AR226_1164

OHIO ENVIRONMENTAL PROTECTION AGENCY MEETING
COLUMBUS, OHIO
July 24,2002

LITTLE HOCKING WATER COMMENTS

By letter dated February 21,2002, Mr. Kevin Hopper, environmental counsel for the Little Hocking Water Association, requested that the ***Ohio Environmental Protection Agency*** enter into a consent order with DuPont for the purpose of establishing the nature and scope of the contamination of the "waters of the State" by C-8 impacted aquifers in Ohio.

The people in this room today represent some of the Ohio water systems that we know C-8 has infiltrated. Collectively, water systems with C-8 contamination supply drinking water to about 37,000 Ohio citizens.

We are here today to request, on behalf of all the water systems and citizens affected by the C-8 contamination, that the Ohio Environmental Protection Agency enter into its own consent order with DuPont. We have a duty to protect our water supplies and our customers, but do we protect them from the harmful effects of C-8 by relying on the 150 ppb number of the West Virginia CAT Team or on the 0.3 ppb number advocated by the class action attorneys? A review of the evidence indicates that an independent review and analysis of the whole C-8 problem is required for the following reasons:

1. **The "Safe Level" Standards for C-8 Keep Escalating**

   The "safe level" standard for C-8 has been a rather fluid number that periodically increases exponentially. Initially, the "safe level" was said to be 1 ppb, which was DuPont's internal Community Exposure Guideline. This number eventually changed after detections of C-8 in the Little Hocking Water Association wells far exceeded it. Later, the U.S. EPA Consent Order with DuPont cited 14 ppb as the interim "action level" for C-8. The Little Hocking Water Association has a monitoring well which has C-8 levels that are more than twice 14 ppb. Now, based on the announced CAT Team findings, the "safe level" is 150 ppb.

   3M and DuPont have probably had more experience with C-8 than anyone in the world. They have manufactured and used it for more than 50 years. With all the knowledge that DuPont had gained from its own lab studies and those of 3M, DuPont set an internal value of 1.0 ppb for their Community Exposure Guideline. This guideline has been in place for more than a decade. Was DuPont realty wrong by a factor of 150?

2. Validity of the CAT Team's "Screening Level" of 150 ppb is Highly Questionable

a. The "screening level" is calculated from a reference dosage. The calculation of the reference dosage is greatly affected by five 'uncertainty factors'. Determination of these factors appears to be quite arbitrary. For the six studies considered, one-half of the 'uncertainty factors' assigned by the CAT Team were the least conservative values that could have been assigned. Therefore, they are also the least protective of the public health They even did this for a monkey study in which a couple of the monkeys apparently died inexplicably. (Refer to Attachment A)

b. The reference dosage used by the CAT team in the calculation of their 150 ppb number is only twice the referenced dosage cited in the ENVIRON report which was the source of the 14 ppb interim level of C-8. With a reference dosage of only twice the ENVIRON dosage, the CAT Team determined the "screening level" of 150 ppb, which is more than ten times the ENVIRON level of 14 ppb. (Refer to Attachment A)

c. In the ENVIRON Report a USEPA default value of 20% was used in the calculation of the 14 ppb to account for exposure of persons to C-8 by means other than drinking water. It appears that the CAT Team in their calculation did not utilize this 20% value even though we know that DuPont also emits C-8 into the air from its stacks in addition to discharging it to the Ohio River. This fact is evidenced by the presence of C-8 in groundwater sources upstream and far inland from the point that DuPont discharges to the Ohio River. The use of this value alone would reduce the CAT Team "screening level" to 30 ppb. (Refer to Attachment B)

d. According to the Conclusions of the ENVIRON Report, the best available and most relevant data for deriving a drinking water level for C-8 are those from the monkey study. The CAT Team used data from a rat study to derive their reference dosage. (Refer to Attachment B)

e. The CAT Team makes the assumption that C-8 it is not a carcinogen. The USEPA Region 9 formula used by the CAT Team to calculate the screening level" for C-8 was for non-carcinogenic contaminants in water. The recent USEPA 'Draft Hazard Assessment of Perfluorooctanoic Acid and its Salts" states, "As the mechanisms of carcinogenic action of APFO have not been fully elucidated, it is assumed that the tumors induced in rats are relevant to humans." (Refer to Attachment C)

The American Conference of Governmental Industrial Hygienists (ACGIH) has classified C-8 in Group A3. The Group A3 classification means that C-8 is a confirmed animal carcinogen with unknown relevance to humans. C-8 shares this Group A3 classification with currently regulated chemicals such as aldrin, chlordane, DDT, Di (2-ethylhexyl) phthalate, para-Dichlorobenzene, dichloromethane, ethylene dibromide, heptachlor, heptachlor epoxide, hexachlorobenzene, and lindane.

Therefore, in light of the preceding information how can the CAT Team assume that C-8 is a non-carcinogen?

3. C-8 has Similar Characteristics to Perfluorooctane Sulfonate (PFOS)

a. PFOS is a chemical, manufactured by 3M, that was used in Scotchgard coatings. In May 2000, 3M, which also manufactured C-8, announced that it was phasing out the production of PFOS. According to a 3M news release, " ...our decision was based on principles of responsible environmental management." The decision followed negotiations between 3M and the USEPA. According to an USEPA document, PFOS appears to combine Persistence, Bioaccumulation, and Toxicity properties to an extraordinary degree. (Refer to Attachment D)

b. 3M was the primary supplier of C-8 to DuPont. 3M has also stopped marketing C-8. Therefore DuPont is producing C-8 at one of its plants in North Carolina.

c. The Risk Assessment Division of the EPA Office of Pollution Prevention and Toxics recently released its "Draft Hazard Assessment Of Perfluorooctanoic Acid (PFOA) And Its Salts". This document states, "Based on the existing data, PFOA may present some concerns similar to those raised by perfluorooctyl sulfonates (PFOS). Like PFOS, it is persistent in the environment and does not appear to degrade. PFOA also appears to have a half-life in humans of between 1 and 3.5 years, indicating that it may bioaccumulate in humans in the same manner as PFOS does, remaining in enterohepatic circulation." (Refer to Attachment E)

Therefore, C-8 definitely has similar characteristics to PFOS, which is a known health hazard.

4. The terms of the West Virginia Consent Order establishing the procedure to determine the Provisional Reference Doses and Screening Levels have been disregarded. These violations call into question the validity both of the process employed and the results obtained.

a. Under the provisions of the West Virginia Consent Order a non-profit scientific organization, Toxicology Excellence for Risk Assessment (TERA) had the primary responsibility for the development of Provisional Reference Doses for C-8 and for then using them as a basis to develop human health risk-based Screening Levels for C-8 in air, water, and soil. (Refer to Attachment F - Consent Order, pp. C-4 and C-8)

After review of WVDEP provided material, TERA was to consult with the toxicologists on the CAT Team, as coordinated by Dr. Staats, regarding its proposed approach for this project. Following the consultation, the Order directed TERA to develop the Provisional Reference Doses and calculate Screening Levels. (Refer to Attachment F - Consent Order, pp. C-4, C-5, C-8)

b. It is apparent from deposition testimony given by Dr. Staats that at the May 6th and 7th meeting of the CAT team and TERA in Cincinnati that the CAT team, coordinated by Dr. Staats, usurped the responsibility of TERA and developed its own Provisional Reference Dose for C-8 in water from which the Screening Level of 150 ppb was then calculated. (Refer to Attachment G - Staats deposition, pp. 122-125)

Apparently Dr. Staats unilaterally decided to abandon the terms of the Consent Order and fashion her own procedure to determine a reference dose and screening level. She directed TERA to omit the values it derived from tables to encourage the CAT team, which included DuPont representatives, to, in essence, come "up with numbers themselves." (Refer to Attachment G - Staats Deposition p. 123)

Once Dr. Staats allowed the CAT team to usurp the responsibilities of TERA, the terms of the Consent Order were violated and the benefit of an independent analysis by TERA lost.

These actions not only violate the terms of the Consent Order but also call into question the impartial, scientific validity of the process employed and the results obtained from the CAT Team proceedings.

5. Citizens in the Community are Concerned about their Water and Air Quality

a. In January 2002 a local newspaper ran a poll – Does the finding of C-8 in water wells in Little Hocking, Ohio disturb you? 77.4% answered 'Yes'

b. In May 2002 another poll, after 150 ppb Screening Level came out - Do you feel safer now that 'health protective levels" have been determined for C-8 and those levels are far higher than what was found in area drinking water? 71.9 % answered 'No' – they do not feel safer.

c. Also in May 2002, another poll after 150 ppb Screening Level came out - How worried are you about C-8, now that the West Virginia DEP has determined a "safe" human level? 68% answered 'still concerned' or "remain very worried"

d. The local sales of bottled water tripled after C-8 was detected in local water supplies.

e. Some people are afraid to drink the water, or even bathe their babies in it.

f. A developer has complained of declining land values.

g. Realtors have told me of people moving to get away from C-8. Other people, who just bought homes, are concerned about the resale value of their property.

h. On June 20,2002, the Marietta Times published a newspaper article that stated that Washington County is the Ohio county with the highest incidence of lung cancer in the state.

The magnitude of the C-8 problem deserves the full attention of the Ohio Environmental Protection Agency. We ask that the Agency enter into a consent order with DuPont so that an independent analysis of the C-8 problem is done. We are seeking a thorough review and analysis, where we can have some degree of confidence in the results.

Regardless of whatever the "safe level" is for C-8, it is still a contaminant that does not belong in our water supplies. Of all the things that have been said about C-8, no one has ever said that it is good for us to have it in our drinking water.

HANDOUT WVDEP PUBLIC MEETING MAY 15, 2002

Reference	Critical Effect	Critical Effect Level[a]	UF_A	UF_H	UF_L	UF_S	UF_D	Composite UF[b]	RfD/RfC
Oral Studies									
Palazzolo et al. (1993)' 90-day rat study	Increased relative liver weight with histopathology in male rats	0.47 (NOAEL in males) 0.72 (BMDL)	10	10	1	1	1	100	0.005 0.0072
York et al. (2002) Two-Generation rat study	Increased liver weight in male rats, supported by histopathology at higher doses.	1 (LOAEL in males)	10	10	3	1	1	300	X
	Increased liver weight in male rats, supported by histopathology **at** higher doses (histopathology was not examined at the lowest dose, but incidence of hypertrophy was 100% at next highest dose).	0.42 (BMDL in males)[d]	10	10	1	1	1	100	0.004) REFERENCE DOSE
3M (1983) Two-year rat study	Tubular hyperplasia of the ovarian stroma and clinical signs (ataxia) in female rats.	1.6 (LOAEL in females) 1.57 (BMDL)	10	10	1	1	1	100	0.0157
	Hepatic megalocytosis in male rats.	0.73 (BMDL in males)	10	10	1	1	1	100	0.0073
Thomford et al. (2001)[e] 26-week cynomolgus monkey study	Decreased thyroid hormone levels in male cynomolgus monkeys, and supported by a NOAEL at the same dose for clinical signs of toxicity in the co-critical rhesus monkey study (Goldenthal et al., 1978)	3-10 (LOAEL in males)	10	10	3	3	1	1000	0.003 - 0.01

ATTACHMENT A

component is 1. When the toxicokinetic component (1) is combined with the toxicodynamic component (1), then the overall value for this factor is 1, but could be as low as 0.3.

Combining these factors results in a conservative MOE of 1000, which would be applied to the point of departure dose of 1.4 mg/kg/day (LED10) resulting in a risk specific dose of 1.4 µg/kg/day. Using default assumptions about drinking water consumption and body weight, a risk specific drinking water level would be 50 µg/L. This is essentially the same value as obtained from the use of the monkey data. Stated differently, as did the USEPA in their chloroform risk assessment (USEPA 2001b), the RfD derived from the monkey study is about 900 times smaller than the lowest LED10 derived from the rat bioassay. Given that the MOE supported by the data is 1000, and could be as low as 300, indicates that the use of the monkey data would be adequately protective of all potential effects.

7.0 CONCLUSIONS

The best available and most relevant data for deriving a drinking water level of PFOA that is protective of human health are those from the monkey study. Unlike the effects consistently observed in rat studies, neither indications of adverse liver effects nor changes in hormone levels occurred in monkeys treated with PFOA. This lack of a response in the monkey (non-human primate) is consistent with the lack of such effects in PFOA workers (human primate). The rat carcinogenicity data are of limited relevance to humans, especially to humans exposed to low levels of PFOA. As discussed in detail in Section 5.0, tumors in rats were produced only at high doses and only in tissues responsive to changes initiated by activation of PPARα by modes of action that have a threshold; similar responses are unlikely to occur in humans exposed to PFOA. Thus, the evidence strongly suggests that PFOA would not be a human carcinogen. Consequently, while a Benchmark Dose/Margin of Exposure approach can be applied to the rat cancer data, its outcome is of highly uncertain relevance to human health, and it is not recommended as a criterion for human health protection.

The RfD derived from the monkey data (2 µg/kg/day), provides a reliable and relevant criterion for human health protection, and should be used to derive a drinking water limit. Using default assumptions for drinking water consumption and body weight, the RfD corresponds to a drinking water of 70µg/L. If the contribution of PFOA in drinking water to the total intake of PFOA by all media is considered, then the final drinking water criterion would be adjusted accordingly. Using the USEPA default of 20%, the resultant drinking water criterion would be 14 µg/L; however, using a more realistic contribution of 50% would result in a drinking water criterion of 35 µg/L.

to 50 mg/kg/day APFO. There was no evidence of maternal toxicity at 50 mg/kg/day, the highest dose tested.

A two-generation reproductive toxicity study is currently being conducted, A two-generation reproductive toxicity study of PFOS showed high mortality of F1 pups at doses as low as 1.6 mg/kg/day. The results of the APFO study will be important to determine whether a similar effect is observed.

Carcinogenicity studies in Sprague-Dawley (CD) rats show that APFO is weakly carcinogenic, inducing Leydig cell adenomas in the male rats and mammary fibroadenomas in the females following dietary exposure to 300 ppm for 2 years (equivalent to 14.2 mg/kg/day in males and 16.1 mg/kg/day in females). The compound (at 300 ppm) has also been reported to be carcinogenic toward the liver and pancreas of male CD rats.

The mechanism(s) of APFO tumorigenesis is not clearly understood. Available data indicate that the induction of tumors by APFO is due to a non-genotoxic mechanism, involving activation of receptors and perturbations of the endocrine system. The liver carcinogenicity/toxicity of APFO appear to be related to induction of peroxisome proliferation following binding to the peroxisome proliferation activation receptor a (PPAR a) in the liver. Available data suggest that the induction of Leydig cell tumors (LCT) and mammary gland neoplasms by APFO may be due to hormonal imbalance resulting from activation of the PPARa and induction of the cytochrome P450 enzyme, aromatase. Preliminary data suggest that the pancreatic acinar cell tumors are related to an increase in serum level of the growth factor, cholecystokinin.

As the mechanisms of carcinogenic action of APFO have not been fully elucidated, it is assumed that the tumors induced in rats are relevant to humans. Review of available mechanistic data of other drugs and chemicals that induce LCT in animals has led a workshop panel to conclude that all but two modes of induction of the luteinizing hormone (LH), "dopamine agonism" and "GnRH agonism", are considered to be relevant to humans, and that the possibility of induction of Leydig cell adenoma in humans by specific agents with other modes of action cannot be ruled out despite the rarity of LCT in humans. At present, there is no evidence that the induction of LCT by APFO is via the "dopamine agonism" or "GnRH agonism" mode of action. It is recognized that there are quantitative differences in certain biological parameters between rats and humans. However, the principal cell control mechanisms appear similar, and the difference in carcinogenic response is probably quantitative. As binding to the PPARa appears to be the critical event leading to hormonal imbalance and APFO tumorigenesis, and the level of PPARa in human livers is lower than that in rodent liver, it appears that humans may be less sensitive than rodents in the development of LCT, mammary gland tumors, or liver neoplasms.

6pp

AR226 - 0629

Charles Aucr
05/16/2000 11:11 AM

To: Barbara Leczynski/DC/USEPA/US@EPA, Mary Dominiak/DC/USEPA/US@EPA, Frank Kover/DC/USEPA/US@EPA, Ward Penberthy/DC/USEPA/US@EPA, Karen Lannon/DC/USEPA/US@EPA

cc:

Subject: Phaseout of PFOS

fyi

---------- Forwarded by Charles Auer/DC/USEPA/US on 05/16/2000 11:17 AM ----------

Charles Auer
05/16/2000 11:06 AM

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Subject: Phaseout of PFOS

I would like to draw your attention to an important development in the US which concerns a persistent. bioaccumulative, and toxic chemical. I will be approaching the OECD Secretariat about setting up a discussion opportunity at some point during the upcoming meeting of the Task Force on Existing Chemicals. A brief summary of the information follows below and this is accompanied by a number of documents which provide additional information (EPA's press statement. 3M's press statement, and several reports submitted to EPA by 3M which provide more detailed background information). The reports from 3M will follow separately as .pdf files and are not being sent to the cc's.

Following negotiations with EPA. 3M Corporation today announced that it will voluntarily phase out perfluorooctanyl sulfonate (PFOS) chemistry. which is used to manufacture a wide range of products. This announcement is the result of a successful production stewardship effort between 3M and EPA. EPA supports this effort which began as a result of data 3M supplied to the Agency which indicated that these chemicals are very persistent in the environment, have a strong tendency to accumulate in human and animal tissures and, based on recent information,

could potentially pose a risk to human health and the environment over the long term. The company plans to exit worldwide from production of these chemicals by the end of the year.

PFOS (perfluorooctane sulfonic acid) is a member of a large family of sulfonated perfluoro-chemicals (total annual production < 5 million kgs) which are used for a wide variety of industrial, commercial, and consumer applications (including use as a component of soil and stain-resistant coatings for fabrics, leather, furniture, and carpets (under the Scotchgard line), in fire-fighting foams, commercial and consumer floor polishes, cleaning products, and as a surfactant in other specialty applications); pesticidal and indirect food use products are also made from this technology. Final formulations for these uses contain less than 1% of the PFOS chemicals. All of these chemicals have the potential to degrade back to PFOS which does not *appear* to degrade further (it is thus highly persistent). 3M Corporation is the sole US manufacturer of the PFOS family of chemicals; 3M also has a production facility in Belgium. Available information suggests that there may be production facilities in Italy, Germany, Japan, and the Russian Federation, although 3M appears to be the dominant producer.

PFOS has been found widely in human blood samples (ppm levels in manufacturing workers, ppb levels in non-exposed workers and in blood bank samples. PFOS has also been found in wildlife species across the US (especially in fish *eating* birds) and in the Baltic in Sweden. It was detected in naive (unexposed) laboratory rats (the PFOS contamination was traced back *to* fish meal used in the rat chow).

PFOS caused postnatal deaths (and other developmental effects) in offspring in a 2-generation reproductive effects rat study (NOAEL of 0.1 mg/kg/day and LOAEL of 0.4 mg/kg/day). At higher doses in this *study,* all progeny in first generation died while at the LOAEL many of the progeny from the second generation died. It is very unusual to see such second generation effects.

PFOS accumulates to a high degree in humans and animals. It has an estimated half-life of 4 years in humans. It thus appears *to* combine Persistence, Bioaccumulation, and Toxicity properties to an extraordinary degree.

Several years *ago,* in response to the blood findings, 3M launched a major research effort on PFOS to characterize its environmental presence, environmental and human effects, and environmental fate.

EPA REVIEW

Preliminary data indicated to EPA that PFOS is of significant concern on the basis of evidence of widespread human exposure and indications of toxicity in a 2 generation rat *study.* In addition, EPA's preliminary risk assessment indicated potentially unacceptable margins of exposure (MOEs) for workers and possibly the general population. There are many assumptions and considerable uncertainty in these arguments and analyses. It is not possible at present to judge the adequacy or accuracy of the MOE analyses or whether the *exposure* levels used in the above estimations may be considered representative of the affected populations at large. EPA requested detailed information from 3M and a large body *of* information has been received but

not reviewed.

3M has raised questions regarding the possible relevance to humans of a proposed mechanism (effects on cholesterol biosynthesis) for PFOS's lethal effect in the 2-generation study. The proposed mechanism, the company argues, affects reproductive outcomes in litter bearing animals due to its inhibitory effect on a burst of cholesterol biosynthesis in the critical period just before birth. The proposed mechanism would, if demonstrated, have broad implications for and present significant potential concerns for humans and environmental organisms.

RECENT DEVELOPMENTS

Following a series of discussions with EPA, and based on concerns about the widespread presence and longer term risks presented by PFOS, 3M decided that it would exit worldwide from this market by about the end of the year, although it may need to extend the time period for some critical uses (e.g., fire fighting foam). The company had previously launched a major research efforts on PFOS to provide an in-depth understanding of the problem and its human and environmental consequences; this research effort would be continued despite the commercial decision. 3M has expressed interest in collaborative efforts with EPA as they withdraw from the market and in the development of safer substitutes.

NEXT STEPS FOR EPA

EPA is preparing a communications strategy for conveying clear messages in response to 3M's announcement. We will be alerting other US Agencies (FDA, CPSC, OSHA, NIOSH), OECD governments, and international agencies (UNEP, IPCS). We do not believe that PFOS presents an imminent harm from use in consumer products during the phaseout (it is used in high molecular weight polymers which do not appear to result in exposure to PFOS during normal use; residual PFOS contamination occurs at very low levels). At the same time, we agree that continued manufacture and use of PFOS represents an unacceptable technology that should be eliminated to protect human health and the environment from potentially severe long term consequences. Regulatory action would have been difficult and time consuming at best and, given EPA's view that a rapid phase out is necessary and appropriate, EPA believes that 3M has taken a responsible corporate decision in quickly moving away from this technology.

EPA is currently examining appropriate regulatory steps necessary to ensure protection of human health and the environment.

PFOA

PFOA (perfluorooctanoic acid) is closely related structurally to PFOS and is used as a solvent for certain polymerization reactions. EPA has requested information from producers and will be preparing an assessment. Based on preliminary information, PFOA presents a different hazard, exposure, and risk picture compared to PFOS. 3M has also committed to ending production of PFOA. There are other producers in the US and EPA is examining its options regarding action on PFOA.

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UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

MAR 28 2002

OFFICE OF
PREVENTION, PESTICIDES AND
TOXIC SUBSTANCES

Dear Interested Party:

Attached is a copy in PDF file format (389KB) of the *Draft Hazard* Assessment *Of Perfluorooctanoic Acid (PFOA) And* Its Salts, prepared by the Risk Assessment Division of the EPA Office of Pollution Prevention and Toxics. The Annex to the assessment, which contains robust summaries of the studies reviewed in the assessment, is not attached because of its very large size (785KB), but is available upon request as a separate file. Both files have been placed in the PFOA section of the publicly accessible Administrative Record file AR226: PFOS, PFOA, Telomers, and Related Chemicals. This preliminary assessment reviews the studies that were available as of July 2001, but does not incorporate any information received by the Agency after that date. The cut-off date was adopted to allow the release of this preliminary assessment while additional studies were still underway. The assessment specifically recognizes that additional data, particularly including the final report on a two-generation reproductive study in rats, are still under development. As the Agency receives further study information, it will be included in AR226 and in the Agency's review process.

Based on the existing data, PFOA may present some concerns similar to those raised by perfluorooctyl sulfonates (PFOS). Like PFOS, it is persistent in the environment and does not appear to degrade. PFOA also appears to have a half-life in humans of between 1 and 3.5 years, indicating that it may bioaccumulate in humans in the same manner as PFOS does, remaining in enterohepatic circulation. PFOA has been found in human blood samples, although at levels lower than PFOS. PFOA is carcinogenic in animals. Reproductive study data on PFOA comparable to the studies that raised the initial concerns on PFOS are not yet available, although the final report on the two-generation reproductive study in rats should be completed in the first half of 2002 and will be included in AR226 as soon as it is submitted to the Agency.

The Agency will continue to review data on PFOA as they are received.

If you have any questions concerning the Assessment, please contact Jennifer Seed by phone at 202-564-7634, or by email at seed.jennifer@epa.gov. If you wish to receive a copy of the Annex, or if you have any difficulties opening the files, please contact Mary Dominiak by phone at 202-564-8104, by fax at 202-564-4775, or by email at dominiak.mary@epa.gov.

Sincerely,

Charles M. Auer, Director
Chemical Control Division

First Public Meeting, CAT Team members shall familiarize themselves with the available toxicological information concerning C8.

A CAT Team meeting shall be held immediately prior to the first public meeting to: (1) conduct a site visit to the three landfills and the Washington Works Plant, and surrounding residential areas; (2) discuss the toxicity of C8 and other pertinent data; **(3)** prepare an agenda for the public meeting; **(4)** coordinate and prepare for the public meeting. Finally, the First Public Meeting will be held and public questions and comments will be recorded by WVDEP.

TABLE 1. TASKS OF CAT TEAM

Task A: Public Meetings (two meetings are anticipated) Objective: to inform the local citizens of the following: (in Meeting #1) intent to perform a groundwater well use survey and analysis for C8; intent to develop Screening Levels; and to ask for their cooperation in conducting the water use survey; and (in Meeting #2) results of survey, chemical analysis, and risk assessment. Note that an interim public meeting may be required should six months pass from the first public meeting and the CAT Team Final Report has not been issued. Primary Responsibility: Staats
Task **B:** Development of Provisional Reference Doses Objective: to develop Provisional References Doses for C8 for the inhalation and ingestion (and dermal, if possible) routes of exposure. Primary Responsibility: TERA
Task C: Development of Screening Levels Based on Protection of Human Health Objective: to utilize the Provisional Reference Doses to develop human health risk-based Screening Levels for C8 in air, water, and soil. Note a determination of the potential carcinogenicity of C8 will be conducted as well. Primary Responsibility: TERA
Task D: Ecological Data Review Objective: to review available information to determine whether sufficient studies have been performed and data have been collected to develop screening criteria for ecological receptors. Primary Responsibility: TERA
Task E: Draft Report and Final Report Objective: to present and discuss the results of the above tasks. Primary Responsibility: TERA

Phase II Tasks B, C, D, and E Development of Provisional Reference Doses and Screening Levels, and Risk Assessment

In Phase II, TERA shall conduct the toxicological and risk assessment activities. After having reviewed the toxicological information regarding C8 provided by WVDEP, **TERA** shall consult with toxicologists **on** the CAT Team, as coordinated by Dr. Staats, regarding its proposed approach for this project. Following such consultation, TERA

shall develop Provisional Reference Doses for C8 for the oral, inhalation, and dermal (if possible) routes of exposure. Then TERA shall calculate Screening Levels for water, soil and air based on the **risk** factors they have estimated. TERA shall perform one general risk assessment involving comparison of exposure concentrations to Screening Levels for the three landfills and the Washington Works Plant, and the Lubeck Public Service District water supply, that focuses on current risk to human health, including workers and residents. This risk assessment shall include: (1) identification of reasonably anticipated land use, surface water and groundwater use; (2) identification of receptors; **(3)** identification of exposure pathways; **(4)** identification of exposure concentrations; and (5) comparison of exposure concentrations to appropriate Screening Levels. TERA shall utilize data obtained from the other efforts discussed above such as air modeling; groundwater C8 concentrations in residential and public wells; residential groundwater well use survey; the USEPA's Draft Hazard Assessment; and ATSDR's Health Consultation (if available). TERA also shall review available information to determine whether sufficient studies have been performed and data have been collected to develop screening criteria for protection of ecological health, particularly aquatic life. TERA shall prepare a draft and a final document that discusses the results of their efforts and summarizes the data utilized from other efforts. **As** the tasks of the CAT Team and other involved parties' progress, data gaps and research recommendations may become evident. These shall be included in TERA's report as suggestions for further research to elucidate the toxicity of C8.

Phase III Second Public Meeting

The purpose of the Second Public Meeting is to present to the citizenry the results of the efforts of the **GIST** and CAT Teams including C8 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.

SCOPE OF WORK FOR TERA

TERA (Toxicology Excellence for Risk Assessment) is a non-profit organization that applies sound toxicological data to the **risk** assessment process to find common ground between environmental, industry, and government groups. TERA will be providing services in toxicology and **risk** assessment. TERA scientists **will** be developing risk factors and screening criteria; and conducting one general risk assessment for the 3 landfills, Lubeck Public Service District water supply and the Washington Works Plant, The specific tasks assigned to TERA are described below.

Phase II Tasks B, C, D, and E: Development of Provisional Reference Doses and Screening Levels, and General Assessment of Risk

Subtask 1 – TERA staff will familiarize themselves with the toxicological data provided to by WVDEP. No literature search or document retrieval **will** be required. Toxicological data to be provided to TERA shall include but is not limited to the following:

a. 8 compact discs of information provided to USEPA under TSCA by 3M Corp (note only a small portion of this information concerns C8);
b. USEPA Draft Hazard Assessment for C8;
c. Journal articles and other information submitted to WVDEP by DuPont.

Subtask 2 – TERA staff will:

a. identify all possible critical toxicological studies suitable for developing Reference Doses for the oral, inhalation, and dermal (if possible) routes of exposure;
b. outline methodology for developing said Reference Doses and for developing Screening Levels for air, water, and soil based on said Reference Doses corresponding to each critical study identified in subtask **2-a;**
c. convene a meeting at the TERA facility in Cincinnati, Ohio, to present their findings in subtask 2-a and **2-b,** and consult with CAT Team toxicologists as coordinated by Dr. Staats;
d. finalize Reference Doses and Screening Levels based on recommendations of the CAT Team toxicologists as coordinated by Dr. Staats.

Subtask 3 – TERA shall conduct one general risk assessment for the three landfills and Washington Works Plant, and the Lubeck Public Service District water supply based on current **risk** to human health. This risk assessment shall include:

a) identification of reasonably anticipated land use, surface water and groundwater uses;

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FROM DR. STAAT's DEPOSITION

A. They may have the table and not the e-mail. Are you asking --

Q. Whatever they have. Whatever they have. So this was a one-page chart similar to Exhibit 11 but all the columns are filled in by TERA?

A. This is multi-page.

Q. What were the numbers TERA picked?

A. I don't recall.

Q. Do you recall what their total screening level number was for drinking water?

A. I don't recall. I am not even positive if I had them take it to a screening level, only to the reference of filling in this table. This table, as you see, doesn't have a column for screening level; it stops at RSD.

Q. Do you know whether the numbers were higher or lower than those that the CAT Team came up with on May 6th and 7th?

A. I don't recall.

Q. You don't recall what generally, even generally speaking what the range of the numbers were in comparison to what --

A. No. I just briefly looked over it, and

as I thought about it it made more sense to me to -- and I discussed it with TERA.

Q. Who at TERA?

A. Andy Maier and Joan Dollrhide probably. I am positive Andy Maier. Sometimes Joan was also on the phone and sometimes not.

But I told them that I wanted them to lead us through the process.

And at the meeting I told them that they could make suggestions. And it would more, that that table that was filled would be, not presented in the sense of given to the people at the meeting but that TERA could make suggestions.

But as the meeting proceeded it became clear that TERA, we were leading, but 10 opinionated toxicologists took the discussion in hand and came up with the numbers themselves. And we were, again, in agreement. And I let it go forward that way.

Q. So you don't remember any, even remotely what any of the values were that TERA had come up with on its own?

A. No.

Q. Do you know, for example, what they had picked, whether they picked uncertainty factors of 10 for any of the UFLs, UFSs or UFDs on the chart?

A. I don't remember the table at all, I mean the numbers that are in it at all. As I said, I simply scanned it and thought about how would, how did I want to set this up, would that influence the other people. I wanted no influence at all. I wanted each and every one of them's opinion. So I didn't want to influence their opinions. So I made the decision -- Of course, TERA would lead us through it. That's their job. If they wanted to make recommendations they could. But quickly the group was very opinionated and made their own decisions quite quickly.

Q. Do you recall whether the opinions that the group came up were different from what TERA had suggested?

A. How could I recall when I don't remember what TERA suggested.

Q. You got this chart from TERA and you golooked at it. Was there something about it

that caught your eye that made you think that we ought to let the CAT Team do this instead of what TERA has come up with?

A. No. Just the fact that those uncertainty factors were already filled in. And I didn't want the team to feel like the decision was already made. It should be a time decision. So it implied, if I would have sent it to them, that the decision had already been made and we were simply reviewing what TERA had already done.

I wanted it to be a group decision, so that everyone's opinion mattered, everyone contributed. We reach a consensus. Hopefully, it is not a consensus then majority rules.

Q. Did anybody else get the preliminary chart that TERA had prepared and e-mailed to you?

A. No.

Q. Did you ask TERA to do anything with that chart or e-mail?

A. Just to remove the uncertainty factors and make one without the minute.

Q. Did you ask them to destroy that chart