

September 10, 2002

W-99-18 NODA Comment Clerk  
Water Docket (MC-4101)  
U.S. Environmental Protection Agency  
1200 Pennsylvania Avenue, N.W.  
Washington, DC 20460

Re: Part 503 Sewage Sludge Use or Disposal Rule:  
Docket Number [MC-4101, OW-2002-0019]

On behalf of the undersigned individuals, we appreciate the opportunity to comment on EPA's Office of Water notice of data availability concerning dioxin, furan, and coplanar PCB limits in land applied sewage sludge.

**Summary of Comments:**

Dioxin is one of the most toxic substances on earth. While its prevalence in the environment in general is decreasing, scientists find increasing evidence of the severity of its damaging human health and ecological impacts. EPA's analysis shows that land application of sewage sludges is one of the most significant remaining sources of dioxin exposure in the U.S. and will likely be an even more significant source in the future as other sources come under control. The average body burden of dioxin in the U.S. is already at a level that poses a background cancer risk of 1 in 10,000 to the general population using the old cancer risk factor and 1 in 1,000 using the new cancer slope factor from the Dioxin Reassessment. 67 Fed. Reg. 40554, 40569 (June 12, 2002). Those levels of risk have generally been considered by EPA to pose an unacceptable risk. Current body burdens of dioxins also expose the U.S. populations to non-cancer risks that are so high that EPA has determined that it cannot even calculate a reference dose that would not involve "an appreciable risk of deleterious non-cancer effects during a lifetime." 67 Fed. Reg. 40569. EPA has a legal obligation not to add to those already unacceptable risks by authorizing the land application of sludges containing dioxins in such a way that they enter the food chain and increases human exposure.

In its current assessment of risks, EPA calculates the incremental cancer risk to the highly exposed individual from exposure to dioxins in land-applied sludge as high as 2.4 in 10,000. 67 Fed. Reg. 40569. Based on our analysis of EPA's risk assessment, we believe that incremental risk to be understated by EPA. EPA calculates the incremental deleterious non-cancer risk to the highly exposed individual to be an increase of 28% at the 99th percentile. 67 Red. Reg. 40570.

EPA must use the best scientific information that it has available in this rulemaking. Irrespective of whether the Agency formally issues its Dioxin Reassessment, the recommendations contained in that multi-year scientific study represent the best thinking of the Agency's scientists about the human and ecological risks associated with dioxins. EPA's failure to use the most recent science, as opposed to risk rates that have been found by EPA's own scientists and the scientific community to be out-of-date and inaccurate, would be arbitrary and capricious.

EPA's ecological risk assessment, which EPA describes as "a simple, efficient indicator of the potential for adverse ecological effects at a high-end exposure," shows that there is a reasonable potential for adverse ecological impacts on avian and mammalian species.

While the revised technical support document is a significant improvement over the previous risk assessment document, there are numerous non-protective assumptions and calculations. For example, various exposure routes are not considered including dermal exposure, simultaneous inhalation and breast milk ingestion for infants, and fish consumption from an on-farm pond. In addition, there is an inconsistent use of probabilistic and deterministic assumptions and some of the deterministic point estimates used are average values or values not reflecting a reasonable maximum exposure. Concentrations of dioxins in soils resulting from land application of sludges is a critical parameter. Due to assumptions regarding depth of incorporation into the soil and use of highly speculative models of erosion and dispersion, the calculated soil concentrations are low and would not represent a reasonably conservative (protective) estimate.

EPA is required under Section 405(d)(2)(D) of the Clean Water Act (CWA) to set minimum standards for sludge management that "shall be adequate to protect public health and the environment from any reasonably anticipated adverse effects of each pollutant." 33 U.S.C. 1345(d)(2)(D). EPA's human health risk assessment and ecological risk assessment document the need for regulatory controls to meet this legal standard.

To meet its statutory obligation in this rulemaking, we urge EPA to minimize the amount of additional dioxins to which the U.S. population is exposed from land applied sludges by: (1) setting protective numeric effluent limitations on dioxins (including furans and PCBs) in land applied sludges and in the soils to which sludges are applied; (2) disallowing land application above those numeric limits; (3) requiring frequent periodic monitoring to identify sludges that contain dioxins that exceed that limit; (4) requiring management practices, including prohibition of application to pastures, to lessen human health exposure to sludges in which dioxins are found, but have not yet exceeded the numeric effluent limitation; and (5) requiring each publicly-owned treatment works (POTWs) that produce sludges in which dioxins are found to have a pollution prevention component of its pretreatment program specifically directed at reducing the amount of dioxins in sludge.

In addition, EPA must meet its statutory obligation by regulating all pollutants in sludge that present reasonably anticipated adverse effects to human health or the environment. There are numerous additional chemicals present in sludges that must be assessed for risks posed to

human and environmental health. EPA needs to undertake an evaluation of current concentrations of chemicals in sludges from all available sources of data. Where a lack of data makes it infeasible to conduct a risk assessment, additional data needs to be collected. Particular attention must be paid to persistent bioaccumulative toxic compounds. For such chemicals, when a risk assessment cannot be conducted, the precautionary principle should be adopted.

More detailed comments follow on a number of these points:

### ***I. Human Exposure to Dioxin***

While the levels of dioxins in humans and in sludges have decreased in the past several decades, that decline seems to have ceased<sup>1</sup> (Commission of the European Communities, Council Regulation amending Commission Regulation (EC) No. 466/2001 setting maximum levels for certain contaminants in foodstuffs, 2001). A recent study from the University of Texas School of Public Health at Houston found no decline in dioxin in food from test samples taken more than a decade ago. The study found that through food alone, Americans are getting 22 times the maximum dioxin exposure suggested by the U.S. Environmental Protection Agency, and that among nursing infants, that level is 35 to 65 times the recommended dosage.<sup>2</sup>

Estimates of the sources of dioxins contributing to these background levels indicate that sewage sludges are one of the more significant sources and will likely be an even more significant source in the future as sources of dioxin other than land application of sludge are increasingly required to be controlled.<sup>3</sup>

Therefore, it thus makes sense to use means of sludge management that reduce the release of dioxins. Land application does not meet that criterion. It provides an opportunity for dioxins to move into the air, water, and food. Since it is often used on cropland and pasture used in raising livestock and since accumulation in animal products is one of the most significant pathways of exposure, agricultural use of sludges represents a significant continuing source of dioxin exposure. EPA must, therefore, establish rules for exposure to dioxins that minimize exposure from land application.

### ***II. Dioxins are Associated with Cancer in Exposed People***

The EPA's "Exposure Analysis for Dioxins, Dibenzofurans, and CoPlanar Polychlorinated Biphenyls in Sewage Sludge" (hereinafter "TBD") is clearly well-versed in the scientific state of the knowledge regarding the adverse effects of very low doses of dioxins on human health. And, yet, despite these acknowledged dangers, EPA is proposing to regulate dioxin in sewer

---

<sup>1</sup> Commission of the European Communities, Council Regulation amending Commission Regulation (EC) No. 466/2001 setting maximum levels for certain contaminants in foodstuffs, 2001.

<sup>2</sup> Study reported by Associated Press, March 30, 2001.

<sup>3</sup> Dioxin Reassessment, pp. 1-19-1-20, March 2001.

sludge so as to allow an incremental risk of cancer to the highly exposed population of as high as 2.4 in 10,000. 67 Fed. Reg. 40569. Using more up-to-date cancer estimates suggest that the risk is greater than one in 1,000. We find this unacceptable, and a violation of the EPA's mandate to protect public health and the environment.

Dioxin (2,3,7,8-TCDD) is listed as a Group 1 (known) human carcinogen by the International Agency for Research on Cancer (IARC; classified in 1997). Section 2.0 of the TBD, Table 2-1 cites a number of epidemiological studies in which occupational exposure to high levels of dioxins lead to elevated levels of cancer among workers (TBD, p. 2-4, 2-5). These important studies include:

- ? A cohort of over 5,000 US chemical manufacturing workers, with results indicating an increase in the most highly exposed group of workers, with exposures 100-1000X above background exposure levels (standard mortality ratio (SMR)=1.6, 95% confidence interval (CI) 1.15-1.82). (Steenland, K., et al., 1999.)
  
- ? A cohort of German phenoxy herbicide and chlorophenol production workers. The total mortality was elevated in all dose groups. The highest relative risk was observed for the highest 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) group (relative risk (RR) = 2.43, 95% CI 1.80 to 3.29). Cancer mortality and mortality due to ischemic heart diseases showed a dose-dependent relation with TCDD and all PCDD/F combined. The highest relative risks for cancer (RR = 3.30, 95% CI 2.05 to 5.31) and ischemic heart diseases (RR = 2.48, 95% CI 1.32 to 4.66) were observed in the highest PCDD/F exposure group. The pattern of effects and tests for trend were similar when the lowest exposure group within the chemical worker cohort served as the reference, but the relative risks were smaller and the confidence intervals were larger. The authors conclude that these findings indicate a strong dose-dependent relation between mortality due to cancer or ischemic heart diseases and exposure to polychlorinated dioxins and furans (Flesch-Janys, D., et al., 1995).
  
- ? A cohort of over 2,000 Dutch phenoxy herbicide and chlorophenol production workers. Male workers exposed to phenoxy herbicides or chlorophenols showed increased relative risks for total mortality (RR=1.8, 95% CI 1.2-2.5), cancer mortality (RR=4.1, 95% CI 1.8-9.0), respiratory cancer (RR=7.5, 95% CI 1.0-56.1), non-Hodgkin's lymphoma (RR=1.7, 95% CI 0.2-16.5), and ischemic heart diseases (RR=1.8, 95% CI 0.9-3.6) compared with an internal referent group of nonexposed workers. In general, relative risks were highest in the highest category, indicating exposure-related increases in risk with TCDD level. The authors conclude that results support the evidence of a high cancer risk in workers exposed to phenoxy herbicides, chlorophenols, and contaminants. (Hooiveld M., Heederick, D.J., et al., 1995.)
  
- ? An international study of over 18,000 workers from 12 countries exposed to phenoxy herbicides and chlorophenols. In a Poisson regression analysis, workers exposed to

TCDD or higher chlorinated dioxins had an increased risk for all neoplasms (rate ratio = 1.29, 95% CI 0.94-1.76) compared with workers from the same cohort exposed to phenoxy herbicides and chlorophenols but with minimal or no exposure to TCDD and higher chlorinated dioxins. The authors suggest that these findings indicate that exposure to herbicides contaminated with TCDD and higher chlorinated dioxins may be associated with a small increase in overall cancer risk and in risk for specific cancers. (Kogevinas M., Becher H., et al., 1997.)

TCDD is a multi-species, multi-site carcinogen in male and female experimental animals, in all species tested to date. This is acknowledged in the EPA TBD (p. 2-6). Moreover, the dose-response curve is remarkably similar for most species tested, for a given endpoint. Cancer is observed in both human epidemiological studies, and in animal studies, at exposure levels that are within 0-100X current body burdens, as acknowledged by the TBD (2-7, 2-8). This concern was stated multiple times in the March 12, 2001 Executive Committee Review Draft of the Office of Research and Development's Reassessment of Dioxin<sup>4</sup>. The EPA in fact, acknowledges this point, in choosing not to calculate a reference dose (RfD), stating that the resulting RfD would be below current background exposure levels (EPA, Sept. 2000). The EPA biosolids draft calculates the human cancer effective dose at the 1% excess risk level (ED01) to be within the range of 5.7-250 ng/kg, consistent with demonstrated effects in animal studies (TBD 2-8). This is easily within the range that Americans are currently exposed to from environmental sources. Yet, biosolids applied to agricultural lands have been shown to contain measurable levels of dioxins as high as 700 ng/kg, clearly posing an unacceptably high risk to human health (TBD 4-3). In fact concentrations in some sludges are even higher. Recent AMSA data on dioxins in sludges in the U.S. showed one sludge with extremely high concentrations of dioxins (3590 ppt). This was determined to be a real observation (not analytic or other error).

We urge the Agency to take very seriously the data it has compiled, and which is summed in part here. Although the human data is limited, the EPA SAB determined that it is compelling, and it is growing.<sup>5</sup> There is sufficient human data, and conclusive animal data, demonstrating elevated and certain risk of multisite cancer to even background levels of dioxins and PCB's. There is no safe level of exposure to these powerful and dangerous chemicals. Unlike many chemicals regulated by the EPA, there is little or no extrapolation needed to predict risk as "low" levels. EPA clearly acknowledges that there is ample evidence from animal studies as well as human data that background is at or close to doses where adverse effects are observed.<sup>6</sup>

---

<sup>4</sup> Op cit. SAB report. May 01, 2001, p. 40.

<sup>5</sup> May 01, 2001. Science Advisory Board Dioxin Reassessment Review Committee (DRRS). Executive Committee Review Draft #2.

<sup>6</sup> William H Farland, acting deputy assistant administrator for science. Memorandum to Donald Barnes, Science Advisory Board. Subject: Comments on dioxin reassessment review subcommittee draft, April 18, 2001.

To the extent that there is uncertainty, it should be reflected in a more protective standard. For example, in setting dioxin standards for land-applied sludge in Maine, the state used a one in a million health risk standard, noting that a very protective standard

is warranted given the uncertainties associated with extrapolating laboratory animal toxicity data to humans. In addition, the risk assessment model is unable to take into account the cumulative exposure of humans and wildlife to PCDDs and PCDFs from other sources (such as solid waste incinerators), as well as the very complex analysis of potential synergistic effects from other pollutants in sludges and residuals.<sup>7</sup>

### ***III. Non-Cancer Health Impacts Must Be Included***

The TBD does not address non-cancer endpoints resulting from exposure to dioxins, furans and co-planar PCBs (hereinafter “dioxins”) such as reproductive and development effects. These non-cancer impacts must be addressed and may be more limiting than cancer risks, especially when the outdated cancer slope factor is used. The EPA Scientific Advisory Board urged EPA to put more emphasis on these non-cancer impacts in their review of the Dioxin Reassessment document.

The TBD itself recognizes the importance of these non-cancer impacts. “Of the many adverse responses observed both in humans and experimental animals after exposure to 2,3,7,8-TCDD, the ones that appear at the lowest dose (more sensitive) are developmental/reproductive effects, alterations in the immune response, and neoplasia.” (TBD, p. 2-2). Yet these impacts are not considered in the risk assessment.

EPA has recognized the particular issues surrounding exposure of children and of fetuses to toxic chemicals. There is increasing evidence that even low dose exposure at critical development stages can cause permanent changes effecting behavior and health. “[A] spate of recent experimental and epidemiologic findings makes clear the exquisite sensitivity of prenatal and postnatal periods.” (Envir. Health Persp., v 109 (6) June 2001, editorial, Axelrod, D. D.L. Davis and L. A Jones.) It appears that exposure to even current background levels of dioxins may have implications for behavioral and other non-cancer responses to fetal exposure (Markowski, V.P., Zareba, G., Stern, S. et al.). Altered operant responding for motor reinforcement and the determination of benchmark doses following perinatal exposure to low-level 2,3,7,8-tetrachlorodibenzo-p-dioxin. Environ. Health Persp. 109(6):621-627.)

The Round 2 rules must take the non-cancer health impacts of dioxins into account in assessing risks posed by these chemicals in sewage sludges and must protect against all reasonably anticipated adverse non-cancer human health impacts.

---

<sup>7</sup> Bryce Sproul, Memorandum of September 17, 1986 (provided to NRDC by David Wright, Solid Waste Division, Maine Department of Environmental Protection).

***IV. Dioxins and PBC's Elicit Adverse Developmental Effects Even at Environmental (Background) Doses: Animal Data***

It is acknowledged by EPA, and by the Scientific Advisory Board, that evidence from human and animal data demonstrates that the general population has body burdens of dioxins and PCBs that are within 10-100X the levels that have been shown to cause cancer and non-cancer adverse effects. The additional exposure through sewer sludge represents an unacceptable risk, required to be avoided through responsible regulation by the EPA.

Exposure of laboratory animals during in utero development or lactation to maternal levels of dioxins that are at or near human body burdens in the general population result in adverse effects on the developing nervous, immune, and reproductive systems. (Birnbaum, L.S., Tuomisto, J., 2000; Gray, L.E., Ostby, J.S., et al. 1997; Gray, L.E., Wolf, C., et al., 1997; Mably, T.A., Bjerke, D.L. et al., 1992; Mably, T.A., Moore, R.W., et al., 1992(a); Mably, Moore, et al. 1992(b).) The animal data is unequivocal on this point; maternal dioxin levels within an order of magnitude of environmental levels will interfere with normal embryonic development. For example, a study by Mably et al, 1992 found such serious effects as altered genital structure and copulatory behavior of rats exposed in utero, following a single dose of 64 ng/kg (ppt) TCDD given to rat dams. (Mably, Bjerke, et al., 1992; Mably, Moore, et al., 1992a; Mably, Moore, et al., 1992b.) In fact, the EPA lists a number of multiple-dose studies to determine the range of body burdens presented in the dioxin assessment (EPA, Sept. 2000, Appendix I). Of the 104 endpoints (adverse outcomes) selected from the studies, EPA determined that 49 studies indicated an ED01 (effective dose 1%, the dose at which 1% of the subjects were affected) below 100 ng/kg. Even more concerning, 29 outcomes were associated with body burdens as low as 10-50 ng/kg. Further, adverse effects on development are also seen within the 10-50 ng/kg body burden range. The SAB felt that this information did not get the consideration that it deserves, given the public health implications of adverse effects at body burdens resulting from current environmental exposures.<sup>8</sup> We concur, and request that the EPA consider carefully in its regulation of sewer sludge the fact that Americans with high fat diets are already exposed to environmental (background) levels of dioxins and PCBs that pose an unacceptably high risk.

***V. Dioxins and PBC's Elicit Adverse Developmental Effects even at Environmental (Background) Doses: Human Data***

A wide range of non-cancer effects are found (Brouwer, et al., 1998). Non-cancer endpoints, especially developmental impacts through fetal and nursing infant exposures, are more sensitive end points than cancer where less than one-in-a-million cancer risk is used as cancer end point (data in U.S. Dept. of Health and Human Services, ATBDR, 1998; WHO, 1998; Dr. Henry Anderson, Wisconsin Department of Health and Social Services, personal communication). The immune system is a sensitive target for toxicity (U.S. Dept. of Health and Human Services,

---

<sup>8</sup> Op cit, SAB report. May 01, 2001, p. 29.

ATBDR, 1998). The risk assessment should include assessment of non-cancer risks to children, including fetuses. Sensitivity to toxicity of PCDDs may be greater during the fetal/neonatal period than for adults and may have an impact on male reproductive system development (U.S. Dept. of Health and Human Services, ATBDR, 1998).

Demonstrated adverse effects of dioxins and related compounds on human health include:

- ? A reduction in male sperm count, reduced sperm quality, and reduced testis function as demonstrated in studies from multiple countries worldwide. This would result in reduced fertility among men, and possible increases in developmental abnormalities resulting from dioxin-affected sperm (Damstra, et al., 2002).
- ? Adversely affected neurological development, neuroendocrine function, and behavior from prenatal exposure to PCB's has been shown in a number of human studies. Some of these effects result from altered thyroid or neurotransmitter function, or from developmental neurotoxicity (Damstra, et al., 2002).
- ? Alterations in sex ratios, resulting in less boys born to men with elevated TCDD levels has been demonstrated, following an explosion in 1976 in Seveso, Italy. Of most concern, the effects were obvious at body burden levels as low as 20 ng/kg, only 10-fold above background levels of men in industrialized nations (Mocarelli, P., et al., 2000).
- ? Disruptions in developmental neurobehavior is indicated by two mass poisoning events in Japan in 1968 (Yusho) and in Taiwan in 1979 (Yu-Cheng) in which 1-2 thousand adults were exposed to high levels of PCB's and other PHAH's through contaminated rice oil. Median PCB serum concentrations in exposed Yu-Cheng mothers were 26.8 ng/ml. Affected children of exposed mothers suffered growth retardation, movement disorders, and reduced IQ. In addition to neurobehavioral dysfunctions in the children, affected adults suffered central and peripheral nervous system problems, including headaches, memory loss, and hypoesthesia or neuralgia of the limbs (Musada, Y., 2001; Iida, T., et al., 1999).
- ? Precocious puberty demonstrated in girls who were exposed in utero to PBBs. In 1973 a flame retardant was accidentally added to livestock feed in Michigan. The meat and dairy products were contaminated with PBBs.<sup>9</sup> Rosen, D.H., Flanders, W.D., et al., 1995).
- ? Developmental behavioral impacts were found at a dose of 0.12 ppt/kg bw/day in rhesus monkeys and developmental effects are found to be among the most sensitive

---

<sup>9</sup> Id., Chapter 5, p. 68.

LOAEL in animals leading to a chronic oral MRL based on these effects (U.S. Dept. of Health and Human Services, ATBDR, 1998).

- ? Cognitive functioning in preschool children negatively impacted by in utero exposure to PCBs and dioxins (Patandin, et al, 1999).
- ? Birth weight and postnatal growth until 3 months of age impacted by in utero exposure to PCBs and dioxins (Patandin, et al, 1998).
- ? Immune suppressive and delayed reproductive effects are also a concern. A probabilistic risk evaluation of organochlorine exposure through breast milk showed that for a significant percentage of the population, PCBs and PCDD/PCDFs provide the greatest concern for non-cancer health effects from chemicals in breast milk (Hoover, 1999).

Given the extraordinary long half-life of dioxin and dioxin-like compounds, estimated for TCDD to be 20-50 years (TBD, p. 5-11), the developmental, reproductive, and immunological effects of dioxin must be quantitatively considered in any scientifically tenable assessment of dioxin exposure. Numerous studies demonstrate that dioxin can interfere with normal embryonic development at levels as low as 10-50 ppt (ng/kg),<sup>10</sup> thus justifying the EPA recommendation that the range of 10-50 ng/kg body burden be set as the “point of departure” for calculating non-cancer effects (EPA, Sept, 2000).

#### ***VI. Children, Infants, and Fetuses Are Most at Risk, and Are Highly Exposed***

- ? Farm children, nursing infants, and fetuses (pregnant women) should be deemed to comprise an especially vulnerable population, and their exposure to sewer sludge contaminants must be considered, where data are available. Further, children and infants who consume high fat diets, including breast-feeding infants, are exposed at particularly high levels, and are of increased developmental susceptibility, and therefore constitute an especially at risk population. There are over 12 million rural children, over 1 million farm children, and over 500,000 farm children under the age of six in the U.S. These children represent a high exposure subpopulation at greatest risk, for a number of reasons. Some chemicals accumulate in the body of the mother (e.g., dioxin) and are passed on to the fetus through the placenta and to the infant while breast-feeding.
- ? The fetal and breast-feeding infant exposure are directly the result of maternal body stores, and “profound toxic effects of dioxins are seen as a result of developmental exposure” (SAB, May, 2001, p. 30).

---

<sup>10</sup> Op cit SAB report. May 01, 2001, p. 29. See also SAB report, Appendix III.

- ? TCDD and related compounds are stored in body fat. About 60% of the structural component of the brain is lipid (fat), and at birth, the human brain is already 1/4 of its adult size, therefore being relatively much larger than the adult brain, per body size.<sup>11</sup> Therefore, fetuses, infants, and children will preferentially store fat-soluble chemicals, including dioxins and TCDD, in the developing brain. Simultaneously, the developing brain is uniquely vulnerable to toxic assault.
- ? The developing neural, immune, and endocrine systems are not only more vulnerable to toxic assault than the adult systems, but any damage to these developing systems is more likely to induce permanent damage, as changes become “hard-wired” into the system. This has been demonstrated for well-known chemicals such as mercury and lead, where doses that have no effect in adults will result in permanent brain damage, memory loss, learning disabilities, and behavioral changes in exposed children.

The legislative history of Section 405 of the Clean Water Act confirms that EPA is obligated to protect individuals and sub-populations that can be anticipated to have higher risks from exposure to dioxin in land applied sludge than the population as a whole. Senator Stafford, author of the provision, explained during floor debates on the 1987 amendments that “EPA’s rules must protect public health and the environment with an ample margin of safety, and must take care to protect the health of individuals or populations which are at a higher risk than the population as a whole.”<sup>12</sup>

### ***VII. Background Exposure Levels***

This TBD does not assume any background exposure and is only an assessment of incremental exposure from sludge application. The average body burden of dioxin in the U.S. is already at a level that poses a background cancer risk of 1 in 10,000 to the general population using the old cancer risk factor and 1 in 1,000 using the new cancer slope factor from the Dioxin Reassessment. 67 Fed. Reg. 40569. That level of risk has generally considered by EPA to pose an unacceptable risk.

A WHO panel recently completed a reevaluation of PCB/PCDD/F and determined that: 1) a revised TDI of 1 to 4 pg/kg body weight is established with 4 considered a maximum for long-term exposure on a provisional basis and 1 as a goal; 2) existing background exposures may be causing subtle effects at current intake levels of 2-6 pg TEQ/kg bw/day; 3) efforts should be made to limit environmental releases to the extent feasible (WHO, 1998; Brouwer, et al .1998). Thus in an industrialized country like the U.S., current exposure from background sources already put us at risk of unknown nature and extent. The TBD should include background exposure in the assessment.

---

<sup>11</sup> Op cit SAB report, May 01, 2001, p. 30.

<sup>12</sup> 132 Cong. Rec. S16427 (daily ed. Oct. 16, 1986).

Current levels of exposure to nursing infants exceed the 4 pg TEQ/kg TDI recommended by WHO (WHO, 1998) for nearly the entire population in Canada (Hoover, 1999) – and we would expect similar results for infants in the U.S. Thus background exposures cannot be ignored in assessing risks to children. Most standards are developed based on adult models. Some research has tried to improve on this by incorporating pharmacokinetic adjustments for infants. “The breast-fed infant’s intake of organochlorines has been found in general to exceed guidance values, raising the possibility that breast-feeding may pose health risks ....” (Hoover, 1999, p. 528).

### ***VIII. Incremental Risk Levels***

EPA calculates the incremental cancer risk to the highly exposed individual from exposure to dioxins in land-applied sludge as high as 2.4 in 10,000. 67 Fed. Reg. 40569. EPA calculates the incremental deleterious non-cancer risk to the highly exposed individual to be an increase of 28% at the 99th percentile. Based on our analysis of EPA’s risk assessment, we believe that incremental risk is likely understated by EPA as discussed more fully below.

In addition, EPA’s estimate of the annual cancer cases from exposure to land application of sludges is ludicrous.<sup>13</sup> First, it appears to assume that the cancer risk for all those except those who live on farms, raise their own crops and animals, and consume 50% of their animal diet from their farms is zero. That assumption ignores the cancer risk of 98% of the population. EPA then further assumes that the cancer risk of 99.8% of those remaining 2% of the population (i.e., those who live on farms, raise their own crops and animals and consume 50% of their animal diet from their farms) is also zero due to their estimate of the percent of farms to which sludges are applied. EPA has, therefore, calculated the cancer risk of only .0004% of the total U.S. population from exposure to dioxin in land applied sludge. Of course that number is small: it bears no relationship to the risk (even the incremental risk) borne by the population as a whole from the 8 million metric tons of sludge produced in the U.S. annually, the majority of which is land applied.

EPA’s failure to have any meaningful assessment of reduced cancer risks and its complete failure to assess regulatory benefits of reduced non-cancer risks or reduced environmental risks makes it nearly impossible to ascertain the potential benefits of promulgating regulations that would meet the statutory standard of protecting public health and the environment from any reasonably anticipated adverse effects. Because the statutory standard is specified, a cost-benefit analysis is not required and could not be relied upon by EPA to deviate from the statutory standard anyway. We do note, however, that EPA’s estimate of the nationwide cost of implementing these regulations has decreased since the time of proposal from the already

---

<sup>13</sup> “Estimate of Population Exposed to Dioxins From the Land Application of Sewage Sludge and Corresponding Number of Annual Cancer Cases from this Exposure (U.S. EPA, March 22, 2002).

modest figure of \$18 million to an even more modest \$13.7 million, much less than most of the proposed regulations contemplated by EPA.<sup>14</sup>

## ***IX. Comments on Incremental Risk Assessment***

Based on our analysis of EPA's risk assessment, we believe that incremental risk to be understated by EPA. While the revised technical support document is a significant improvement over the previous risk assessment document, there are numerous non-protective assumptions and calculations.

### **A. The Revised Technical Support Document/Risk Assessment is Not Reflective of the RME**

The new approach to assessing the risks posed by dioxins in sewage sludges embodied in the TBD is a significant improvement over the previous risk assessment. However, there remain a number of concerns with the method and its execution. The major flaw is that the assessment does not identify the scenario that represents a reasonable maximum exposure and risk. It correctly identifies the farm family as the receptor to be evaluated, but much of the analysis is based on assessing excess cancer risk to the average sludge-using farm family. The goal is to assess the risks to a reasonable maximally exposed person (RME). The TBD recognizes that farmers and their children who use biosolids on crops, pasture and eat home-produced foods are the RME. However, in the TBD calculations are made that often use average conditions or and thus are clearly not protective for farm families living in regions or employing practices or using sludges that are "more risky" than average.

The development and use of data based on regional variation among 41 U.S. regions is a novel and useful approach. Linking several parameters that vary from region to region and developing the risk analysis on these regional data, the TBD used a random distribution of possible outcomes based on the 41 climate regions. This does not provide for zeroing in on the risks posed to the reasonably maximally exposed receptor. Each climate region was equally weighted in the probabilistic analysis. Within the regional data, mean values were used for temperatures, runoff, winds, etc. For soil properties within each region, GIS maps were used and a regional average texture and properties were calculated. An area-weighted average slope was calculated.

Summing of risks across the different pathways is an appropriate revision to the previous approach, although for some unexplained reason infants are considered to be exposed only through breast milk and not through inhalation or any other pathway. This should be corrected since inhalation, while representing a relatively low exposure route for adults, may be higher for

---

<sup>14</sup> 64 Fed.Reg. 72045, 72051 (Dec. 23, 1999); Memorandum, Costs Associated with Regulating Dioxins, Furans, and PCBs in Biosolids (Office of Water, June 4, 2002).

infants given their greater respiration rate. This should be evaluated. In addition, a more holistic evaluation of the exposure of young children is needed (see below).

The dermal exposure pathway is not addressed at all in the TBD, nor in the EPA 1998 document. It appears that this path may be among the several more significant exposure routes when soil levels are the concern. It represented 8% of the contribution for farm family exposure as calculated in the useful evaluation of exposure scenarios to only 1 ppt in soil in the EPA Estimating Exposure to Dioxin-like Compounds (Table III-4, US EPA 1994).

The analysis does not include a farm pond used for fishing and swimming, but rather assesses an adult fishing in a 3rd order stream. Other EPA assessments suggest that on-farm ponds may be a source of farm family fish and should be evaluated (U.S. EPA 1998). Such a pond would likely be downslope of the sludged crop and pasture land and would have far higher concentrations of dioxins than the 3rd order stream considered in the TBD.

## **B. Inconsistent Use of Probabilistic vs. Deterministic Estimates**

The TBD makes use of a mix of probabilistic methods and point estimates. It is not clear why choices are made to use point estimates for some parameters that have significant variability and are important in calculating risks. An example is soil ingestion. When point estimates were used, it is not clear why choices were made to use central tendency values for most parameters and high end values for others. Performing a sensitivity analysis is a valuable addition. However, it is of limited use since sensitivity to some parameters that are important were not included (for example soil ingestion rate for people and for animals).

The TBD makes use of probabilistic analysis tools, but does so in an inconsistent way. For example, point estimates are used rather than distributions for a number of highly uncertain or variable and critical parameters such as duration of exposure, diet and soil ingestion. In Chapter 6, a number of tables present data for “deterministic analysis,” but the document does not describe the deterministic analysis that was performed. These tables list central tendency and “high end” values, but the meaning of high end is not defined. Why is the central and high end tendency for breast milk consumption the same? (Table 6-21). Table 6-27 lists 10 yrs as the central tendency adult duration data. This seems inconsistent with the EPA Exposure Factors Handbook data where the mean for farmers is 18 years as stated in the TBD (6-29). The large standard deviation and high variability (the high end estimate which pertains to 10% of the farm population, is 47 years), combined with the fact that the sensitivity analysis in the TBD showed this to be an extremely important variable in calculating risks makes it one for which probabilistic analysis should be performed.

A maximum of 20 applications over 40 years at a rate of 5-10 MT/ha is assumed. This is not consistent with the previous risk assessment in which 100 applications were considered. This assumption in the TBD would result in less exposure to dioxins.

### C. Concentration in Soils

Critical to the assessment of risks from a number of pathways is the soil concentration of dioxins resulting from the application of sludges. The TBD calculates this by using models for the loss and deposition via vaporization, erosion, leaching and degradation (assumed to be zero for dioxins). Applying the models, concentrations in soil are presented in table 5-4. The predicted concentrations are very low which is a result of the application of the models and the assumptions regarding sludge concentrations and application and management practices. The uncertainty inherent in application of the models and the use of many “average” values embedded in the assessment make these low values questionable.

Volatilization and leaching are thought by most researchers to be insignificant for dioxins (Fries and Paustenbach, 1990). Even following surface application of sludge, when volatilizations and photodegradation would be greatest, PCDD/Fs were found to be fully persistent (McLachlan et al., 1996). Various field studies show little or no loss of dioxins from soils (Orazio, et al. 1992; Hagenmaier, et al., 1992 b).

Use of models to predict key transfers of compounds through the agricultural ecosystem, particularly the air to plant transfer, is problematic for a number of reasons (Douben, et al., 1997). For some congeners of PCBs, model predictions of volatilization are quite accurate, while for others predicted fluxes underestimated by more than an order of magnitude (Cousins et al., 1999). Since there is significant question about the validity of the models, it has been suggested that use of measured concentrations is a more accurate approach (U.S. EPA, 1994). Such data indicate very low disappearance rates for dioxins. With a half life of over 20 years being realistic (McLachlan, 1997). Research on related chlorinated hydrocarbons further indicates that such compounds become more recalcitrant over time such that the concept of a half life obeying first order kinetics is inappropriate (Linz and Nakels, 1997).

A calculation of soil concentrations of dioxins on agricultural land to which sludge has been applied and where no loss through leaching, erosion or volatilization is predicted and where dilution via mixing into the soil is the only factor diminishing concentration would be an upper bound for soil concentrations. Table 5-4 shows the highest TEF in soil as the 95%ile at 5.9 ng/kg for pasture (mixing only into the top 2 cm is assumed). This is far from a value based only on dilution through mixing into the soil. While dilution alone may be overly conservative, the models used appear to overestimate losses.

Depth of incorporation of the sludge into the soil is an important variable since it is a significant determinant of the concentration of dioxins to which plants, animals and people are exposed due to dilution of sludge with soil. The TBD uses point estimates that have been revised from those used in the previous risk assessment. For an undisclosed reason, point estimates are used in TBD rather than treating it as a variable parameter. For pasture an incorporation depth of 2 cm is assumed, a significant improvement over the previous risk assessment. For crop land, incorporation is estimated at 20 cm, up from 15 cm in the previous TBD, despite a peer

reviewer's comment on previous dioxin risk assessment for sludge that suggested that 10 cm would be more appropriate than 15 cm. Tilling to a depth of 20 cm "multiple times during the year" (TBD 4-3) is assumed for cropland. This larger tillage depth and multiple plowing would overestimate incorporation for many situations where no-till or other less intensive tillage is practiced. That in turn would underestimate the concentration of dioxins in cropland soils.

Calculation of the concentration of dioxins in the buffer area is an important parameter. In the TBD, the concentration in the buffer is based on models of erosion and runoff. These are in turn based on various regional values. While useful for calculating average conditions, the relevance to assessing risks posed in areas where slopes may be higher than average and sludge may be applied under conditions that favor runoff – the reasonable maximum exposure – is not clear. It would have been useful to do a sensitivity analysis to see just how significant the concentration of dioxins in the buffer zone is since it is used in several pathways. Several values for dioxin concentrations in the buffer, cropland, pasture and sediment soils are listed (Table 5-4), but which are used in calculations or whether the analysis used a probabilistic approach is unclear.

#### **D. Underestimated Exposure of Livestock**

Since bioaccumulation in animal products is a critical exposure parameter, it is important to scrutinize the assumptions that went into assessing that pathway. The TBD used values for agricultural practices, duration of sludge use, etc. that were selected "to be consistent with common agronomic practices." (TBD 4-1). These were assumed to be the same throughout the U.S. (not to vary regionally). Some of these are clearly not protective in some regions where practices are different. For example, why only 8% of the dairy cow diet is assumed to be forage from pasture is unclear and is not reflective of conditions in many parts of the U.S. In New York, for example, a more reasonable estimate of average conditions would be 60-70% (personal communication, Dr. Larry Chase, Cornell University Department of Animal Science). There is no discussion or rationale in the TBD to justify this very low pasture consumption rate.

Another critical component of the pathway leading to accumulation of dioxins in animal tissue is the percentage of the animal diet that is soil/sludge. The estimate of 2% of dairy cow diet as soil from pasture in the TBD is also on the low side, though higher than the 1.5% estimate used in the previous TBD. The assumptions made regarding the amount of soil ingested by grazing animals seems to be based on best management practices. Grazing cattle ingest from 1-18% of their dry matter intake as soils and sheep may ingest as much as 30% depending upon management and the seasonal supply of grass (Fries, 1996; Thornton and Abrahams, 1981). One or 2% intake was a low value obtained when availability of forage was greatest in the spring and an average yearly intake of 6% for cattle was observed when exclusively pasture-fed (Fries, 1996). Other researchers use estimates such as 6% of diet as soil and point out the critical importance of assessing the amount of sludge ingested through material adhering to vegetation – and the lack of data on appropriate values for that variable (Wild, et al, 1994). A previous EPA document suggests 4% or 0.5kg/day soil ingestion by grazing beef cattle and 3% or 0.4kg/day for dairy cows (U.S. EPA, 1998).

When sludge is spread on pasture or growing crops, a significant amount ends up adhering to the leaves of plants. This residue is not easily removed by rainfall. Thus it is likely to be an important route of exposure to pastured animals and also to animals eating harvested forage (Wilson, et al., 1997). The TBD does not seem to account for this exposure. The amount of sludge ingested due not to uptake or soil ingestion but to ingestion of sludge adhering to plants needs to be included in the assessment and this material would contain the full concentration of pollutants contained in the sludge and not be diluted by any soil mixing.

It is not clear how the TBD does or does not take into account the fact that significant amounts of soil are contained in harvested field crops, with several percent soil being estimated in harvested grass silage (Berende, 1990 in McLachlan, 1997). For grasses, it is estimated at 100 mg soil g<sup>-1</sup> grass DW (Smith and Jones, 2000). This soil which “tags along” with harvested forage needs to be included in the assessment of risks. Calculations of the contribution of particulate contamination accounted for 30% of the total PCDD/Fs found in grass containing a conservative background estimate of 20 mg soil g<sup>-1</sup> grass DW, while soil was responsible for a majority of the pollutant load under a worst case situation of 100 mg soil g<sup>-1</sup> plant DW (Smith and Jones, 2000). PCBs were less related to soil load.

#### **E. Other Issues**

In calculating the concentration of dioxins in plants, the current TBD has included the contribution of particulate deposition which is an improvement over the previous risk assessment. It is not clear, however, how the particulate deposition term is calculated.

The TBD is unclear about whether “field” or “farm” acreage is being used in calculating runoff and air emissions. The body of the document says “the agricultural field area was assumed to be the median area for farms in each climate region. The agricultural field sizes used in this analysis are presented in Table 4-2.” (4-4). However, the legend for Table 4-2 is “median Farm Size for each climate region.” The assumption is also made that this land is half crop and half pasture.

Groundwater is not considered as a significant exposure path. However, in regard to the groundwater pathway, the role of facilitated transport of dioxins bound to organic matter needs to be investigated. Sorption onto organic matter may give rise to the facilitated transport of these compounds into ground water (Nelson, et al. 1998). This is a particular concern as complexation of hydrophobic chemicals with organic matter can also inhibit the ability of microorganisms to degrade these compounds even though they may still be available and therefore toxic to higher organisms (Rinella, 1993).

A significant improvement over the previous TBD was the inclusion of adult ingestion of soil. A point estimate of 50mg/day was used, although this is a highly uncertain value. More reasonable would be to use a distribution for both child and adult soil ingestion and to assess the sensitivity

of the results to these values. The assumption is that the ingested soil for a child is from the buffer zone and thus has sludge only from runoff and airborne deposition. This is a very major change from the previous 503 assessment in which the soil ingestion pathway was limited only to children and the soil was assumed to be 100% sludge. For the adult, the assumption is that the soil is from the crop land which has a lower concentration than soil in the buffer. Thus the TBD seems to minimize assumed soil concentration for adult exposure while in fact the adult would be working the garden and living in the buffer, working directly with the sludge and otherwise would be likely to be exposed to higher, not lower, concentrations.

#### ***X. Environmental Risk from Dioxin in Land Applied Sludge***

EPA's ecological risk assessment, which EPA describes as "a simple, efficient indicator of the potential for adverse ecological effects at a high-end exposure," shows that there is a potential for adverse ecological impacts on osprey, American robins, belted kingfishers, and mink from biosolids with average as well as high end dioxin levels in sludge. EPA indicates that it considered a hazard quotient of greater than one to be above the protective ecological benchmark, 67 Fed. Reg. 40570, and the hazard quotient for these species ranged from a low of 2 (osprey at 50th percentile dioxin TEQ) to a high of 209 (mink at maximum TEQ dioxin in biosolids). TBD, 9-28.

Moreover, EPA's own assessment of the risk of dioxin in land applied pulp and paper sludges estimated average hazard quotients of greater than one for mammals and birds based on exposure to soil concentrations of dioxin in sludge averaging 14 ppt on pasture land, 6.7 ppt on row cropland, and 4.2 ppt on silviculture land (Meyn 1997). The expected risks were highest for mammals that eat insects, earthworms, and other ground-dwelling invertebrates. For example, an average risk quotient from exposure to dioxin for shrews was calculated to be 200 (or 200 times the ecologically protective benchmark rate). Id.

Similarly, an ecological risk assessment prepared by Abt Associates, Inc. for EPA in 1994 on risks to terrestrial wildlife from dioxins in paper mill sludge found risk quotients exceeding one under high-end exposure scenarios for each of the bird species modeled on every site, which included Cooper's hawks, red-tailed hawks, and eastern bluebirds, as well as for the masked and southeastern shrew (Abt, 1994). These risk quotients were based on dietary exposure alone and do not include dermal and inhalation exposures. Therefore, they likely underestimate the total risk to terrestrial wildlife. Id. The study concluded:

This analysis determined that pulp and paper sludge land-application practices can lead to chronic individual risks (e.g. mortality, growth, reproductive, etc.) to terrestrial wildlife. The species particularly at risk are those that consume large amounts of food that bioconcentrates TCDD or TCDF from the soil or that bioaccumulates TCDD or TCDF from contaminated prey items.... This analysis determined that there are a significant number of scenarios (iterations of model) that are expected to lead to risks to each of the representative bird and mammal species in this analysis.

Id. Section 4.3. Based on its analysis, Abt identified a reference soil concentration for the protection of the masked shrew at agricultural, mine-reclamation, and silvicultural sludge application sites of 0.08 ppt, for the Cooper's hawk in silvicultural sludge application sites of 6 ppt, and for Cooper's hawk embryos of 3 ppt.

EPA should also include the specific endpoint that the HQ is based upon. In addition, EPA should describe whether any correction factors were applied when an HQ was based on mortality rather than a more subtle index of toxicity. For example, in birds TCDD exposure is known to cause both adult and developmental mortality, decrease hatchability and growth (Janz, 1996), decrease egg-laying (Nosek, 1992), delay onset of egg-laying in offspring whose mothers were exposed to 8.6 ng/day (Alonso, 1998), and cause structural malformations (Peterson, 1993).

The screening ecological risk analysis (SERA) ignores aquatic species entirely because adequate data were identified only for mammals and birds. (67 Fed. Reg. 40571). A comprehensive ecological risk assessment must include aquatic species, which are often the most sensitive to chemical exposure. Two recent studies highlight this sensitivity. In one, the lowest dose tested of TCDD (1.8 ng/kg) was found to cause mortality in adult female trout and increase both mortality and incidence of deformities in their offspring following chronic exposure (Giesy, 2002). Another recent study found that chronic exposure to a low dose of TCDD (2 pg/L TCDD for 120 days) causes cellular toxicity to hepatocytes in the Chinese rare minnow (Wu, 2001).

***XI. EPA is Legally Obligated to Protect Human Health and the Environment from Any Reasonably Anticipated Adverse Effects of Land Application of Sludges***

EPA is required under Section 405(d)(2)(D) of the Clean Water Act (CWA) to set minimum standards for sludge management that "shall be adequate to protect public health and the environment from any reasonably anticipated adverse effects of each pollutant." 33 U.S.C. 1345(d)(2)(D). EPA's human health risk assessment and ecological risk assessment document the need for regulatory controls to meet this legal standard.

EPA has a legal obligation not to add to the already unacceptable risk posed by background level exposures to dioxin in the U.S. population by authorizing the land application of sludge containing dioxins. Current body burdens of dioxins also expose the U.S. populations to non-cancer risks that are so high that EPA has determined that it cannot even calculate a reference dose that would not involve "an appreciate risk of deleterious non-cancer effects during a lifetime." 67 Fed. Reg. 40569. EPA has a legal obligation not to add to those already unacceptable risks by authorizing the land application of sludge containing dioxins in such a way that it enters the food chain and increases human exposure.

## **A. EPA Has Traditionally Used a 1 in 1 Million As Its Acceptable Risk Level for Regulation**

EPA has characterized the risks of cancer due to dioxin-like chemicals in the environment to be about 1 in 1,000 for the average American population on a high animal fat diet (EPA, Sept, 2000). We consider this to be an unacceptably high risk and adding to it unjustifiable given the plethora of scientific data indicating both the devastating health and environmental effects of dioxins and PCB's, and the understanding of the sources of exposure (TBD, sections 5.0, 6.0). Traditionally, the EPA has used a 1 in 1 million risk as acceptable to set regulatory standards. We believe that the EPA has both the proof required to act, and the data demonstrating how to act effectively to further reduce human and wildlife exposure to these dangerous chemicals. There is no justification for setting less stringent standards in sewer sludge, which adds to the health risks of millions of Americans.

The EPA is required under the CWA Section 405(d)(2)(A) to use management practices and numerical criteria in establishing limits for each pollutant which, on the basis of available information on their toxicity, persistence, concentration, mobility, or potential for exposure, may be present in sewage sludge in concentrations which may adversely affect human health or the environment. 33 USC §1345(d)(2)(A). These limits must be set at a level that is adequate to protect human health and the environment from any reasonably anticipated adverse effects of each pollutant. 33 USC §1345(d)(2)(D). This level should be set at no greater risk than one-in-one million.

The EPA has generally regarded an adverse effect as one that results in one-in-one million as the acceptable level of risk. This is the level that should be adopted in protecting the public health for the danger posed by the land application of dioxin containing sewer sludge. To do otherwise would be inconsistent with other agencies, other EPA programs, other Clean Water Act programs and other risk levels specific to dioxin exposure that are contemplated by the EPA. There is no rational basis for EPA to set the risk level here higher than the one in one million level. Such an arbitrary action would be an abuse of the Agency's discretion.

Other federal agencies have adopted the one in one million is level of risk. For example, 50 Fed. Reg. 51551, 51557 (1985) states that the "[Food and Drug Administration] FDA cannot, with assurance, state that the 1 in 100,000 level would pose an insignificant level of risk of cancer to people. FDA can state, and comments agree, that the one-in-one million level represents an insignificant level of risk of cancer to people."

Examples of the one-in-one million standard within the EPA are plentiful. For cancer risks to humans, under the Resource Conservation and Recovery Act (RCRA), one-in-one million, over a lifetime, is the risk target.<sup>15</sup> The Clean Air Act, section 112, establishes a one in one million

---

<sup>15</sup> See, e.g., 59 Fed. Reg. 9808, 9817 (1984) (using one incremental cancer risk in a million based on lifetime exposure to determine health-based level for carbamate wastes).

annual per-capita risk as the threshold for acceptable risk. 42 U.S.C. § 7412(f)(2)(A). “If standards promulgated pursuant to subsection (d) and applicable to a category or subcategory of sources emitting a pollutant (or pollutants) classified as a known, probable or possible human carcinogen do not reduce lifetime excess cancer risks to the individual most exposed to emissions from a source in the category or subcategory to less than one in one million, the Administrator shall promulgate standards under this subsection for such source category.”<sup>16</sup> This limit of risk has been clearly established by the EPA over a wide range of programs. EPA must have a compelling justification for departing from this established risk level.

Within the Clean Water Act, the EPA’s 2000 Human Health Methodology states that, for the purpose of Section 304(a) of the Clean Water Act, water quality criteria use an incremental cancer risk criteria at a 10<sup>6</sup>-risk level for the general population.<sup>17</sup> Here, the Agency is required to protect sensitive populations and individuals, not just the general population from reasonably anticipated adverse risks. Therefore, the one-in-one million level of protection should be used by EPA in this rule to protect more sensitive populations, like farm families, rural children, and so forth. The Agency offers no justification for using less protective criteria in this regulation.

The EPA has also specifically addressed the appropriate level of risk that is to be associated with dioxin. EPA has stated that it would use the 2000 Human Health Methodology in developing Ambient Water Quality Criteria (AWQC) for the protection of human health including development of revised AWQC for “pollutants of high priority and national importance (including, but not limited to chemicals that bioaccumulate, such as PCBs, dioxin, and mercury).”<sup>18</sup> This implies that the EPA would use the one-in-one million-risk level in developing these criteria for risk from dioxin exposure in ambient water, as this is the level that is described as acceptable in the 2000 Human Health Methodology. There can be no justification for the EPA, in carrying out its legal obligation to protect human health and the environment under Section 405(d)(2)(D) of the CWA, to set an acceptable level of incremental cancer risk for dioxin exposure at a level that puts the public at greater risk than contemplated as being appropriate for exposure to dioxin in the water column. The EPA’s duty to protect public health under section 405 is at least as great as its obligation in setting these AWQC. To adopt a different level of risk from exposure from land application of dioxin containing sludge than exposure to dioxin elsewhere in the environment would be arbitrary and an abuse of the EPA Administrator’s discretion.

## **B. EPA Must Use Up-to-Date Scientific Information on Risk**

---

<sup>16</sup> See also National Research Council (1994), *Science and Judgment in Risk Assessment*, NRC Committee on Risk Assessment of Hazardous Air Pollutants, Board on Environmental Studies and Toxicology, Washington, D.C: National Academy Press.

<sup>17</sup> Revisions to the Methodology for Deriving Ambient Water Quality Criteria for the Protection of Human Health, 65 Fed. Reg. 66444, 66447 (Nov. 3, 2000).

<sup>18</sup> *Id.* at 66448.

The TBD makes use of recent data developed as part of the Draft Dioxin Reassessment Document (U.S. EPA, 2000). The Dioxin Reassessment draft represents an enormous effort undertaken by EPA to make use of the most current science to assess the impacts of dioxins on human health. While the TBD appropriately uses the information compiled and synthesized in the Reassessment, it does not use the cancer slope factor in that Reassessment citing the fact that the document has not been officially adopted by EPA, despite approval of the Scientific Advisory Board.

The cancer slope factor used in the TBD is  $1.56 \times 10^5$  (mg/kg-d)<sup>-1</sup> which is an out-of-date figure. The new cancer slope in the draft Reassessment document estimates a cancer risk approximately 6-fold higher. While not yet officially adopted by EPA, the analysis of risk provided in the Reassessment is reflective of current science. Recent papers support this newer risk estimate (e.g., Becher et al., 1998). The agency must make use of the best and most current science in developing the Round 2 regulations. Using an outdated cancer slope factor is not acceptable.

As to be expected, use of different assumptions in a risk assessment process leads to different standards. Using a risk assessment process, the state of Wisconsin evaluated dioxin risks from paper sludge land application and derived a cancer-based standard of 1.2ppt in soil associated with a one-in-a-million risk and a value of 0.19 ppt where grazing is allowed (Goldring, 1992). If a higher allowable cancer risk of one-in-ten-thousand is accepted by EPA in the sludge rules (which we do not accept), acceptable levels based on cancer risk as evaluated in the Wisconsin assessment would be 19 ppt in soils where grazing is allowed.

As discussed with respect to ecological risk above, even more protective soil standards are necessary to protect against adverse environmental impacts from dioxin exposure in land-applied sludge. The soil level necessary to protect against harm to the most sensitive species, the masked shrew, is 0.08 ppt. Because alternative measures, like banning application of sludge on grazing land, will not protect against this adverse environmental impact, strict limits on the amount of dioxin that is allowed to accumulate in the soil must be set.

### **C. EPA is Obligated to Regulate All Pollutants in Sludge That Have Reasonably Anticipated Adverse Effects on Human Health or the Environment.**

The limitation of this Round 2 regulation only to dioxins is not acceptable. There are numerous additional chemicals present in sludges that must be assessed for risks posed to human and environmental health. EPA needs to undertake an evaluation of current concentrations of chemicals in sludges from all available sources of data. EPA's continuing obligation to review current sludge regulations and to identify additional toxic pollutants that warrant regulation adequate to protect public health and the environment against any reasonably anticipated adverse effects is found in Section 405((b)(2)(D) of the CWA. 33 U.S.C. 1345(b)(2)(D). Where a lack of data makes it infeasible to conduct a risk assessment, additional data needs to

be collected. Particular attention must be paid to persistent bioaccumulative toxic compounds. For such chemicals, when a risk assessment cannot be conducted, the precautionary principle should be adopted.

### **Recommendations**

To meet its statutory obligation in this rulemaking, we urge EPA to minimize the amount of additional exposure to which the U.S. population is exposed from land applied sludges by taking the following actions:

- 1) Setting a numeric effluent limitation on dioxins (including furans and PCBs) in land applied sludges at a level that would protect public health and the environment from any reasonably anticipated adverse effects of each pollutant. This standard must be set at a level that protects the highly exposed population at a risk of no more than one in a million additional cases of cancer. It must also protect against non-cancer impacts to that population at the same risk level. Furthermore, exposure to dioxin in sludges must be kept to an absolute minimum given the high risks to which the population is already exposed from current background levels of dioxin in the environment.
- 2) Banning land application to sites where dioxin levels in the soil exceed levels that would protect public health and the environment from any reasonably anticipated adverse effects of each pollutant. EPA's own analysis of ecological risk suggests that soil levels of less than 1 ppt are needed to protect against adverse effects to the most sensitive mammalian species. These risks cannot be alleviated by the types of management measures, like banning application to pasture lands, that would reduce human health risks.
- 3) Requiring frequent periodic monitoring to identify sludges and soils containing dioxins that exceed those limits.
- 4) Requiring management practices to lessen human health exposure to sludges in which dioxins are found, but have not yet exceeded the numeric effluent limitation, including prohibition of application to pastures.
- 5) Requiring each publicly-owned treatment works (POTWs) that produces sludges in which dioxins are detected to have a pollution prevention component of its pretreatment program specifically directed at reducing the amount of dioxins in sludge.

We also urge EPA to meet its statutory obligation under Section 405((b)(2)(D) by regulating additional toxic pollutants in sewage sludges.

Additional detail on some of these recommendations is below:

### ***Management Measures to Minimize Exposure***

There is agreement among the scientific community that the background levels of dioxins are already at levels that pose a risk to human health. Thus EPA needs to promulgate rules and measures to reduce those levels. The primary source of human exposure is through food of animal origin. The source of dioxins in the animal products is in turn primarily their food. Sewage sludges that are applied to agricultural lands are often used on lands used to grow feed crops for livestock or on pasture lands where livestock graze. This provides a route for the sludge-borne dioxins to enter the food chain. Pasture application and application to standing crops (such as forage) is particularly likely to lead to accumulation of dioxins in animal tissues and entry into the human food chain. Restriction of application practices to prohibit such applications would be an important measure to reduce exposure not only to dioxins but to other persistent bioaccumulative toxics in sludges. Prohibition of the use of sludges on crops used to feed animals and particularly on pasture lands would be one important means of reducing background levels of exposure to dioxins. Many other countries restrict application to pasture. Restricting the total quantity of sludge that may be applied over a give time period is another common practice in other countries. Thus only 5 tons may be applied per acre every five years in under some rules. Such a restriction in the U.S. would help to decrease the rate at which toxic contaminants are added to the soil.

### ***Pretreatment and Other Source Control Measures***

EPA should take steps to address dioxin-contaminated sludge through source control as well as end-of-pipe management measures. In particular, EPA should require POTWs that have detectable levels of dioxin in their sludge to take steps to identify the sources of the dioxins and, where feasible, to set local limits or take other steps to minimize the amount of dioxin discharged into the sewer system. Because chlorine is necessary to the formation of dioxins, reducing the amount of chlorine that is used in commercial or industrial enterprises that discharge to the sewer system, may reduce the amount of dioxin in the sludge at a relatively low cost. According to a 1993 Greenpeace report, four products are the major sources of dioxin releases into the environment – polyvinyl chloride (PVC) plastic, chlorinated solvents, chlorinated pesticides, and chlorine bleached paper (Thornton, 1993). Reducing the amount of these chemicals discharged into a sewer system or in sewer system piping could be used to decrease the amount of dioxins in the sludge. EPA's regulations should require source control approaches be used to minimize the amount of dioxin that reaches the public through land-applied sludge.

### ***Warning to Farm Families***

The persons at greatest risk are sludge-using farm families raising livestock and ingesting animal products grown on the farm. In addition to limiting the amount of dioxin to which these families are exposed, these persons should be told of the risks and how they can minimize them. This would include informing them about the potential accumulation of certain persistent bioaccumulative contaminants, including dioxins, in animal products when animals are exposed to sludges. To minimize exposure, they should take care to minimize the amount of sludge the

animals ingest by avoiding application to pasture and to standing forage. They should also consider the quantity of animal products they ingest from animals grown on their farm.

### ***Monitoring***

We urge EPA to include mandatory monitoring requirements in the final rule as well. Voluntary monitoring programs are ineffectual. Those most likely to have a problem are least likely to monitor. Annual monitoring is too infrequent to provide the regulators or the public with any assurance that the sludge is meeting with regulatory standard. More frequent monitoring, geared toward those conditions under which the treatment works or other sewage sludge preparers have historically had the highest rates of dioxin TEQ, is necessary.

Recent AMSA data on dioxins in sludges in the US showed one sludge with extremely high concentrations of dioxins (3590 ppt). This was determined to be a real observation (not analytic or other error) (Kester, Greg, personal communication, Wisconsin Biosolids coordinator). This points to the problem of using average concentrations to evaluate risk since that particular sludge would represent a high risk to anyone using it. It also speaks to the need for monitoring. The farm family that might be using that highly contaminated sludge would be at significant risk. It is common practice for a particular farm to receive a particular sludge (from one WWTP) from year to year. It is likely that the source of that contamination would continue to contaminate that sludge until and unless the sludge were tested and a process was undertaken to discover and eliminate the source. If no testing were required, the source would likely continue.

The concept that monitoring frequency be reduced if low concentrations of dioxins (less than 10% of standard) are detected in sludge monitoring conducted in the first two years has merit. We have reservations, however, about the adequacy of the current level of knowledge regarding fluctuations in dioxin concentrations from a particular POTW from sample to sample and in knowledge about trends over time on which this concept is based.

### ***Regulate Additional Contaminants In Sewage Sludge***

There are a number of other halogenated compounds that could contribute to the total concentration of compounds exhibiting similar toxicity. These include brominated analogues of PCDD and PCDFs (Van den Berg, et al., 1998). Polybrominated biphenyls and dioxins seem to pose similar risks to dioxins and PCBs (U.S. EPA, 1994; Hornung et al, 1996; Helleday, et al, 1999, Weber and Greim, 1997; Henck, et al, 1994). They appear to be carcinogenic (Hoque, et al, 1998; Henderson, et al, 1995). They are detected in sludges (Hagenmaier, et al., 1992a). If they act in similar toxicologic or oncogenic mode to dioxins, they need to be factored into the risk assessment or at least recognized as contributing to the risk. Numerous other PCBs might also be included.

In addition to the need to assess the risks posed by chemicals such as PBDEs which may act in modes similar to dioxins and should thus be included in this assessment of risks, there are additional chemicals of concern in sludges which Round 2 should address. The recent National

Research Council report found that the 1988 National Sewage Sludge survey was not adequate for evaluating the risks posed by currently known chemicals. It calls for a new survey including additional chemicals and using methods that provide for lower limits of detection. It also suggests that persistent bioaccumulative toxics are of particular concern.

In addition, many chemicals were excluded from Round 2 due to limitations in the available data. The NRC report suggests that these decisions need to be revisited since new data may be available. Lack of data does not mean lack of risk. Where data gaps exist, they need to be clearly identified and filled. This was promised but not carried through by EPA.

Table 1 displays data from the literature regarding organic chemicals detected in sludges in the years since the National Sewage Sludge Survey. Also, where available, the soil screening numbers used by EPA as well as the drinking water standards are displayed to provide context. The table shows that there may be chemicals for which the concentrations found in sludges may be at levels posing risks that need to be evaluated. This exercise suggests that a number of organic chemicals beyond dioxins should be considered in the Round 2 regulation of sewage sludges.

EPA needs to undertake an up-to-date survey of sludge quality for a wide range of contaminants in order to identify additional contaminants of concern.

We urge that you re-evaluate not only this proposal, but all others applicable to land applied sewage sludges to ensure that they are consistent with those all proposed uses of sludges, including home and garden use, and that all pollutants in sludges that pose a risk of human or ecological harm are regulated.

Thank you for considering these views.

Sincerely yours,

Nancy Stoner  
Director, Clean Water Project  
Natural Resources Defense Council  
1200 New York Avenue, N.W., Suite 400  
Washington, DC 20005  
202-289-6868

Jennifer Sass  
Senior Scientist, Public Health Program  
Natural Resources Defense Council  
1200 New York Avenue, N.W., Suite 400  
Washington, DC 20005  
202-289-6868

Ellen Z. Harrison, Director  
Cornell Waste Management Institute  
Center for the Environment  
100 Rice Hall  
Ithaca, NY 14853  
607- 255-8576

Enclosure (Table 1)

## References

“Exposure Analysis for Dioxins, Dibenzofurans, and CoPlanar Polychlorinated Biphenyls in Sewage Sludge. Technical Support Document,” November 30, 2001 prepared for US EPA, Office of Water. EPA Contract # 68-W-6-0053

Abt Associates Inc. 1994. Revision of Assessment of Risks to Terrestrial Wildlife from TCDD and TCDF in Pulp and Paper Sludge. Bethesda, MD.

Alonso, K. R., M. M. Peden-Adams, J. Y. Liu, et al. (1998). <sup>3</sup>Effects of in ovo exposure to 2,3,7,8-TCDD on F1 generation adult chickens (*Gallus gallus*).<sup>2</sup> *Chemosphere* 37(9-12): 1873-83.

Anderson, Dr. Henry, Wisconsin Department of Health and Social Services, personal communication

Axelrod, D. D.L. Davis and L. A Jones, *Envir. Health Persp.*, v 109 (6) June 2001, editorial.

Becher, H., Steindorf, K., Flesch-Janys, D. 1998. Quantitative cancer risk assessment for dioxins using an occupational cohort. *Environ. Health Perspect.* 106(2):663-670.

Birnbaum LS, Tuomisto J. Non-carcinogenic effects of TCDD in animals. *Food Addit Contam* 2000;17(4):275-88.

Brouwer, A., U. G. Ahlborg, F. X. R. van Leeuwen and M. M. Feeley. 1998. Report of the WHO Working Group on the Assessment of Health Risks for Human Infants from exposure to PCDDs, PCDFs and PCBs. *Chemosphere.* 37(9-12):1627-1643.

Chase, Dr. Larry, Cornell University Department of Animal Science, personal communication.

Commission of the European Communities, Council Regulation amending Commission Regulation (EC) No. 466/2001 setting maximum levels for certain contaminants in foodstuffs, 2001.

Cousins, I.T., A.J. Beck and K.C. Jones, 1999. A review of the processes involved in the exchange of semi-volatile organic compounds (SVOC) across the air-soil interface. *Science of the Total Environment.* 228(1): 5-24.

Damstra, T., Barlow, S., Bergman, A., Kavlock, R., Van Der Kraak, G., 2002; reviewed in the report by the World Health Organization (WHO) and the International Programme on Chemical Safety (IPCS), 2002. *Global Assessment of the State-of-the-Science of Endocrine Disruptors.*

Douben, P.E.T., R.E. Alcock and K.C. Jones, 1997. Congener specific transfer of PCDD/Fs from air to cow's milk: an evaluation of current modeling approaches, *Environmental Pollution*, 95:333-344.

Flesch-Janys D, Berger J, Gurn P, Manz A, Nagel S, Waltsgott H, et al. Exposure to polychlorinated dioxins and furans (PCDD/F) and mortality in a cohort of workers from a herbicide-producing plant in Hamburg, Federal Republic of Germany. *Am J Epidemiol* 1995;142(11):1165-75

Fries, G.F. 1996. Ingestion of sludge applied organic chemicals by animals. *Science of the Total Environment*. 185:93-108.

Fries, G.F. and D.J. Paustenbach, 1990. Evaluation of Potential Transmission of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin – Contaminated Incinerator Emissions to Humans Via Foods. *Journal of Toxicology and Environmental Health*, 29:1-43.

Giesy, J.P., P.D. Jones, K. Kannan, et al. (2002). Effects of chronic dietary exposure to environmentally relevant concentrations to 2,3,7,8-tetrachlorodibenzo-p-dioxin on survival, growth, reproduction and biochemical responses of female rainbow trout (*Oncorhynchus mykiss*). *Aquat Toxicol* 59(1-2): 35-53.

Goldring, J. 1992. Assessment of Potential Human Health Impacts of Dioxin and Furan Contaminated Sludge Amended to Soils. Wisconsin Department of Health and Social Services.

Gray LE, Ostby JS, Kelce WR. A dose-response analysis of the reproductive effects of a single gestational dose of 2,3,7,8-tetrachlorodibenzo-p-dioxin in male Long Evans Hooded rat offspring. *Toxicol Appl Pharmacol* 1997;146(1):11-20

Gray LE, Wolf C, Mann P, Ostby JS. In utero exposure to low doses of 2,3,7,8-tetrachlorodibenzo-p-dioxin alters reproductive development of female Long Evans hooded rat offspring. *Toxicol Appl Pharmacol* 1997;146(2):237-44

Hagenmaier, H., J. She, T. Benz, N. Dawidowsky, L. Dusterhoft and C. Lindig. 1992 a. Analysis of Sewage Sludge for Polyhalogenated Dibenzo-p-Dioxins, Dibenzofurans, and Diphenylethers. *Chemosphere*. 25(7-10):1457-1462.

Hagenmaier, H., J. She and C. Lindig. 1992 b. Persistence of Polychlorinated Dibenzo-p-Dioxins and Polychlorinated Dibenzofurans in Contaminated Soil at Maulach and Rastatt in Southwest Germany. *Chemosphere*. 25(7-10):1449-1456.

Hart, L. E., K. M. Cheng, P. E. Whitehead, et al. (1991). <sup>3</sup>Dioxin contamination and growth and development in great blue heron embryos.<sup>2</sup> *J Toxicol Environ Health* 32(3): 331-44.

Helleday, T., K. L. Tuominen, A. Bergman, D. Jenssen. 1999. Brominated flame retardants induce intragenic recombination in mammalian cells. *Mutation Research-Genetic Toxicology and Environmental Mutagenesis*. 439(2):137-147.

Henck, J. W., J. L. Mattsson, D. H. Rezabek, C. L. Carlson and R. H. Rech., 1994. Developmental Neurotoxicity of Polybrominated Biphenyls. *Neurotoxicology and Teratology*. 16(4):391-399.

Henderson, A. K., D. Rosen, G. L. Miller, L. W. Figgs, S. H. Zahm, S. M. Sieber, H. E. B. Humphrey and T. Sinks. 1995. Breast-Cancer Among Women Exposed to Polybrominated Biphenyls. *Epidemiology*. 6(5):544-546.

Hooiveld M, Heederik DJ, Kogevinas M, Boffetta P, Needham LL, Patterson DG, Jr., et al. Second follow-up of a Dutch cohort occupationally exposed to phenoxy herbicides, chlorophenols, and contaminants. *Am J Epidemiol* 1998;147(9):891-901

Hoover, S. M. 1999. Exposure to Persistent Organochlorines in Canadian Breast Milk: A Probabilistic Assessment. *Risk Analysis*. 19(4):527-545.

Hoque, A. A. J. Sigurdson, K. D. Burau, H. E. B. Humphrey, K. R. Hess and A. M. Sweeney. 1998. Cancer among a Michigan cohort exposed to polybrominated biphenyls in 1973. *Epidemiology*. 9(4):373-378.

Hornung, M. W., E. W. Zabel and R. E. Paterson. 1996. Toxic equivalency factors of polybrominated dibenzo-p-dioxin, dibenzofuran, biphenyl, and polyhalogenated diphenyl ether congeners based on rainbow trout early life stage mortality. *Toxicology and Applied Pharmacology*. 140(2):227-234.

Iida T, Hirakawa H, Matsueda T, Takenaka S, Yu ML, Guo YL. Recent trend of polychlorinated dibenzo-p-dioxins and their related compounds in the blood and sebum of Yusho and Yu Cheng patients. *Chemosphere* 1999;38(5):981-93.

Janz, D. M. and G. D. Bellward (1996). <sup>3</sup>In ovo 2,3,7,8-tetrachlorodibenzo-p-dioxin exposure in three avian species. 1. Effects on thyroid hormones and growth during the perinatal period.<sup>2</sup> *Toxicol Appl Pharmacol* 139(2): 281-91.

Kester, Greg, Wisconsin Biosolids Coordinator, personal communication.

Kogevinas M, Becher H, Benn T, Bertazzi PA, Boffetta P, Bueno-de-Mesquita HB, et al. Cancer mortality in workers exposed to phenoxy herbicides, chlorophenols, and dioxins. An expanded and updated international cohort study. *Am J Epidemiol* 1997;145(12):1061-75.

Linz, D.G. and D.V. Nakels, editors, 1997. Environmentally Acceptable Endpoints in Soil. An American Academy of Environmental Engineers Publications. Book.

Mably TA, Bjerke DL, Moore RW, Gendron-Fitzpatrick A, Peterson RE. In utero and lactational exposure of male rats to 2,3,7,8- tetrachlorodibenzo-p-dioxin. 3. Effects on spermatogenesis and reproductive capability. *Toxicol Appl Pharmacol* 1992;114(1):118-26.

Mably TA, Moore RW, Goy RW, Peterson RE. In utero and lactational exposure of male rats to 2,3,7,8- tetrachlorodibenzo-p-dioxin. 2. Effects on sexual behavior and the regulation of luteinizing hormone secretion in adulthood. *Toxicol Appl Pharmacol* 1992;114(1):108-17.

Mably TA, Moore RW, Peterson RE. In utero and lactational exposure of male rats to 2,3,7,8-tetrachlorodibenzo-p-dioxin. 1. Effects on androgenic status. *Toxicol Appl Pharmacol* 1992;114(1):97-107.

Masuda Y. Fate of PCDF/PCB congeners and change of clinical symptoms in patients with Yusho PCB poisoning for 30 years. *Chemosphere* 2001;43(4-7):925-30

McLachlan, M.S., 1997. A simple model to predict accumulation of PCDD/Fs in an agricultural food chain. *Chemosphere*, 34 (5-7):1263-1276.

McLachlan, M.S., A.P. Stewart, J.R. Bacon and K.C. Jones, 1996. Persistence of PCDD/Fs in a Sludge-Amended Soil. *Environmental Science and Technology*, 30:2567-2571.

McLachlan, M. S., M. Hinkel, M. Reissinger, M. Hippelein and H. Kaupp, 1993. A Study of the Influence of Sewage Sludge Fertilization on the Concentrations of PCDD/F and PCB in Soil and Milk. *Environmental Pollution*. 85:337-343.

Meyn,O., M.Zerman, M. Wish, S. Keane, *Environmental Toxicology and Chemistry*, Vol 16, No.9, 1789-1801, 1997.

Mocarelli P, Gerthoux PM, Ferrari E, Patterson DG, Jr., Kieszak SM, Brambilla P, et al. Paternal concentrations of dioxin and sex ratio of offspring. *Lancet* 2000;355(9218):1858-63.

National Research Council, Science and Judgment in Risk Assessment, NRC Committee on Risk Assessment of Hazardous Air Pollutants, Board on Environmental Studies and Toxicology, Washington, DC: National Academy Press, 1994.

Nelson, S. D., J. Letey, W. J. Farmer, C. F. Williams, and M. Ben-Hur. 1998. Facilitated transport of napropamide by dissolved organic matter in sewage sludge. *Journal of Environmental Quality*. 27(5):1194-1200.

Nosek, J. A., S. R. Craven, J. R. Sullivan, S. S. Hurley and R. E. Peterson (1992); Toxicity and reproductive effects of 2,3,7,8-tetrachlorodibenzo-p-dioxin in ring-necked pheasant hens; J Toxicol Environ Health 35(3): 187-98.

Orazio, C. E., S. Kapila, R. K. Puri and A. F. Yanders. 1992. Persistence of Chlorinated Dioxins and Furans in the Soil Environment. Chemosphere. 25(7-10):1469-1474.

Patandin, S., C. Koopman-Esseboom, M. A. J. De Ridder, N. Weisglas-Kuperus and P. J. J. Sauer. 1998. Effects of environmental exposure to polychlorinated biphenyls and dioxins on birth size and growth in Dutch children. Pediatric Research. 44(4):538-545.

Patandin, S., C. I. Lanting, P. G. H. Mulder, E. R. Boersma, P. J. J. Sauer and N. Weisglas-Kuperus. 1999. Effects of environmental exposure to polychlorinated biphenyls and dioxins on cognitive abilities in Dutch children at 42 months of age. Journal of Pediatrics. 134(1):33-41.

Peterson, R. E., H. M. Theobald and G. L. Kimmel (1993); Developmental and reproductive toxicity of dioxins and related compounds: cross-species comparisons; Crit Rev Toxicol 23(3): 283-335.

Rinella, J. F., P. A. Hamilton, and S. W. McKenzie. 1993. Persistence of the pesticide DDT in the Yakima River Basin, Washington. U.S.G.S.

Rosen DH, Flanders WD, Friede A, Humphrey HE, Sinks TH. Half-life of polybrominated biphenyl in human sera. Environ Health Perspect 1995;103(3):272-4

Smith, K.E.C. and K.C. Jones, 2000. Particles and vegetation: implications for the transfer of particle-bound organic contaminants to vegetation. The Science of the Total Environment. 246(2-3):207-236.

Steenland K, Piacitelli L, Deddens J, Fingerhut M, Chang LI. Cancer, heart disease, and diabetes in workers exposed to 2,3,7,8- tetrachlorodibenzo-p-dioxin. J Natl Cancer Inst 1999;91(9):779-86.

Thornton, 1993, Greenpeace, *Dioxin: From Cradle to Grave*, p. 4.

Thornton, I., and P. Abrahams. 1981. Soil ingestion as a pathway of metal intake into grazing livestock. In: Proc. Int. Conf. Heavy Metals in the Environment. Edinburgh, Scotland: CEP Consultants.

33 U.S.C. 1345(d)(2)(D).

U.S. EPA, *Estimating Exposure to Dioxin-like Compounds* (Table III-4, US EPA 1994).

U.S. EPA, Risk Assessment Forum. 1997. Guiding Principles for Monte Carlo Analysis. EPA/630/R-97/001. <http://www.epa.gov/oppefed1/ecorisk/backgrn.htm>. 35pp.

Van den Berg, M.L. Birnbaum, A.T.C. Bosveld, B. Brunstrom, P. Cook, M. Feeley, J.P. Giesy, A. Hanberg, R. Hasegawa, S.W. Kennedy, T. Kubiak, J.C. Larsen, F.X.R. van Leeuwen, A.K.D. Liem, C. Nolt, R.E. Peterson, L. Poellinger, S. Save, D. Schrenk, D. Tillitt, J. Tysklind, M. Younes, F. Waern and T. Zacharewski, 1998. Toxic Equivalency Factors (TEFs) for PCBs, PCDDs, PCDFs for Humans and Wildlife. *Environmental Health Perspectives*. 106:12.

Weber L. W. D. and H. Greim. 1997. The toxicity of brominated and mixed-halogenated dibenzo-p-dioxins and dibenzofurans: An overview. *Journal of Toxicology and Environmental Health*. 50(3):195-215.

Wild, S. R., J. Stuart, H. & K. C. Jones. 1994. The Influence of Sewage Sludge Applications to Agricultural Land on Human Exposure to Polychlorinated Dibenzo-p-Dioxins (PCDDs) and Furans (PCDFs). *Environmental Pollution*. 83(3):357-369.

Wilson, S.C., R.E. Alcock, A.P. Sewart and K.C. Jones, 1997. Persistence of Organic Contaminants in Sewage Sludge-Amended Soil: A Field Experiment. *J. Environ. Qual.* 26:1467-1477.

World Health Organization. 1998. Assessment of the health risk of dioxins: re-evaluation of the Tolerable Daily Intake (TDI), Executive Summary. WHO Consultation. Geneva, Switzerland. May 25-29, 1998. (<http://www.who.int/pes/pubs/dioxin-exec-sum/exe-sum-final.html>)

Wu, W. Z., W. Li, Y. Xu and J. W. Wang (2001). Long-term toxic impact of 2,3,7,8-tetrachlorodibenzo-p-dioxin on the reproduction, sexual differentiation, and development of different life stages of *Gobiocypris rarus* and *Daphnia magna*. *Ecotoxicol Environ Saf* 48(3): 293-300.