



Center for Health, Environment & Justice

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EPA Docket Center
Environmental Protection Agency
Mail Code: 5305T, 1200 Pennsylvania Ave., NW
Washington, DC 20460

RE: EPA Draft Recommended Interim Preliminary Remediation Goals for Dioxin In Soil at CERCLA and RCRA Sites: Docket ID Number: EPA-HQ-SFUND-2009-0907

These comments are submitted on behalf of the Center for Health, Environment & Justice on the EPA's Draft Recommended Interim Preliminary Remediation Goals for Dioxin in Soil at CERCLA and RCRA Sites (Docket ID Number: EPA-HQ-SFUND-2009-0907).

CHEJ has been following and participating in EPA's activities on dioxin since 1994. We have submitted comments on the draft reassessment document released in 1994, 2000, and 2003. We have participated and submitted comments to the EPA's Science Advisory Board (SAB) when they reviewed the agency's draft reassessment documents in 1995 and 2001. We provided public comments to the National Academy of Sciences committee that reviewed EPA's 2004 draft reassessment document. We have published two significant reports on dioxin, *Dying from Dioxin* in 1995 and *The American People's Report on Dioxin* in 1999. We have contributed to the organization of two major citizen's conferences on dioxin in Baton Rouge, LA in 1996 and Berkeley, CA in 2000. We have contributed to the organization of numerous sign-on letters with communities impacted by dioxin, local, state and national environmental health, environmental justice, consumer, labor, parenting and health-affected organizations encouraging the agency to finish its work on dioxin and release the reassessment report. These and other dioxin related activities are briefly summarized in the timeline of dioxin events enclosed as Attachment #1.

I have presented papers at the Dioxin 1997 and Dioxin 2000 conferences that summarize many of these activities. I have also traveled to Russia with a delegation of American scientists and to Hanoi, Vietnam to present my experiences working with grassroots community organizations in the U.S. that have been impacted by exposures to dioxins and dioxin-like compounds. In my capacity as Science Director at CHEJ (since 1983), I have worked with hundreds if not thousands of grassroots communities where

exposure to dioxin was a major concern for the community. These communities include Superfund sites in Jacksonville and Pensacola, FL, Lock Haven, PA, Columbia, MS, Jacksonville, AR, and Times Beach, MO; other contaminated sites including New Bedford, MA, Missoula, MT, Quincy, WA, and Mossville, LA; communities impacted by waste incinerators including Columbus, OH, Hemtramck, MI, and Rosamond, CA. The experiences of these and other dioxin-impacted communities are summarized in our report published in 1998 *Standing our Ground*. The organized opposition of many of these grassroots community organizations is directly responsible for shutting down some of the worst incinerators and other sources of dioxin releases to the environment including the Columbus, OH garbage incinerator, the Miami “monster” garbage incinerator in Miami, FL, and the Occidental Petroleum garbage incinerator in Niagara Falls, NY. These groups took matters into their own hands because of inaction on the part of the government. This experience is the basis for the comments that we submit today.

We want to begin by commending the agency for moving forward to complete the dioxin reassessment document and release this report to the public. It is long overdue. We also want to commend the EPA for taking the initiative to develop interim preliminary remediation goals (PRGs) for dioxins that can be put into effect while the agency completes its review of the dioxin reassessment. This is an important step to protect the health of the American people from exposure to dioxins. We urge EPA to continue this process by revising the PRGs and finalizing them as remediation goals in a timely manner. In relation to the proposed PRG’s, we offer the following comments and recommendations. In these comments, we refer to dioxin and dioxin-like compounds as simply “dioxin.”

- 1) We agree that EPA should update the 1998 OSWER Guidance on PRGs.** The 1998 guidance is not consistent with the best available science on dioxin and needs to be updated as acknowledged in the Public Review Draft (PRD) document. We agree that the revised PRGs should take into consideration factors not considered in the 1998 guidance including non cancer effects, dermal exposure for residential use, and outdoor exposure for workers. In addition, the revised guidance should take into consideration a more protective cancer risk value. The 1998 OSWER guidance document only considers a cancer risk level of $2E-4$ (PRD, p. 13). Instead, we support the derivation of PRGs using a one-in-a million ($1E-6$) cancer risk level. These changes should be made in the derivation of the proposed recommended interim PRGs.
- 2) It is not clear why EPA chose to use only non-cancer effects as the basis for establishing PRGs for both residential use and commercial/industrial use.** The agency does not provide any reason for selecting non-cancer effects as the basis to derive the proposed recommended interim PRGs. The agency states that it believes that “these recommended interim PRGs generally provide adequate

protection against non-cancer effects (PRD, p. 13). EPA also states that it believes the proposed recommended interim PRGs “are generally protective for cancer effects at approximately the 1E-5 risk level, which is within EPA’s protective risk range of 1E-4 to 1E-6” (PRD, p. 13). No other explanation is given. The agency needs to provide its rationale for selecting non-cancer effects as opposed to cancer risk as the basis for establishing the proposed recommended interim PRGs. Without this information, it is not clear why it is better to use non cancer effects as opposed to cancer effects to derive the proposed recommended interim PRGs.

- 3) EPA must address dioxin’s classification as a human carcinogen in its derivation of the proposed recommended interim PRGs.** We are extremely concerned the EPA is proposing PRGs for dioxin that are based only on non-cancer health effects and does not adequately address the risk of developing cancer. This may result in unsafe PRGs since the PRGs derived to protect against a one-in-a-million cancer risk are lower (more protective) than those for non-cancer end points. Dioxin has been classified as a human carcinogen by the World Health Organization’s International Agency for Research on Cancer and the U.S. Department of Health and Human Services’ National Toxicology Program. The human epidemiological evidence provides consistent findings of increased risk for all cancers combined and lung cancer in occupational studies as well as evidence of tissue specific increases in cancer. Increased mortality from soft-tissue sarcomas and all cancers among workers exposed to dioxin has also been reported. Dioxin is also generally considered the most potent man-made carcinogen ever tested.

Although the agency “believes” the proposed recommended interim PRGs “are generally protective for cancer effects at approximately the 1E-5 risk level,” we do not agree that this risk level is sufficiently protective of public health, especially given dioxin’s potency as a carcinogen, as described in the previous paragraph. EPA should establish its PRGs using a cancer risk of one-in-a-million, or 1E-6, as required by the National Contingency Plan (NCP), the legislation that directs EPA to establish PRGs (see 40 CFR §300.430(e)(2)(i)(A)). The NCP requires the agency to use the one-in-a-million cancer risk value as the point of departure for determining PRGs. Given the strong evidence that dioxin (as TCDD) is a human carcinogen, the one-in-a-million point of departure should be the value the agency uses to establish the PRGs in order to adequately protect the public from exposures to dioxin in soil. The EPA should lower both the residential and commercial/industrial proposed recommended interim PRGs to reflect this requirement.

- 4) CHEJ supports the alternative PRG of 3.7 parts per trillion (ppt) for residential use and 17 ppt for commercial/industrial use.** For the reasons provided in Comment #3 above, CHEJ believes that one-in-a-million cancer risk value is needed to protect the health of the American public. As stated above, this value is consistent with the NCP and will be protective of non-cancer effects as well.
- 5) CHEJ disagrees with EPA that the alternative PRG values are in the range of background.** EPA states that PRGs based on a 1E-6 cancer risk would likely be within or possibly below background concentrations for dioxin in soil (PRD, p. 13). The agency cites a 2007 EPA report that found dioxin levels in soil that ranged from 0.2 to 11.4 ppt TEQ dioxin (PRD, p.13). The most recent draft of the dioxin reassessment gives the mean background value for dioxin in soil as 2.7 ppt TEQ for rural areas and 9.3 ppt TEQ for urban areas (see Table 3-15, Volume 2 of the 2004 NAS Draft of the Dioxin Reassessment). An analysis done by CHEJ of earlier estimates of dioxin background levels in soil found that many of the samples collected and analyzed by EPA to determine background levels were collected from areas where there were substantial sources of dioxin (see Attachment #2). Many of these samples were taken from areas that are NOT consistent with EPA's own definition of "background" which is defined as "an area where you would not expect to find any dioxin" (see EPA's National Dioxin Study, 1987). The level of dioxin in soil collected from areas consistent with this definition of background is likely less than 1 ppt according to our analysis. This analysis is enclosed as Attachment #2.

We have not analyzed the specific sources used to collect the samples in the 2007 EPA report cited in the Public Review Draft (p. 13), but we urge EPA to look carefully at the sources cited in this report and to determine first hand whether in fact there were sources of dioxin nearby to the places where the samples used to derive the "background" values cited in this report were collected. We suspect that these values were derived in a manner consistent with the earlier EPA reports which do not reflect true background values, but rather a level of dioxin found in urban or rural areas where there are sources of dioxin.

Furthermore, finding dioxin levels in soil in areas near dioxin sources says nothing about the health risks posed by the levels found and they should not be the basis for determining whether a health risk exists. If the EPA risk model using a 1E-6 cancer risk derives a PRG of 3.7 ppt for residential use and 17 ppt for commercial/industrial use, this risk analysis should then be trusted as reflecting a risk derived from the best available scientific methods accepted by EPA and other scientists as the gold standard of estimating risks. If the EPA is unwilling to propose recommended interim PRGs that are derived using this standard risk model, then it needs to reconsider this approach altogether for all chemicals, not just dioxin.

- 6) EPA's proposed recommended interim PRGs of 72 ppt TEQ for residential use and 950 ppt TEQ for commercial/industrial use are significantly higher than "background" levels and the cancer risk value of one-in-a-million.** In the most recent draft of the dioxin reassessment, the mean background value for dioxin in soil is 2.7 ppt TEQ for rural areas and 9.3 ppt TEQ for urban areas (See Table 3-15, Volume 2 of the 2004 NAS Draft of the Dioxin Reassessment). We are concerned that 72 ppt and 950 ppt are well above the one-in-a-million cancer risk value, as well as this background range. In the Public Review Draft document, EPA's proposed alternative value of 3.7 ppt TEQ is more in line with recognized "background" levels of dioxin. There is no reason why EPA should not aim to clean up dioxin contaminated soil to levels that are consistent with or slightly above background levels.
- 7) Many state agencies have set cleanup values comparable to EPA's alternative PRGs that are consistent with values derived from a one-in-a million cancer risk.** In the supporting document *Review of State Soil Cleanup Levels for Dioxin* (December 2009) released by EPA with the Public Review Draft on PRGs. 23 states are listed that have established unrestricted residential cleanup values for dioxin in soil. All of these states used cancer risk as the basis for establishing their soil cleanup level for dioxin. Twelve of these states used a one-in-a-million cancer risk (1E-6) value as the target cleanup goal. These cleanup values ranged from a low of 4 ppt to a high of 19 ppt. The mean value was 7.18 ppt. Eight states used a 1E-5 cancer risk value as the target cleanup goal with levels ranging from 20 ppt to 120 ppt. The mean was 61.1 ppt. Only one state (Hawaii) used a 1E-4 cancer risk value in setting their cleanup goal at 450 ppt. A total of 17 states currently have cleanup guidelines that are less than the EPA's proposed recommended interim value for residential use of 72 ppt. A decision by EPA to derive PRGs based on a 1E-6 cancer risk would be consistent with the decisions made by more than 50% of the states in the U.S. that have already set cleanup guidance levels for dioxin in soil. EPA should set PRGs for residential use and for commercial/industrial use that are consistent with most other state agencies that have already set cleanup guidelines. The agency may want to consider contacting these states to determine what if any problems any of these states may have run into using cleanup guidelines for dioxin in soil that range from 2 to 19 ppt.
- 8) EPA must strengthen the commercial/industrial cleanup goal for dioxin in soil.** The EPA is proposing an interim commercial/industrial PRG for dioxin at 950 ppt, significantly higher than the proposed "residential" level of 72 ppt. This level fails to protect workers and sensitive populations from exposure to dioxin, and does not adequately address dioxin's cancer risks. Moreover, these sites are not used exclusively by workers. These sites often become candidate sites for schools, parks, and recreational areas open to the public. It's not unusual for

land originally designated as commercial or industrial property to later become available to a school district interested in building a new school. The Love Canal disaster in Niagara Falls, NY is one such example. If such a site was contaminated by dioxin, it would only be remediated to 950 ppt, a far cry from what would be needed to protect children attending a school built on such property. There are no federal laws prohibiting schools from building on such land. Many remediated areas are also often used for parks and playgrounds, which again would result in hazardous exposures to young children and parents if the property was cleaned only to the proposed commercial/industrial PRG. In addition, the public has access to many commercial/industrial sites that can be sources of contamination to adjacent property. In rural areas, wildlife that may be hunted as a source of food may also have access to these sites and can become contaminated. For these reasons, EPA should lower the proposed recommended interim PRGs targeted for commercial/industrial use.

9) EPA must consider children's unique vulnerabilities to the harmful effects of dioxin. The proposed recommended interim PRGs should be protective of children, taking into account children's unique vulnerability to dioxin and their soil hand-to-mouth ingestion behaviors. It is not apparent that the exposure scenarios developed by EPA to derive the proposed recommended interim PRGs take into consideration this unique vulnerability. No uncertainty factor for this unique vulnerability is mentioned in the text or included in Table 3 of the Public Review Draft where the factors used to derive the proposed recommended interim PRGs are listed (PRD, p. 25). Children can be exposed to relatively large quantities of chemicals through normal developmentally appropriate pica behavior – the intentional ingestion of non-food items. Pica is estimated to occur in about half of children ages one to three. Because young children's breathing zones are so much closer to the ground, their exposure to contaminated soil via inhalation is more likely. Developing children are uniquely vulnerable to the toxic effects of chemicals due to an increased susceptibility and an increased risk of exposure; children's metabolic pathways, especially in the first months after birth, are immature; their ability to metabolize, detoxify, and excrete many toxicants differs from that of adults; they are less well able to deal with a number of toxic chemicals such as lead and organophosphate pesticides. We urge EPA to revise its proposed recommended interim PRGs by adding an additional safety factor/uncertainty factor that will take into account children's unique vulnerability to dioxin.

10) The exposure scenario used by EPA to compute non-cancer PRGs for exposure of residents to dioxin in soil is limited and does not take into account a lifetime of exposure. The factors used to derive the proposed recommended interim PRGs for residential use are included in Table 3 of the Public Review Draft (p. 25). This table is very helpful and EPA is to be commended for including this table in the document. This table shows that the PRGs for residential use derived from

non-cancer effects assume that exposure occurs only as child. EPA states that this “assumption is thought to be generally conservative since exposure to soil is higher than for an adult resident (PRD, p. 11). While this may be true in a relative sense, there is no reason to think that a typical child in this worst case scenario will not continue to be exposed to dioxin for more than six years. Older children will continue to play in dirt even if they do not directly ingest the soil through pica behavior. They will play in the soil and lick their hands and ingest more dirt than adults. EPA should revise this exposure scenario to include longer exposure periods for a child with adjusted ingestion rates. In addition, why does EPA assume that the child does not also have exposure as an adult? EPA should further revise its exposure scenario to include adult exposure for the pica child scenario to provide an estimated lifetime exposure.

11) EPA’s estimate of infant child soil ingestion through pica behavior is low and likely underestimates total ingestion. The EPA estimate of the soil ingestion rate for computing non-cancer PRGs for residential use assumes that infant children ingest 0.2 grams (gm) of soil per day through pica ingestion behavior (PRD, Table 3, p. 25). This estimate may not be protective enough. A 2004 review by Jacqueline Moya et al. of typical pica ingestion rates found that soil ingestion rates for children ranged from 39 to 271 milligrams per day (mg/day) with an average of 193 mg/day (see *Pediatrics* Vol. 113 (4) April). The upper percentile values ranged from 104 mg/day to 1,432 mg/day with an average of 358 mg/day for soil ingestion and 790 mg/day for soil and dust ingestion combined. A study in 1997 by Ed Calabrese et al. estimated that the range of soil ingestion by infants for a single event could be as high as 25 to 60 grams, substantially higher than EPA’s estimate of 0.2 grams (see *Environmental Health Perspectives* Vol. 105 (12) December). A soil pica workshop convened by ATSDR in 2000 concluded that “ATSDR should err on the side of being protective and should use 5,000 mg [as a daily soil ingestion rate] until more data are collected (see Summary report for the ATSDR Soil-Pica Workshop June 2000, prepared by Eastern Research Group March 20, 2001). EPA’s estimate is clearly a mid-range value that does not provide adequate protection for children who consume higher than expected amounts of soil through pica behavior. EPA should reconsider its estimate of soil ingestion for this analysis to reflect a higher rate of ingestion which would be more protective of the general population.

12) EPA’s relative source contribution (RSC) default factor is not accurate and needs to be re-evaluated. EPA assumes that 100% of the exposure for the child in their exposure scenario results from the pica ingestion of soil. Yet we know that this is not the true. We know that the national average dioxin contribution from diet is estimated to be more than 90% (PRD, p. 11). The pica child in the exposure scenario consumes dioxin in his/her diet no differently than any other person in the general public. What makes this person unique is that he/she has a second significant source of exposure - from pica behavior ingestion. While it

may not be clear what the contribution is from each of these two sources, it is unreasonable for EPA to assume 100% of the exposure results from pica behavior and none from dietary ingestion. EPA needs to rethink this exposure scenario and recalculate its RSC default factor to include contributions from both pica behavior ingestion and from dietary ingestion. Perhaps a 50-50 split would be reasonable.

13) EPA's exposure estimates for dioxin fail to consider contributions resulting from inhalation. We recognize that inhalation in general is the smallest contributor to the total daily intake of dioxin. However, there are some scenarios in communities where there are substantial air sources of dioxin such as downwind from a medical waste incinerator that this contribution may be greater. The agency should further examine an exposure scenario for communities such as this to ensure that the contribution from inhalation in these communities is in fact small.

14) We support EPA's use of the relative bioavailability (RBA) default factor of 1.0. It makes sense for EPA to define relative bioavailability of dioxin in soils as the ratio of the absorption of dioxin from soil to the absorption that occurred in the study used to derive the oral cancer slope factor or the oral reference dose for dioxin. It further makes sense to assume a relative bioavailability default factor of 1.0 to ensure protectiveness given the uncertainty of the bioavailability of dioxin in soil.

In closing, EPA must set health-protective PRGs for dioxin that are based on both cancer and non-cancer health effects. There is no justification for only using non-cancer endpoints. EPA needs to revise the PRGs to ensure that the cleanup goals for dioxin in soil take into consideration a cancer risk of one-in-a-million, as well as non-cancer effects. EPA should reevaluate its default factors for Relative Source Contribution (RSC) and the soil ingestion rate for residential exposure and take into consideration the special vulnerabilities of children when calculating the proposed recommended interim PRGs for residential use. The agency should also lower the proposed recommended interim commercial/industrial PRG to be more protective of workers and to protect children who would use these sites. Land originally designated as commercial/industrial property often later become sites for schools, parks, and recreational areas open to the public. These sites will need substantially more cleanup than would be provided by 950 ppt.

Thank your attention to this critical environmental health and environmental justice issue. We appreciate the opportunity to comment on this important matter.

Respectfully submitted,

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EPA's Dioxin Study – Over Twenty Years of Delays

1985 – EPA completes the first health assessment of dioxin, finding that the cancer risk to humans from dioxin exposure is by far the highest defined for any man-made chemical.

1986 – Dioxin found in paper products, due to chlorine bleaching.

1986 – The paper and chlorine industries pressure EPA to reconsider no threshold cancer risk model used by agency to establish “acceptable daily dose” of dioxin which industry claims is too low.

1986 – EPA Administrator asks staff to re-examine data and methodology used by EPA to derive 1985 cancer risk. First reassessment begins.

1986 – EPA sets up internal working group to review models for estimating cancer risk. Group cannot agree on best model to use, so they decide to average the risks predicted by different models.

1988 – EPA releases draft of first reassessment of dioxin that only addresses data and methodology used to estimate 1985 cancer risk.

1988 – EPA Science Advisory Board (SAB) criticizes working group for combining risks from different models but finds no new data to support changing the cancer risk estimate. EPA maintains its 1985 cancer risk estimate.

1990 – EPA and Chlorine Institute (later to become the Chlorine Chemistry Council) sponsor scientific conference on dioxins held at Banbury Center on Long Island, NY.

1991 – First Citizens Conference on Dioxin held in Chapel Hill, NC; organized to provide the public and grassroots activists with scientific information on the toxicity of dioxins.

1991 – EPA Administrator William Reilly announces EPA will conduct a new (second) reassessment of the health effects of dioxins.

1991 – NIOSH cancer mortality study of U.S. workers finds strong link between cancer and dioxin exposures.

1991 – Scientists report evidence that dioxin acts like a hormone, disrupting many systems in the body.

1993 – Researchers in Italy find increased cancer in residents living near pesticide plant in Seveso that exploded exposing thousands to dioxin in toxic cloud.

1994 – Second Citizens Conference on Dioxin held near Times Beach, MO.

1994 – EPA releases new draft reassessment report that confirms cancer risk estimate and finds that non-cancer effects may have greater impact on public health than cancer effects.

1994 – CHEJ kicks off the Stop Dioxin Exposure Campaign.

1995 – EPA Science Advisory Board completes its second review of EPA’s draft reassessment of dioxins and finds no major issues with report.

1996 – Third Citizens Conference on Dioxin held in Baton Rouge, LA.

1997 – IARC classifies dioxin (TCDD) as a human carcinogen.

1998 – The World Health Organization (WHO) reduces its daily tolerable intake (TDI) for dioxins.

July 1999 – 167 signers from communities impacted by dioxins, local, state and national environmental health, environmental justice, consumer, labor, parenting and health-affected organizations send letter to EPA Administrator Carol Browning demanding the release of the dioxin reassessment.

June 2000 – EPA releases revision of 1994 Reassessment Report. The revision finds even stronger links between exposure to dioxins and adverse impacts on human health. The EPA found the cancer risk to be 10 times higher than in the 1994 report.

August 2000 – Fourth Citizens Conference on Dioxin held in Berkeley, CA.

January 2001 – The National Toxicology Program concludes that dioxin (TCDD) is *known to be a human carcinogen*.

May 2001 – EPA Science Advisory Board completes third review of EPA’s draft reassessment of dioxins and recommends that the “agency proceed expeditiously to complete and release” the dioxin reassessment.

August 2001 – Nancy Pelosi and 40 Congressional Representatives write to EPA Administrator Christine Whitman urging the EPA to complete and release the agency’s reassessment of dioxin.

September 2001 – EPA announces it will send draft reassessment of dioxin to the White House’s Interagency Working Group for review.

February 2002 – Congressman James Walsh (R-NY) requests that EPA submit the draft reassessment of dioxin to a full review by a committee of the National Academy of Sciences.
April 2002 – Government Accounting Office (GAO) report supports scientific methods used by EPA in draft reassessment of dioxin.

July 2002 – Nancy Pelosi and 65 Congressional Representatives write to EPA Administrator Christine Whitman urging the EPA to complete and release the agency’s reassessment of dioxin.

February 2003 – A rider to the 2003 EPA appropriations bill add by Rep. James Walsh requires the National Academies to review the EPA’s reassessment if the White House’s Interagency Working Group does not come to consensus on the dioxin report within 60 days.

April 2003 – White House’s Interagency Working Group fails to come to consensus on the draft dioxin reassessment and supports request for the National Academy of Sciences to review the EPA’s reassessment of dioxin.

December 2003 – EPA releases revision of 2000 Reassessment Report and continues to conclude that there are strong links between dioxin exposure and adverse impacts on human health. This draft is sent to the NAS for review.

November 2004 – The National Academies holds first meeting of the Committee to Review EPA’s Exposure and Human Health Reassessment of TCDD and Related Compounds.

July 2006 – The National Academies releases a [report](#) confirming earlier studies that found dioxin to be a potent cancer-causing chemical.

October 2008 – Weeks before leaving office, President George W. Bush’s EPA orders the formation of another EPA Science Advisory Board to review the EPA’s response to the National Academies report.

January 2009 – Over 100 environmental health groups ask newly inaugurated President Barack Obama to stop former President Bush’s ‘last minute gift to the chemical industry’ of a further delay of the dioxin reassessment.

February 2009 – CHEJ and scores of environmental health groups call on EPA Administrator Lisa Jackson to release the Dioxin Reassessment.

April 2009 – [Health advocates and dioxin impacted communities](#) call on the EPA to release the Dioxin Reassessment Report and share their [stories](#).

May 2009 – Doctors, nurses and scientists around the country join CHEJ to urge the EPA to release the Dioxin Reassessment Report.
(<http://besafenet.com/pvc/documents/2009/letters/Dioxin%20Letter%20Nurses%20Doctors%20and%20Scientists%20FINAL.pdf>)

May 2009 – EPA Administrator [Lisa Jackson releases EPA’s Science Plan](#) for activities related to dioxin including its intent to release the final Dioxin Health Assessment by December 2010.

November 2009 – CHEJ submits comments on EPA dioxin cleanup goals plan.

December 2009 – The Chlorine Chemistry Division of the American Chemistry Council asks EPA to postpone the development of new Dioxin soil cleanup guidelines until the Dioxin Reassessment is finalized.

December 2009 – EPA misses deadline to release to the public its response to the NAS report, but does release Draft Recommended Interim Preliminary Remediation Goals for Dioxin in Soil at CERCLA and RCRA Sites.

Background Levels of Dioxin in Soil
Stephen Lester Science Director
January 1998

The USEPA has estimated the background level for dioxin in soil to be 8 parts per trillion (ppt) of Total Dioxin Equivalents (TEQ). This estimate was made in the 1994 draft reassessment document and is based on 95 samples "selected as representing background conditions in the United States." EPA did not, however, define or identify the specific locations of the samples they used to derive their background estimate. Upon reviewing the actual studies and the individual data cited by EPA, it appears that EPA's estimate of "background" dioxin in soil included data from testing conducted in urban and industrial areas where there are known dioxin sources.

The following is an analysis of EPA's data on dioxin levels in soil. This analysis is subject to several limitations: 1) It is not clear what specific data EPA used to generate the 8 ppt national background level in soil; and 2) Some data are reported as 2378-TCDD, some as "TCDD", and some as TEQ (Total Equivalent value which is the sum total of all forms of dioxin including the most toxic form - 2378-TCDD). This inconsistency makes direct comparisons of the data more difficult.

Given these limitations, a number of conclusions/observations can be made:

- Dioxin was not found in areas defined as pristine.
- Background is not defined by EPA in the 1994 reassessment document. EPA uses the term to indicate levels of dioxin currently found in soil and defined as "representative." EPA makes no effort to consider the contribution of dioxin from fallout over time from emission sources.
- A reasonable definition of background can be found in the 1987 National Dioxin Study where background is defined as "an area where you would not expect to find any dioxin."
- "Background" levels of dioxin in soil as described by EPA varied from one community type to another with a clear trend: Dioxin levels were highest in industrial areas and lowest in pristine areas with a clear gradient in between (see summary chart below).

Table 1 - Summary of "Background" Levels of Dioxin in Soil

<u>Type of Area</u>	<u>Dioxin Level</u>	<u>Form of Dioxin Measured</u>
PRISTINE	ND (1.0 ppt)	2378-TCDD
AGRICULTURAL	ND (3.75 ppt)	2378-TCDD
RURAL	<1 - 24 ppt	2378-TCDD/TEQ
RESIDENTIAL	6 ppt	TEQ
	158 ppt *	TEQ *
URBAN	22-67 ppt	TEQ
INDUSTRIAL	2,192 ppt	TEQ

* Samples from Midland, MI
 ND = Not Detected (detection level)

- In EPA's estimate of "background," they included data from studies in communities where there are known dioxin sources. For example, residential samples included data collected from Midland, MI, home of Dow Chemical Corp., a significant source of dioxin. In addition, the National Dioxin Study, conducted by EPA in the mid 1980's, only found positive urban background samples in Tacoma, WA, Lake Charles, LA, Gary IN, Pittsburgh, PA, Evansville, IN, San Francisco, CA and Washington, DC. Most of these areas are urban communities that contain significant industrial areas with known producers/sources of dioxin and thus are likely contaminated with dioxin.
- The highest numbers recorded in the "background" samples came from Midland, MI, the home of Dow Chemical, a significant source of dioxin in the U.S.
- The National Dioxin Study found an average mean value of 2.83 ppt of 2378-TCDD in 17 of 221 urban soil samples. When non detect values are included (204 of 221 samples), the average mean dioxin soil level is less than 1 ppt. Only 1 positive dioxin value was found in 138 samples taken in rural soil with an average mean value of less than 0.5 ppt 2378-TCDD (the limit of detection used on the sampling).
- Because "background" dioxin soil levels vary substantially from one community type to another, it may be difficult to define a single national background level.

- Because EPA has included data from communities with known dioxin sources, EPA has biased the data upwards. Their national background estimate of 8 ppt is therefore likely to be high.
- The true estimate of "background" dioxin in soil is likely to be less than 1 ppt.

More information on the specific studies and data used by EPA is needed in order to assess and determine an accurate and true background level of dioxin.

The data that EPA used to derive the 8 ppt background level of dioxin came from the following studies (full citations are listed at the end):

- 1) The 1987 National Dioxin Study which collected samples from all over the United States. These samples were analyzed primarily for 2378-TCDD (USEPA, 1987).
- 2) Samples collected from four Midwestern sites including Westlake, OH that were analyzed for a full range of dioxin congeners (USEPA, 1985).
- 3) Samples collected from industrialized areas in cities from Midwestern and Mid-Atlantic states (MI, IL, OH, TN, PA, NY, WV) that were analyzed for 2,3,7,8-TCDD only (Nestrick, 1986).
- 4) Samples collected from the vicinity of the Elk River, MN "generating station" that were analyzed for a full range of dioxin congeners (Reed, 1990).
- 5) Samples collected from around a sludge incinerator that were analyzed for full range of dioxin congeners (Pearson, 1990).
- 6) Samples collected from industrial, urban and rural sites in Ontario and some U.S. Midwestern states that were analyzed for full range of dioxin congeners (Birmingham, 1990).

The National Dioxin Study (EPA, 1987) reported the following summary of dioxin levels in soil (all data are reported as 2378-TCDD):

- 17 of 221 urban soil samples were positive with results ranging from 0.2 to 11.2 ppt
- 1 of 138 rural soil samples were positive with a result of 0.5 ppt

The 17 positive urban soil samples were broken out as follows (all results in ppt):

Washington DC	- 3.0	San Francisco, CA	- 2.0
	- 2.0	Tacoma, WA	- 0.4
	- 4.0		- 0.5
Pittsburgh, PA	- 5.0		- 0.6
	- 2.0		- 0.8
Evansville, IN	- 1.3		- 1.9
Gary, IN	- 0.5		- 8.7
	- 4.1		- 11.2
Lake Charles, LA	- 0.2		

- Average 2378-TCDD in urban soil: 2.83 ppt based on 17 positive samples
- Average 2378-TCDD in urban soil: 0.88 ppt when the 221 non-detect values are given a value of 0.5 ppt and combined with the 17 positive values.
- Average 2378-TCDD in rural soil: 0.50 ppt based on 1 positive sample
- Average 2378-TCDD in rural soil: 0.50 ppt when the 138 non detect values are given a value of 0.5 ppt and combined with the one positive value.

Combining rural and urban together:

- Average 2378-TCDD in soil: 2.70 ppt based on 18 positive samples
- Average 2378-TCDD in soil: 0.74 ppt based on 359 sample samples

Additional studies of dioxin in soil are discussed in the 1994 EPA reassessment document. The data from these studies are summarized in Table 2. The different forms of dioxin shown in this table are combined in Table 3 to show a single TEQ value for the same set of samples.

Table 2 - Data on Dioxin Levels in Soil from 1994 Reassessment

	Midland, MI		Middleton, OH		Elk River, MN		Ont/MW		MN	
2378-TCDD	59/62	55	6/22	<1	0/4	ND *	-	-	-	-
TCDD	5/7	109	0/5	ND	0/4	NA *	11/47	40	0/3	ND
PeCDD	2/6	37	0/5	NA	0/4	NA *	0/3	NA	0/3	ND
					1/4	10				
HxCDD 5/7	172	1/5	14		0/4	NA *	0/3	NA	0/3	ND
HeCDD 7/7	930	5/5	113		4/4	346	25/47	212	3/3	54
					1/4	4	3/30	5.4		
					2/4	9				
					4/4	48				
OCDD	7/7	4473	5/5	2418	4/4	1655	38/47	1599	3/3	54
							17/30	67		

Note: For each sample location:

First column - Number of positive samples/total number of samples

Second column - mean concentration in parts per trillion (ppt)

ND = Not detected; detection limit = 1.0 ppt unless marked with * which indicates detection limit = 3.75 ppt.

NA = No data available

Other data: Henry, IL (Ref 5) 1/13 samples found 2378-TCDD at <1 ppt

References for Table 2:

Midland, MI - USEPA, 1985 (2)

Middletown, OH - USEPA, 1985 (2)

Elk River, MN - Reed, 1990 (4)

Ontario, Canada/Midwestern States - Birmingham, 1990 (6)

Minnesota - USEPA, 1985 (2)

Note: numbers in "()" refer to reference number at end of report.

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Although it is unclear what 95 samples EPA selected as "representing background conditions in the United States," several points jump out at you when you look at the data in Table 2:

- Data from Midland, Michigan dominate the numbers. The highest numbers recorded in these "background" samples come from Midland, MI, the home of Dow Chemical, a large producer/source of dioxin in the U.S.
- One study looked only at industrialized areas (Ref 3) and another at soil from around an incinerator (Ref 5); neither area represent true background conditions.
- Some studies looked at all dioxin congeners (Refs 2,4,5,6), several looked only at 2378-TCDD (Refs 1,3) and others looked at "TCDD" (Refs 2,4,6). As a result, the data were collected using different methods and are difficult to compare.

If you take out the Midland, MI samples as not being "representative of background," the overall average dioxin level in soil drops substantially to less than 1 ppt TEQ (non detects reported in the data equal half the detection limit, which in most cases was 1 ppt). It is unclear, however, how many of the Midland samples were actually used by EPA to determine their background value of 8 ppt.

This estimate of less than 1 ppt is consistent with the estimate of the combined studies shown in Table 2 which separate the data from Midland from other areas. In addition, the average 2378-TCDD in 221 urban soil samples as determined in the National Dioxin Study which looked at "background sites that were not expected to have contamination" was less than 1 ppt. Based on these observations, it seems that background dioxin levels in soil, where background is defined to be "an area that is not expected to have contamination," is likely to be less than 1 ppt.

Table 3 - Summary of Data Reported by EPA in the 1994 Reassessment Document

	# Samples	Level (ppt) mean conc.	Form of Dioxin	Location
Pristine	4	ND (1.0) 0.6	2378-TCDD	Minnesota (2)
			TEQ	Minnesota (2)
Agricultural	4	ND (3.75)	2378-TCDD	Elk River, MN (4)
Rural	4	24	TEQ	Elk River, MN (4)
	30	0.12	TEQ	Ontario and Midwestern US (6)

Table 3 - Summary of Data Reported by EPA in the 1994 Reassessment Document (cont'd)

Residential	7	158	TEQ	Midland, MI (2)
	5	6	TEQ	Middletown, OH (2)
	1	<1	2378-TCDD	Henry, IL (2)
Urban	62	55	2378-TCDD	Midland, MI (2)
	47	67	TEQ	Ontario and Midwestern US (6)
	20	22	TEQ	Canada/US (3,6)
Industrial	67	2192	2378-TCDD	Midland, MI (3)
	1	1965	TEQ	Midland, MI (2)

References:

(1) National Dioxin Study, USEPA, Office of Solid Waste and Emergency Response, EPA/530-SW-87-025, Washington, DC, August, 1987.

(2) Soil Screening Survey at Four Midwestern Sites, USEPA, Westlake, Ohio: Region V. Environmental Services Division, Eastern District Office, EPA-905/4-805-005, June, 1985.

(3) "Perspectives of a large scale environmental survey of chlorinated dioxins: overview and soil data. Nestruck, TJ, Lamparski, LL, Frawley, NN, Hummel, RA, Kocher, CW, Mahle, NH, McCoy, JW, Miller, DL, Peters, TL, Pillepich, JL, Smith WE, Tobey, SW, *Chemosphere* 15: 1453-1460 (1986).

(4) "Baseline assessment of PCDDs/PCDFs in the vicinity of the Elk River, Minnesota generating station," Reed, LW, Hunt, GT, Maisel, BE, Hoyt, M, Keefe, D, Hackney, P, *Chemosphere* 21 (1-2): 159-171 (1990).

(5) "Concentrations of PCDD and PCDF in Ontario soils from the vicinity of refuse and sewage sludge incinerators and remote rural and urban locations," Pearson, RG, McLaughlin, DL and McIlveen, WD, *Chemosphere* 20:1543-1548 (1990).

(6) "Analysis of PCDD and PCDF patterns in soil samples: use in the estimation of the risk of exposure," Biringham, B. *Chemosphere* 20 (7-9): 807-814 (1990).

(7) "Estimating Exposure to Dioxin-Like Compounds, Volume II: Properties, Sources, Occurrence and Background Exposures," USEPA, Office of Research and Development, EPA/600/6-88/005Cb, External Review Draft, June