214**

Air:

Note, both Region 3 and Region 9 Screening level guidance indicate that an RfDi, in units of mg/kg-day, is to be used in the air screening level calculation. The RfDi is obtained from the RfC by multiplying by the default breathing rate and dividing by the default

body weight. However, the screening level calculations for air then multiply by the default body weight and divide by the default breathing rate in order to return to air concentration units of ug/m^3. Because the body weights and breathing rates "cancel out", there is no difference in the air concentration based on adult vs. child exposure. Therefore, the screening level concentration for air was modified to eliminate the redundant step.

$$\text{RBC (ug/m}^3\text{)} = \text{RfC} \times \frac{\text{THQ} * \text{AT} * 1000\ \text{ug/mg}}{\text{EF} * \text{ED}}$$

$$= \text{RfC} \times \frac{1 * 10950 * 1000}{350 * 30}$$

$$= \text{RfC} \times 1043$$