AR226 – 1156

ewg/science policy

Critique of community briefings by West Virginia DEP

>>| EWG Letter to West Virginia DEP

>>| EWG Policy Memo

The following is the text of the letter written to West Vi DEP stating EWG's findings:

November 12, 2002

Mr. Michael Callaghan
Secretary
West Virginia Department of Environmental Protection
1356 Hansford Street
Charleston, WV 25301

Dear Secretary Callaghan:

Contamination of drinking water supplies by the toxic industrial chem perfluorooctanoic acid (PFOA, or C8) is a continuing concern to the res Parkersburg and surrounding areas of Wood County near the source o pollution, DuPont's manufacturing operation in Washington, West Virg

As part of an ongoing assessment of human health and environmental by C8 and related compounds, researchers at the Environmental Work (EWG) examined materials on the subject prepared by the Departmen Environmental Protection (DEP) and presented by agency staff at seve meetings last summer. Our focus was on the science behind DEP's wo conclusions the Department presented to Wood County residents who exposed to C8 pollution from the DuPont Plant.

EWG's over-arching conclusion is that the DEP's assessment is serious numerous crucial — and often very basic aspects of C8 science and to review of the science available at the time of the briefings shows that presentations made by the DEP did not accurately represent what was known about C8 toxicity. These shortcomings are compounded by the misinterpretation and misapplication of standard procedures for estab drinking water screening levels and contaminant limits.

Last July, in the context of a controversy surrounding a review undert DEP on the hazards posed by C8 contamination in the state, you state

> "If there is any reasonable question about the science behind the DEP's work, I welcome the input. Constructive criticism of a proc or result is part of science and it is our intent to assure citizens o Wood County that the work done regarding C8 has been accurat and reliable." (*The Charleston Gazette*, July 5, 2002)

We have five major concerns with the scientific conclusions and stater by the DEP at these public meetings. Each of them is explained in gre and fully referenced in the attached review by Dr. Kristina Thayer of o Specifically:

1. DEP materials assert that C8 does not cause developmental and reproductive effects or present a long-term risk to people who d in their tap water. This contention is not supported by industry s of C8 toxicity and is contradicted by high-ranking scientists and at US Environmental Protection Agency.
2. The DEP concludes that tumors caused by C8 in animals are irrel to humans. The US EPA, in contrast, considers C8 to be carcinog causing liver, pancreatic, testicular, and mammary gland tumors company documents, DuPont scientists assert that the testicular pancreatic tumors found in animals could be relevant to humans
3. The DEP fails to account for documented food and air exposures when setting the drinking water contaminant limit, and then fails the public of this omission, even though it constitutes a major de from standard US EPA protocols. Assuming that water is the only of exposure is a one big reason that the DEP arrived at a drinkin screening level that allows between 15 and 100 times more C8 i water than would be normally permitted. In addition, the DEP di

account for bottle fed infants and children, who are likely more susceptible to the toxic effects of C8 and who certainly get a high dose relative to their size than an adult when drinking a liter of C contaminated water.

4. DEP's public presentation materials assert that the allowable amc C8 contamination in drinking water is between 10 and 100 times protective than it actually is. If DEP staff had actually applied the factors they describe at the community meetings, the screening for C8 in drinking water would be between 1.5 and 15 parts per l (ppb), not 150 ppb.
5. The DEP review concludes that the allowable dose of C8 (the refe dose, or RfD) is five times more protective than that for the aver chemical. It is not. The reference dose for C8 is five times lower stringent) than the average RfD because C8 is five times more tc than the average chemical. The amount of protection provided b' C8 reference dose is equivalent to, or less than (see below) that provided for the average compound.

Overall, in every important matter of scientific assessment and regula judgment, the DEP appears consistently to have come down on the sic less protective of human health and the environment in Wood County highly favorable to the polluting company, DuPont.

This tilt not only does a serious disservice to affected West Virginians, in marked contrast to the evolving regulatory posture towards C8 at U September 27, the agency initiated a rare, priority review of C8 out of about its developmental and reproductive effects, and because "additi sample analysis data indicate low level exposures to the general popul are unexplained at this time."

Significantly, the EPA's motivating concern is with risks to the general population, where the potential for exposure is generally much lower t West Virginian's whose drinking water supplies have been directly con with C8 from manufacturing and disposal operations.

The studies that instigated US EPA's priority review were available to I advance of your agency's public presentations earlier this year. A perf review of industry documents on file with the agency would have yield and scientific conclusions very much at odds with the "science behind on C8.

EWG is aware of, but has not been party to, the controversy surround DEP's review of C8, specifically the procedures followed by the depart Science Advisor Dee Ann Staats, and her decision to include DuPont p the DEP review panel. We are at a loss to explain how the weight of e readily available from EPA and published sources about the serious ris could differ so dramatically from the conclusions the DEP reached, anc reassurances it communicated to the people of Wood County.

In order to remove even the appearance of bias in the DEP process, f other reason, we urge you to impanel another, impartial group of scie excluding industry-affiliated experts, to re-review C8 science and the application of drinking water regulation to the contamination problems County. It is our judgment that such a review would result in much str regulatory action against C8, and vastly stricter protection for West Vi whose tap water was once pure, but is now contaminated by a danger industrial chemical.

Sincerely,

[signed]

Richard Wiles
Senior Vice President

ewg/science policy

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A Critique of Community Briefings by The West V Department of Environmental Protection on the I of Drinking Water Contaminated With C8 (perfluorooctanoic acid — PFOA)

Kristina Thayer Ph.D.

Throughout the spring and summer of 2002, the West Virginia Departi Environmental Protection (DEP) conducted a series of community mee hazards of drinking tap water contaminated with perfluorooctanoic aci C8) from DuPont's manufacturing operations in Washington, West Virg analysis of the content of the materials presented by the DEP at these finds them to be in conflict with positions of the U.S. Environmental Pr Agency (U.S. EPA), with studies in peer-reviewed scientific literature, industry-sponsored toxicity studies.

The following analysis refers specifically to statements made, and slide presentation made by DEP staff and expert consultants on May 15 anc In describing the health concerns associated with C8 and the safety st devised by the State, the DEP:

1. Clearly indicates that C8 does not cause developmental and reproductive effects or present a long-term risk to people who d in their tap water. This contention is not supported by industry s of C8 toxicity and is contradicted by high-ranking officials at U.S
2. Claims that tumors caused by C8 in animals are irrelevant to hur The U.S. EPA considers C8 to be carcinogenic — causing liver, pancreatic, testicular, and mammary gland tumors [U.S. Environ Protection Agency (U.S. EPA) 2002b, p. 6]. In company documer DuPont scientists assert that the testicular and pancreatic tumor in animals could be relevant to humans.
3. Fails to account for documented food and air exposures to C8 wh setting the drinking water contaminant limit, and then fails to tel public of this omission, even though it constitutes a major deviat from standard US EPA protocols. DEP's method allows between 1 100 times more C8 in tap water than would be allowed applying assumptions which account for other sources of exposure to C8 a include protections for the fetus, infants and children from the ef suspected developmental toxins like C8 [Ammonium perfluorooct (C8) assessment of toxicity team (CATT) 2002, p. 34; Appendix
4. Asserts that the allowable amount of C8 contamination in drinkin is between 10 and 100 times more protective than it actually is. DEP applied the safety factors described at the community meeti the screening level for C8 in drinking water would be between 1 15 parts per billion (ppb), not 150 ppb.
5. Claims that the "safe" dose of C8 (the reference dose, or RfD) is times more protective than that for the average chemical. It is n reference dose for C8 is five times lower than that for the averag chemical because C8 is five times more toxic than the average chemical. The amount of protection provided by the C8 reference is equivalent to, or less than (see below) that provided for the av compound.

C8 and developmental/reproductive toxicity

On May 15th or 16th, 2002, DEP staff presented the following informa public [West Virginia Department of Environmental Protection (WVDEP Slide #3]:

How toxic is C8?
- Not developmentally toxic
- Not a reproductive toxicant

[Excerpt | Full document]

Far from considering C8 "not developmentally toxic," the U.S. EPA has "priority review" of C8 to determine whether expedited regulatory acti the compound is warranted under Section 4(f) of the Toxic Substances Act (TSCA) due to concerns that the compound causes adverse develo and reproductive effects in animals.

As characterized by Oscar Hernandez, the director of the U.S. EPA's O Prevention, Pesticides and Toxic Substances Risk Assessment Division "Toxicological studies in rodents and primates have shown that exposu (C8) can result in a variety of effects including developmental/reprodu toxicity, liver toxicity, and cancer" (U.S. EPA 2002c, p. 2:) [Excerpt | F document].

According to Charles Auer, Director of the U.S. EPA's Office of Toxic Su the agency is taking this action because it has received data suggestin has "a potential for reproductive/developmental toxicity," and because blood sample analysis data indicate low level exposures to the general that are unexplained at this time" (U.S. EPA 2002d, p. 1) [Excerpt | Fu document]

Our review of the studies available in the scientific literature shows tha findings are not new, and were available to the DEP well in advance of presentations.

For example, C8 causes numerous effects on male and female reprodu organs, such as testicular tumors (Biegel et al. 2001; Sibinski 1987), sperm production (Sibinski 1987), mammary gland tumors (Sibinski 1 cellular effects in the ovary (Sibinski 1987). C8 also affects the prosta (York 2002) and increases estrogen levels (Biegel et al. 2001). In add causes a significant increase in low birth weight pups in animal studies weight is recognized as a risk factor for insulin resistance or Type II di high blood pressure, and cardiovascular disease later in life (Godfrey a 2001, Hales and Barker 2001). Studies have also shown that health ri even for those low-birth weight children who achieve normal weight la childhood, including risks for high blood pressure, stroke, insulin resist glucose intolerance (Eriksson et al. 2000a; Eriksson et al. 2002; Eriks 2000b; Eriksson et al. 1999; Eriksson and Forsen 2002; Forsen et al. and Dunger 2002; Stettler et al. 2002).

More recent work shows that C8 significantly increases mortality in developmentally-exposed rats at a dose that did not affect parent sur 2002). These findings suggest that fetuses, infants and children may b sensitive to C8 exposure than adults. Additional effects observed in developmentally-exposed male rats - at the lowest doses tested - inc decreased body weight; increased liver, seminal vesicle (a part of the reproductive tract), and kidney weight; and decreased weight of the s important organ in the immune system. At higher doses, C8 delayed s maturation of male and female rat pups (York 2002).

C8, cancer, and other long-term health risks

Also, in Slide #3 from DEP's public presentation on May 15 or 16, 200 incorrectly represent the relevance to humans of C8 cancer studies in animals:

How toxic is C8?
- Cancer — rodents; mechanism not relevant to humans

[Excerpt | Full document]

DEP implies that all types of tumors (liver, mammary gland, testicular pancreatic) caused by C8 in rodents are not relevant to humans. This is not supported by the science, nor has any regulatory body conclude Indeed, not even DuPont or 3M have attempted such a sweeping deni health risk to humans.

The manufacturers have argued that one of the mechanisms of action tumors in rats is a cellular process called peroxisome proliferation, a n generally thought to be much less relevant for humans than for roden studies in primates, however, showed liver toxicity for C8 without perc proliferation. Primates are similar to humans in liver physiology and fu Specifically, C8 has been found to cause death in monkeys along with gastrointestinal tract toxicity, increased triglycerides levels and transie decreases in thyroid hormone even though "there was no evidence of proliferation... suggesting a different mode of action than observed in EPA 2002b, p.5, 53-56) [Excerpt | Full document]. In fact, the lowest administered to monkeys caused "liver toxicity and possible mortality" 2002b, p.56) [Excerpt | Full document].

3M and DuPont failed to persuade the European Union with this same proliferation argument when attempting to refute liver tumors caused chemical cousin to C8, known as PFOS. The EU review, which is the m definitive review of PFOS toxicity to date, concluded "there was no evi peroxisome proliferation in the livers of the treated animals" - even th animals had liver tumors (Organisation for Economic Co-operation an Development 2002, p. 7) [Excerpt | Full document] - indicating that li could be induced by a different mechanism that may well be relevant t

According to the DEP's Dr. Staats in her May 15, 2002 presentation at Blennerhassett Jr. High School, one of the impetuses for the DEP inve C8 stems from the "toxicity of similar compound PFOS" (WVDEP 2002 [Excerpt | Full document], a chemical that 3M phased out of productio under pressure from U.S. EPA, when studies showed widespread huma and fetal deaths in low-dose animal studies.

Regardless of how the arguments for the C8-induced liver tumors evo also been shown to cause tumors in the mammary gland, testes, and (Biegel et al. 2001; Sibinski 1987; U.S. EPA 2002b, p.6). Scientists at have not concluded that a mechanism unique to rodents is responsible these tumors (U.S. EPA 2002b, p. 75-78). Even DuPont found that for and pancreatic tumors, "the mechanism of tumorigenesis is not compl understood, and therefore the relevance to humans can not be comple out" (DuPont 1997, p.25) [Excerpt | Full document].

DEP's "safe" C8 level in tap water is based on an a methodology that allows far more C8 in tap water the standard assumptions used by the U.S. EPA.

In a May 9, 2002 press statement, DEP writes:

> "The team determined that a water level that contained less thar parts per billion of C8 would not cause harm to humans," said Dr Dee Ann Staats, West Virginia Department of Environmental Protection science adviser.
>
> "The team thoroughly reviewed the scientific studies and unanimously agreed to the human health protective levels for drinking water," Staats said. "We brought together some of the t scientists in the country. I am confident the human health protec levels established by the team are supported by the data." (WVD 2002d)
>
> [Read full press release on DuPont's website]

What the DEP didn't tell the public is that in setting this standard, the deviated from its draft assessment and from standard assumptions us U.S. EPA when setting contaminant standards for drinking water. The

these omissions was to permit substantially more C8 in tap water thar allowed using standard assumptions.

First, your Department stripped an important safety factor from its fin calculations of a drinking water safety limit, tripling the allowable leve WVDEP initially used this safety factor in draft calculations to account C8's unusual properties, including its long half-life in the human body persistence in the environment (WVDEP 2001) [Excerpt | Full Docume not known to ever break down in water, soil, air, or the human body, property that heightens public health concerns and that merited an ad safety factor in your Department's draft calculations. Not only did you this safety factor from final calculations, but also they then presented the public implying that the safety factor had been left in place [Excer document]. If this were true, the allowable level of C8 in drinking wat 50 ppb, not 150 ppb.

Second, the state ignores potentially substantial background exposure from contamination in air and food. Water is not the only – and may n primary – route of exposure to C8. 3M reports that PFOA is present in beans, apples, bread, and ground beef at concentrations comparable t been found in drinking water supplies around DuPont's Washington W (U.S. EPA 2001b, p. 1) [Excerpt | Full Document]. Recent modeling of concentrations conducted by DuPont show significant levels of C8 in th especially northeast of the Washington Works Plant across the Ohio Ri exceeding safety limits set by the WVDEP (DuPont 2002).

For contaminants with multiple routes of exposure, U.S. EPA's standar assumption is that 20 percent of contaminant comes from drinking wa percent comes from other sources (EPA 1992). Had DEP used this star assumption, allowable amount of C8 in tap water for an adult would b 30 ppb, not 150 (Appendix A). Adding back in the Department's safety persistence would bring the drinking water screening level to protect a to 10 ppb.

In addition, the Department fails to set a protective level for infant ex Specifically, your Department did not account for the significant body difference between adults and infants. Bottle-fed infants fed formula reconstituted with C8-contaminated tap water are a population of high Bottle-fed infants receive the highest dose of all drinking water contan pound for pound, of any other segment of the population. This is beca an adult and an infant drink a liter of contaminated water, C8 will be r concentrated in the child — resulting in a higher dose per unit of body

A C8 level protective of bottle-fed infants would incorporate three moc factors your Department has ignored – a correction for infant body we safety factor for C8 persistence, and a factor to account for multiple rc exposure (such as air). Incorporating all three factors gives an allowat C8 in drinking water of 1.5 ppb (Appendix A), well within the range of found in the local drinking water sources. For instance, the peak C8 le springs that provide drinking water within one mile of the Washington facility were above 10 ppb (WVDEP 2002a, Slide 8) [Excerpt | Full Doc Average C8 levels in this same water were 4 parts per billion. During t 1990's, DuPont established its own Community Exposure Guideline (C in drinking water at 1 ppb (CATT 2002, p. 46) [Excerpt | Full Docume

Considering that infants may be more vulnerable to C8, the standard s be more conservative than what we have outlined here. Animal studie C8 is more toxic to newborns; for example, it causes mortality in your doses that did not kill parent animals. Studies also show that the high concentrations of C8 in non-worker populations occur in children (U.S 2002b, p. 3) [Excerpt | Full Document] reinforcing the need for DEP tc C8 safety limits to protect infants.

DEP implies that tap water safety limits of C8 are 100 times more protective than they actually are.

In another public meeting presentation, on either May 15 or May 16, staff imply that they applied a large safety buffer when deriving the dr water safety limit for C8 (WVDEP 2002b, Slide 5) :

How are safe levels developed?

No Observed Effect Level (NOEL) typically divided by at least 1,0 up to 10,000 Safety Factor

- Animals to humans
- Sensitive subpopulations
- Chronic lifetime exposure
- Other criteria such as biopersistence

[Excerpt | Full document]

The problem here is that the DEP did not apply the "typical" safety ma 10,000 — or even 1000. Instead, DEP applied a safety factor of only 1 account for possible differences between small groups of rats in the la and large populations of humans exposed through contaminated drink Also, some of the listed criteria, such as biopersistence, were not take account at all. The most recent data from 3M shows that it takes hum years to get rid of just half the amount of C8 in the body after all C8 e end (U.S. EPA 2002a, p. 2) [Excerpt | Full document]. If persistence w including when determining a safe dose of C8, the allowable amounts been much smaller, in part because there is no reason to believe the (exposures are ending.

DEP states, wrongly, that protections from C8 in d water are somehow more protective than protecti applied to other chemicals.

During a May 15, 2002 public meeting at Blennerhassett Jr. High Scho staff presented the following slide:

> How does the C8 RfDo (reference dose, which means the daily "s dose) compare to that for other chemicals?
>
> The average EPA-developed oral RfD is 0.02 mg/kg-day which is times higher than C8's of 0.004 mg/kg-d. Therefore, C8's risk fa is more conservative or health protective than that for the avera chemical. (WVDEP 2002c, Slide #15)
>
> [Excerpt | Full document]

C8's reference dose is not more health protective than that for the ave chemical. C8 carries a lower reference dose than the average chemica is more toxic than the average chemical, approximately 5 times more according to DEP's own calculations. C8's low reference dose simply re difference.

In truth, far from being more protective than average, there are sever why the DEP risk factor may not be sufficiently protective. First, a dos causes no observed adverse effect level, or a NOAEL, was not identifie critical studies, the cancer study, and the reproduction study. The repr study was used by the DEP-led team to set a RfD for C8. Typically, to design animal studies with the expectation that the lowest dose testec produce adverse effects in laboratory animals. C8's manufacturers underestimated the toxicity of C8, and missed the no effect level in bc studies — even the lowest doses tested caused observable adverse eff not at all accurate to characterize a risk factor derived from studies th adverse effects at all doses as "more conservative or health protective average risk factor.

Second, the study used to derive the RfD (a two-generation reproduct study) is not a chronic study, and is not truly indicative of the hazards term consumption of C8 in tap water. In this study, animals exposed i followed, at a maximum, until they are young adults (about 4 months

design makes it impossible to address questions such as whether in ut life exposure to C8 can predispose an individual to cancer, degenerati system disorders, diabetes, or other diseases more prevalent at the e Third, C8 is an extremely biopersistent chemical, which was not taken account in setting the reference dose (see above). Biopersistence is a characteristic of C8, and must be explicitly considered in determining faced by consumers of C8.

Finally, the RfD assumes that the control animals in the critical studies directly administered C8 in the study) had no exposure to C8. This is Control animals have been consistently shown to have significant liver levels of C8 (U.S. EPA 2002b, p. 48, 57-60) [Full document]. The fact RfD is based on studies with no true controls means that important to may have been overlooked because they were also caused by C8 in th contaminated control animals.

Conclusions

On September 27, U.S. EPA initiated a priority review of C8 because o concerns about its developmental and reproductive effects, and becau "additional blood sample analysis data indicate low level exposures to population that are unexplained at this time" (U.S. EPA 2002d, p. 1) [Full document].

The EPA's concerns focus on exposures in the general population, amc who face lower overall potential for exposure than do the West Virgini communities whose drinking water supplies have been directly contam C8 from manufacturing and disposal operations. The studies that insti EPA's priority review were available to DEP well in advance of its publi presentations.

In light of the federal government's position on the potential for C8 to reproduction and development, and its potential to cause cancer in hu appears that DEP's interpretation of C8's toxicity, and the drinking wa limits derived from that interpretation, fail to give the affected commu adequate margin of protection.

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APPENDIX A

WVDEP (C8 Assessment of Toxicity Team, or CATT APPROACH:

The approach used by WVDEP to calculate a drinking water screening is outlined below.

$$\text{Water Screening Level} = \frac{THQ \times AT \times BW}{EF \times ED \times [IR / RfD_{DEP}]}$$

THQ = Target Hazard Quotient, assumed to be 1

RfD_{DEP} = The oral reference dose estimated by DEP, 0.0042 (truncate mg/kg-day

BW = Body weight, assumed to by 70 kg for adults (children are not c in DEP calculations)

AT = Averaging time, 10950 days, the exposure duration expressed in

EF = Exposure frequency, 350 days/year, the average number of days people are exposed

ED = Exposure duration, 30 years, the average number of years peop exposed

IR = Ingestion rate, assumed to be 2 L/day for adults (children are no considered in DEP calculations)

So,

$$\text{Water Screening Level} = \frac{1 \times 10950 \times 70}{350 \times 30 \times [2 / 0.004]} = 0.146 \text{ mg/L}$$

U.S. EPA APPROACH:

The standard U.S. EPA approach for developing drinking water safety presented below. U.S. EPA incorporates a factor called the Relative So Contribution in cases of chemicals like C8 associated with multiple rou exposure. Calculations below also reflect the safety factor of 3 that DE from its final calculations that were originally in place to account for C persistence and long half-life in the human body. Both of these chemi characteristics contributed to U.S. EPA's decision to conduct a priority C8.

Adult Lifetime Drinking Water Health Advisory (DWHA):

$$DWHA = DWEL \times RSC$$

DWHA = 50 µg/L x 0.2 = 10 µg/L (ppb)

$$DWEL = \frac{RfD \times BW}{IR}$$

$$DWEL = \frac{0.0014(70)}{2} = 0.05\ mg/L\ (ppm)\ or\ 50\ \mu g$$

$$RfD = RfD_{DEP}/SF = 0.0042/3 = 0.0014\ mg/kg\text{-}day$$

DWHA = Drinking Water Health Advisory; "safe" dose of C8 via drinkir

DWEL = Drinking Water Equivalent Level; conversion of reference dos reference concentration

RfD = The oral reference dose that accounts for persistence and long l the human body

BW = Body weight, assumed to by 70 kg for adults, 10 - 15 kg for chi 5.2 for bottle fed kids

IR = Water ingestion rate; 2 L/day for adults and 1 L for children and infants.

RSC = Relative Source Contribution, which has a U.S. EPA default valu (0.2)

SF = safety factor to account for persistence and long half-life in the h (3X)

Bottle-fed Infant Drinking Water Health Advisory (DWHA):

In this case, we assume that a bottle-fed infant weight 5.2 kg and dri L /day, consistent with the scientific literature and U.S. Department of formula consumption data.

$$DWHA = 7.3\ \mu g/L \times 0.2 = 1.5\ \mu g/L\ (ppb)$$

$$DWEL = \frac{0.0014(5.2)}{1} = 0.0073\ mg/L\ (ppm)\ or\ 7.3\ \mu g/L$$

Drinking Water Health Advisory for a 10 or 15 kg (DWHA):

For comparison against the limit derived for an exclusively bottle-fed protective of a 10 or 15 kilogram child are presented below.

000013

For a 10 kg child:

$$DWHA = 14\ \mu g/L \times 0.2 = 2.8\ \mu g/L\ (ppb)$$

$$DWEL = \frac{0.0014(10)}{1} = 0.014\ mg/L\ (ppm)\ or\ 14\ ug$$

For a 15 kg child:

$$DWHA = 21\ \mu g/L \times 0.2 = 4.2\ \mu g/L\ (ppb)$$

$$DWEL = \frac{0.0014(15)}{1} = 0.021\ mg/L\ (ppm)\ or\ 21\ ug$$