Association of
Metropolitan
Sewerage Agencies

September 10, 2002

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

Re: *Standards for the Use or Disposal of Sewage Sludge*; *Notice of Data Availability*; 67 *Fed. Reg*. 40554 (June 12, 2002)

Dear Sir or Madam:

The Association of Metropolitan Sewerage Agencies (AMSA) is pleased to provide comments on the U.S. Environmental Protection Agency's (EPA's) *Standards for the Use or Disposal of Sewage Sludge; Notice of Data Availability* (*NODA*). Founded in 1970, AMSA represents the interests of over 270 of the nation's publicly owned wastewater utilities (POTWs). AMSA members serve the majority of the sewered population in the United States and collectively treat and reclaim over 18 billion gallons of wastewater every day. As generators of treated sewage sludge, or biosolids, AMSA's members are responsible for finding environmentally safe and cost effective ways to manage millions of tons of biosolids every year. Land application continues to be one of the most viable and environmentally sound management options for many communities in the United States.

AMSA commends EPA for taking further comment before making a final regulatory decision on dioxins in land applied biosolids. The additional time has allowed the Agency to collect and evaluate new data and to perform additional risk assessment analyses using that data. The information in the *NODA* provides a current picture of the issues surrounding dioxins in biosolids and a solid foundation upon which to base a regulatory decision.

AMSA's comments focus on whether a national numeric limit for dioxin in land applied biosolids is appropriate and respond to a number of issues on which the

Agency solicited input. Based on a thorough review of the Agency's new risk assessment, AMSA's comments also highlight a few areas where additional review is needed as EPA works toward final action. AMSA's comments on the *NODA* are outlined below.

No Action Warranted for Dioxin in Land Applied Biosolids

The *NODA* presents extensive new information regarding the presence of dioxins in land applied biosolids. Based on the information presented in the *NODA*, AMSA believes that a national numeric limit for dioxins in land applied biosolids is not warranted. The *NODA* makes some key observations regarding EPA's new information that support a no action decision:

- **Current concentrations of dioxins in biosolids are low and do not present a significant risk to human health or the environment.** Results from surveys of dioxin concentrations in biosolids conducted by both EPA and AMSA over the last two years demonstrate that the average concentration of dioxins in biosolids is very low. EPA's 2001 Dioxin Update Survey found an average or mean concentration of dioxins of 31.6 ppt TEQ (toxic equivalents) (expressed with non-detects at ½ the detection limit). AMSA's 2000/2001 Survey of Dioxin-Like Compounds in Biosolids[1] found a mean concentration of 48.5 ppt TEQ (expressed with non-detects at ½ the detection limit). As outlined in the *NODA*, these concentrations do not appear to pose a significant risk to human health or the environment, even when EPA's highly conservative risk estimates are applied to the exposure assessment used in the *NODA*.

- **Dioxins concentrations in biosolids have been and continue to significantly decline, which will likely ensure further future reductions in potential risks.** A comparison of the results from EPA's 2001 Survey with the results of EPA's 1988 National Sewage Sludge Survey (NSSS) reveals that concentrations of dioxins in biosolids appear to be declining. EPA states that the significance of these differences is not certain due to changes in the sampling procedures and analytic methods. However, the results of AMSA's survey revealed similar trends when the 2000/2001 results were compared with a survey conducted in 1994 and 1995. The observed trend in decreasing dioxin concentrations in biosolids is consistent with the generally observed decreases in dioxin levels in other media and biological organisms, including humans. As dioxins decrease in the environment and humans, the amount of dioxin entering POTWs is expected to decrease – resulting in continued decreases of dioxins in biosolids.

- **Dioxin "spikes" in sewage sludge are transient.** The new data presented reveal that to the extent a "spike" (i.e., higher concentration) in dioxin levels is detected in a POTW's sewage sludge, that effect is transient. No POTWs have consistently high levels of dioxins in their biosolids. The biosolids samples that had the highest levels of dioxins in either the 1988 NSSS or the 2001 EPA Survey displayed the largest fluctuations in dioxins concentrations. Because any dioxin spikes tend to be transient, it is unlikely that biosolids from any one POTW will pose an unusual threat to human health or the environment.

[1] AMSA's 2000/2001 Survey of Dioxin-Like Compounds in Biosolids is available at *http://www.amsa-cleanwater.org/advocacy/dioxin/dioxin.cfm*.

The *NODA* also presents the results of EPA's screening ecological risk analysis (SERA) and a new probabilistic risk assessment for human health risks. These results also support a no action decision for dioxins in land applied biosolids:

- **Low probability of adverse ecological effects.** While the SERA does not establish definitive estimates of risk, it does provide insight into the potential for ecological risk. Using a two phased approach, the SERA first identified any habitats, receptor categories, and exposure routes that may be of concern. EPA then conducted a number of deterministic assessments for representative receptor categories (species) in two major habitats: terrestrial and waterbody margin habitat. None of the values obtained from these assessments exceeded the protective ecological benchmark (Hazard Quotient = 1), demonstrating with a "high level of confidence that there is a low probability of adverse effects to ecological receptors" resulting from dioxins in land applied biosolids (67 *Fed. Reg.* 40570).

- **Incremental human health risk is minimal.** The results of EPA's new probabilistic risk assessment suggest that the incremental increase in individual lifetime risk of developing cancer due to exposure to dioxins potentially present in biosolids is minimal. Using the Agency's current cancer slope factor, the high-end individual excess lifetime risk to the highly exposed modeled population could range from 2×10^{-5} to 1×10^{-6} (two in one-hundred thousand to one in one million) for exposure by multiple pathways. These excess risk levels fall within the range of risks considered acceptable by the Agency, 1×10^{-6} to 1×10^{-4}, and are lower than the risk level used for Round 1, 1×10^{-4}. Furthermore, EPA uses highly conservative slope factors, which provides additional assurance that dioxins in biosolids do not pose an unacceptable risk.

Any regulatory limit or control placed on dioxins in biosolids must reduce the risk posed by the land application of those biosolids. EPA initially proposed a limit of 300 ppt TEQ as the key threshold for minimizing the risk posed by dioxins in biosolids. When the new data are evaluated and the risks calculated, however, the need for such a numeric limit or threshold is not evident. Although the baseline level of risk may change slightly (with a change in cancer slope factor or other adjustment in the exposure or risk assessment), EPA found that there is no quantifiable decrease in risk when biosolids with greater than 300 ppt TEQ dioxins or even greater than 100 ppt TEQ dioxins are restricted from land application. Because the concentrations of dioxins in biosolids are so low (i.e., there are very few biosolids samples with levels above 100 ppt TEQ), a regulatory limit or threshold that restricts the application of biosolids with dioxins concentrations above these levels would have little or no impact on decreasing the overall risk. Therefore, placing such a regulatory limit on biosolids may cause a misallocation of limited agency and public resources for no gain in protection of human health.

EPA also found that the continual application of biosolids with significantly higher concentrations of dioxins than currently measured would be necessary to predict quantifiable increases in risk. Given the already low levels of dioxins, the demonstrated decline in dioxin concentrations over time (in human tissues and the environment, and consequently in biosolids), and the observed transient nature of elevated dioxin concentrations, such a scenario seems unlikely.

AMSA's Review of the New Risk Assessment

In light of the importance assigned to the results of EPA's new probabilistic risk assessment, AMSA conducted a careful review of the Agency's methodology to ensure it is consistent with sound risk assessment practices. AMSA's review revealed a number of very conservative assumptions, methods, and parameter values used by EPA that likely lead to overestimates or overstatements of risk. Below is a brief list of those elements that may have resulted in an overstatement of risk. Additional detail on these items including an estimate of the impact they may have on the risk estimates and suggested remedies is provided in Attachment 1. AMSA notes that despite these numerous conservative assumptions and other sources of overestimation, the resulting incremental human health risk calculated for the *NODA* is still within the Agency's range of acceptable risks. This should provide the Agency further assurance that a no action approach will be protective of human health and the environment.

Potential Sources of Overestimate/Overstatement

1. Common to All Pathways

   - *Field versus farm size*
     EPA incorrectly assumes that a field and a farm are equivalent, so that if biosolids are used on a field, they are used everywhere on that farm. In particular, for the major pathways EPA assumed that biosolids are used wherever they could affect the cows or steers contributing to milk or beef eaten by the farmer.

   - *Period of biosolids use*
     EPA appears to assume that any farm that uses biosolids will continue to use them regularly for long periods (it is not clear how the distribution for application period and the distributions for exposure periods interact). This affects the distribution of results – it is likely that a larger farmer population than calculated is exposed, but they are affected less on average, while the extremes of the distribution are less affected.

   - *Method used to estimate the total number of cancers*
     EPA does not use the mean value of the variability distribution to calculate total numbers of cancers. Using the upper-end values, as given in the *NODA*, gives a very large overestimate of expected cancers. The *NODA* uses as examples the 90th, 95th and 99th percentiles of risk, the last of which is 10 times the mean value.

   - *Estimates of the size of the population modeled*
     EPA's estimates of the size of the population affected appear to be based on the total number of farmers. This fails to take into account that not all farmers eat home-produced beef (about 39% do) or drink home-produced milk (only about 13%) and, therefore, overestimates exposure.

   - *Carcinogenic potency*
     EPA's dioxin risk estimates presented in the *NODA* are based on highly conservative slope factors. Many in the scientific community have argued that the available evidence suggests that dioxin poses no human cancer risk at the low levels encountered in the

environment. Further, EPA appears to have staked out a highly conservative position on dioxin's potential cancer risks that is inconsistent with other federal and international bodies. The Agency for Toxic Substances and Disease Registry (ATSDR) recently published an overview concerning how different Federal and international bodies assess the potential risks posed by dioxin.[2] Whereas EPA relies solely on a conservative linear default model to quantify dioxin cancer risks, other federal and international bodies (e.g., ATSDR, the European Commission Scientific Committee on Foods (EC), the Joint FAO/WHO Expert Committee on Food Additives (JECFA)) have determined that a threshold model is more consistent with the science concerning dioxin's carcinogenic action.[3] According to ATSDR, this has led to a "world-wide convergence" on an acceptable dioxin exposure of 1 to 4 pg/kg/day. This is in contrast to EPA's determination in its draft Dioxin Reassessment that a one in a million cancer risk exists at approximately 0.001 pg/kg/day. ATSDR observed that EPA's dioxin reassessment "may place too much confidence in the ability to accurately predict cancer risks at low doses. This approach dramatically increases cancer risk estimates that are not based on compelling new data but rather on the application of statistical models . . . [that] are not yet fully validated . . . ." (Pohl, 2002).

Despite EPA's highly conservative approaches for estimating the potential cancer and non-cancer risks posed by dioxin, it is important to note that the *NODA* states that using either EPA's current cancer slope or the one presented in its Draft Reassessment, the dioxin risks calculated for biosolids are below or around 10^{-4}. Despite the acceptable risk estimates for biosolids presented in the *NODA*, as a general matter AMSA believes that EPA should take into account the conservative nature of its risk estimates and the likelihood that use of those estimates overstate the potential risks posed by dioxins when managing biosolids.

- *Exposure frequency*
  EPA incorrectly assumes all exposures on a farm continue for 350 days a year. However, periods away from the farm are likely to exceed two weeks a year.

- *Concentrations of dioxins in biosolids applied to land*
  EPA treats the contaminant distributions measured in the biosolids survey as a variability distribution between fields for long-term average concentration. Since any single field will effectively receive many "samples" of biosolids over a long period (so that variation between such samples will tend to average out), this overestimates the upper percentiles of long-term average concentration.

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[2] Pohl, HR, *et al.* 2002. Public Health Perspectives on Dioxin Risks: Two Decades of Evaluations. Hum. Ecol. Risk Assess. 8(2):233-250.

[3] In a recent comparison of EPA's Dioxin Reassessment and the World Health Organization's dioxin cancer assessment, the U.S. General Accounting Office concluded that a "major difference in the organizations' assessments concerns whether there are threshold levels below which exposure to dioxins would pose negligible risk of cancer." While EPA assumes there is no safe threshold level for cancer effects, the WHO assumes there is. U.S. General Accounting Office, April 2002, "Environmental Health Risks, Information on EPA's Draft Reassessment of Dioxins," GAO-02-515.

- *Exposure duration for those initially exposed as children*
  EPA bases the distribution of exposure duration for those initially exposed as children not on farmers, but on the general population and may well overestimate exposures for children on farms.

- *Concentration trends in biosolids*
  EPA fails to account for future decreases in dioxin concentrations. EPA has undertaken many highly successful efforts over the last two decades to manage releases of dioxin to the environment. Through effective controls on dioxin sources and emissions, levels of dioxin in humans and the environment (important sources of dioxins in biosolids) have dramatically decreased. As those levels of dioxins decrease, concentrations of dioxins in biosolids can reasonably be expected to decrease. Indeed, EPA's and AMSA's survey data indicate such a decrease. EPA should expect to observe further future decreases in dioxins in biosolids. EPA's failure to take those expected decreases into account leads to an overestimation of future risks posed by dioxins in biosolids.

2. Major exposure pathways (beef, milk, maternal milk)

   - *Soil vapor emission model*
     The soil vapor emission model as described has three principal failures affecting dioxin and PCB emissions from soil – it applies an incorrect solution of the diffusion equation at the soil surface (effectively ignoring the stated boundary condition); it fails to account for the boundary layer of air just above the soil surface; and it fails to account for infiltration of rainwater at short times after tilling, when the majority of emission occurs. The first reduces estimates of emission rates, the second principally affects very involatile materials (like the higher chlorinated dioxins) and results in overestimates of emission rates, and the last affects more volatile materials (like TCDD and PCBs), again resulting in an overestimate of emission rates.

   - *Food preparation losses*
     Food preparation losses for beef are based on observed weight losses during cooking and post-cooking operations. However, EPA does not account for the preferential loss of fat (in which dioxins and PCBs are primarily concentrated) in such preparation losses.

   - *Correlations between milk and beef consumption*
     The correlations between milk and beef consumption are not considered. This omission principally affects distribution shapes, rather than averages, and results in some overestimation.

3. Minor pathways of exposure

   - *Worst-case farm layout*
     EPA's adopted scenario appears to correspond to approximately the worst possible case, with all runoff from biosolids affecting the same area that happens to be used for other purposes (e.g., raising chickens), and all affecting the same stream, used by the farmer for fishing. Use of this scenario overestimates potential exposures.

   - *Soil erosion modeling*
     The soil erosion modeling likely overestimates soil erosion to areas that then act as source terms to the minor pathways of exposure and may, therefore, overestimate exposure.

   - *100% fraction of contaminated fish*
     EPA's assumption concerning the fraction of contaminated fish may lead to an overestimation of exposure.

   - *100% contaminated fraction for ingested soil*
     EPA's assumption concerning the fraction of ingested soil that may be contaminated may lead to an overestimation of exposure.

   - *Soil ingestion rates*
     EPA uses the standard regulatory assumptions of 100 mg/day for young children, and 50 mg/day thereafter. Available evidence indicates that these regulatory assumptions overestimate actual average ingestion rates.

   - *Food preparation losses*
     Food preparation losses are estimated from weight losses during food processing, but losses of dioxins/PCBs sorbed to the surfaces of foods (particularly root vegetables) will be higher, so the loss is underestimated and the risk overestimated.

   - *Soil mixing depth*
     The fixed, small, soil mixing depth in the barrier area results in an overestimate of risk.

   - *Correlations between food ingestion rates*
     Food ingestion rates are treated as uncorrelated for different foods.

AMSA's review did reveal some potential errors in EPA's risk assessment that might understate estimates of risk.

Potential Sources of Underestimate/Understatement

   - *Soil column model*
     The soil column model appears to overestimate dispersion of contaminants in the soil from the mechanisms considered (although it omits a possible mechanism, bioturbation).

Therefore, it may underestimate surface soil concentrations in the field where the biosolids are applied.

- *Vapor dispersion modeling*
  The vapor dispersion model used omits the principal physical effects (it evaluates dispersion of vapor above the grass layer and from some distance away, omitting the diffusion of vapor up from the contaminated soil immediately beneath the grass), resulting in a possible underestimate of vapor concentrations around plants, which may ultimately impact the estimated dioxin concentrations in milk and beef.

- *Vapor uptake by plants*
  The vapor uptake estimates are based on the measured ratio of concentration in grass to the concentration in vapor dispersed above the grass in field conditions. This approach might underestimate the uptake from an equal concentration of vapor in the immediate vicinity of the grass leaves (as would be obtained from vapor diffusing up from the soil directly below the grass). This is because the resistance to diffusion of vapor from the plume above the grass is higher than the resistance to diffusion of vapor adjacent to the grass leaves.

- *Maternal milk route*
  A scoping calculation using the model applied in the Technical Background Document (TBD) demonstrates that infant exposure to breast milk might contribute a larger fraction to EPA's risk estimates (the TBD assigns a negligible contribution). However, EPA's approach in the *NODA* appears consistent with its extensive consideration of breast milk risk outlined in its draft Dioxin Reassessment, which concluded that dioxins in breast milk generally do not pose an unreasonable risk to nursing infants.

- *Fraction home-produced*
  The intake equation for foods (including beef and milk) erroneously includes a "fraction home-produced." The distributions for intake quantity are already of the home-produced component of food components only, so such factors are not needed.

AMSA continues to believe that EPA's decision regarding dioxins in land applied biosolids must be based on sound science. For this reason, AMSA strongly recommends that EPA strengthen the underlying foundation of the information presented in the NODA by further investigating the issues outlined above concerning the risk assessment and addressing them as appropriate and in accordance with sound risk assessment practices as the Agency works toward final action.

No Regulatory Action Does Not Mean "No Action"

Should EPA conclude that the risk assessment and other scientific data dictate that no federal regulatory action is appropriate for dioxins in land applied biosolids, AMSA notes that this does not mean that nothing will be done at POTWs to monitor and reduce the presence of dioxins in biosolids. In fact, many of the nation's wastewater treatment agencies already monitor for dioxins in biosolids, despite the lack of a regulatory requirement, to address local concerns and issues. These practices are unlikely to change in the event that EPA decides not to regulate dioxins in land applied biosolids. AMSA is confident that voluntary monitoring will continue to demonstrate that dioxins in biosolids do not pose an unacceptable

risk to human health or the environment. That monitoring may also demonstrate the anticipated decrease of dioxins in biosolids.

Response to Specific Comment Requests

Below are AMSA's responses to the 12 issues on which EPA sought comment in Section XIII of the *NODA*.

1. Request for Comments
   The significance of the differences in dioxin concentrations in sewage sludge measured at facilities with wastewater flows greater than one MGD (million gallons per day) compared to dioxin concentrations in sewage sludge at facilities with wastewater flows less than one MGD.

   AMSA Response
   The data in Table 2 suggest that the concentrations of dioxins in biosolids at small facilities (less than one MGD) are lower than concentrations at large facilities. However, EPA points out that the significance of these differences may be difficult to assess due to a variety of factors, including a small sample size. The AMSA 2000/2001 survey also examined the mean concentrations of dioxins in biosolids from small and large facilities to determine if there was a significant difference. Although AMSA's survey was not conducted according to the same statistical procedures as EPA's survey, the *t*-tests conducted for the AMSA survey did not suggest any significant difference between the mean TEQ mass fractions of small and large facilities.

2. Request for Comments
   The significance of the differences in dioxin concentrations in sewage sludge measured in the EPA 2001 dioxin update survey compared to dioxin concentrations in sewage sludge measured in the 1988 National Sewage Sludge Survey.

   AMSA Response
   As stated above, AMSA believes this difference is significant. Although AMSA's dioxin surveys were not conducted according to the same statistical procedures as the EPA surveys, the data from the AMSA surveys (1994/1995 and 2000/2001) provide further support for this trend of decreasing dioxin concentrations over time. Both the overall low concentrations of dioxins in biosolids and the decline in those concentrations over time suggest that controls on dioxins in other regulatory programs are having a noticeable effect on releases to the environment. Although data is not provided for PCBs, it would not be unreasonable to assume that PCBs would show the same decreasing trend with time.

3. Request for Comments
   Choice of the highly exposed farm family as the modeled population for the revised risk assessment and the assumptions related to this choice of modeled population.

   AMSA Response
   We believe that the assumptions used to define the farm family lead to an overestimation of exposure and risk estimates. EPA has used a highly exposed individual (HEI) concept to define

exposure. The recently completed National Research Council (NRC) study *Biosolids Applied to Land: Advancing Standards and Practices* recommends the use of a reasonable maximum exposure (RME) approach when conducting risk assessments as opposed to characterizing exposure to a maximally exposed individual (MEI) or a highly exposed individual (HEI). The NRC report stated that use of a RME will result in a more accurate characterization of exposure/risk, minimizing the compounding effect of overly conservative assumptions associated with the MEI or HEI approach. We encourage EPA to redefine exposure to the farm family using assumptions that are more consistent with a RME approach.

Attachment 1 provides a more comprehensive list of the issues AMSA identified in the Agency's risk assessment.

4. Request for Comments
All of the assumptions related to exposure, fate and transport used in the revised risk assessment, including the specific assumptions related to the farming and grazing practices used by the modeled farm family.

AMSA Response
Attachment 1 provides a more comprehensive list of the issues AMSA identified in the Agency's risk assessment, but a few of the assumptions are worth highlighting:

- No distinction is made between a field within a farm and the farm itself. In other words, EPA appears to assume that biosolids are applied to the entire farm (except the buffer area). In addition, all fields within a farm appear to have biosolids applied at the same rate and frequency. AMSA recommends that EPA consider an application distribution that more closely approximates actual operating practices. One possible approach would be to assume that one third of the fields within a farm receive biosolids in any given year and that application rotates between fields from year to year.

- EPA assumes that the farmer does not rotate the pasture to grow row crops where tilling of biosolids in the soil would mitigate dioxin volatilization transport. EPA should more closely consider actual farming practices and factor in some level of crop rotation.

- All farms are assumed to have a fishable stream immediately adjacent to the farm, with the stream always located downhill from the farm. The stream therefore receives runoff from the biosolids amended fields. The farmer is assumed to catch fish from these streams and consume the fish, which have some level of dioxin contamination. This description may fit a very limited number of farms, but it is clearly not appropriate to use this assumption to describe all farms. AMSA believes the exposure assessment should use a distribution where a small, but conservatively defined percentage of the farms are assumed to have a layout similar to the above description.

- All farms are assumed to raise beef cattle, dairy cattle and free range chickens for direct consumption. All of these animals are assumed to consume dioxins through direct ingestion of the biosolids-soil mixture and/or through consumption of plant products that have accumulated

dioxins through plant uptake or volatilization/re-deposition. While AMSA believes that the potential routes of dioxin intake are appropriate, assuming that 100% of the farmers would raise beef cattle, dairy cattle and free range chickens for direct consumption is unrealistic. The percentage of home produced products does not appear to be consistent with earlier EPA estimates. We encourage EPA to use a more realistic assumption regarding the percentage of home produced animal products.

- The concentration of dioxins in biosolids is assumed to remain constant over the period of application. In addition, dioxins are not assumed to degrade in the soil environment with time. The EPA exposure assessment should reflect dioxin degradation and should also reflect some variation in dioxin concentration in the source material over time.

5. Request for Comments
The treatment of non-detects in the revised risk assessment and the effect on estimating risk.

AMSA Response
AMSA agrees with EPA's treatment of non-detects in the revised risk assessment. Because of the sensitivity and limits of detection achieved by the analytical procedures used in EPA's 2001 survey, the reported dioxin congener values are relatively unchanged and there is no quantifiable difference in risk if non-detects are treated as zero, one-half the detection limit, or at the detection limit. AMSA's 2000/2001 survey treated non-detects in the same manner.

6. Request for Comments
The assumptions and values used to estimate how much dioxins are being transported to individuals in the modeled farm family (e.g., the sources [store-bought versus farm-produced], types and dioxin contamination levels of poultry feeds.)

AMSA Response
See Response to Comment 4 and Attachment 1.

7. Request for Comments
The methodology and data used for the screening ecological risk assessment, and the results derived from the screening ecological risk analysis.

AMSA Response
AMSA believes EPA has taken a reasonable approach to evaluate the ecological risk posed by dioxins in land applied biosolids. Calculation of relative ecological risks using a hazard quotient (HQ) approach is consistent with other EPA programs.

Some of the biosolids concentrations used for the Screening Ecological Risk Analysis (SERA) are likely overestimates (the first screen assumed the ecological receptors were exposed to the raw biosolids). The concentration estimates were derived from the human health risk assessment and used as surrogates for ecological receptors, an approach that needs further evaluation. This approach leads to some overestimates that are not identified in the Technical Background Document. For example, earthworms were effectively assumed continuously exposed to soil

concentrations calculated to be present in the top 20 cm, 2 cm, or 1 cm of the soil column (in cropped areas, pasture, and in the buffer, respectively), although such an assumption is not valid.

See also discussion above "No Action Warranted for Dioxin in Land Applied Biosolids"

8. Request for Comments
The significance of the finding that setting a 300 ppt TEQ limit would make no detectable difference in the risk of cancer to the highly exposed farm family.

AMSA Response
The finding that a 300 ppt TEQ limit would make no detectable difference in the risk of cancer is a function of the fact that very few biosolids samples from EPA's 2001 survey exhibited dioxins concentrations over 300 ppt TEQ. The same is true if a limit of 100 ppt TEQ is assumed. If the Agency decides to establish a numeric limit, that limit should be risk based and consistent with the risk level used in Round 1 (1×10^{-4}).

See discussion above "No Action Warranted for Dioxin in Land Applied Biosolids"

9. Request for Comments
Taking no action with respect to regulating dioxins for land application.

AMSA Response
See discussion above "No Action Warranted for Dioxin in Land Applied Biosolids"

10. Request for Comments
The proposed monitoring schedule and the threshold concentration of dioxin that would allow for less frequent monitoring, and specifically, on whether other schedules which would require more or less frequent monitoring would be more appropriate.

AMSA Response
Assuming EPA decides that a numeric limit for dioxins in biosolids is necessary, and develops a risk-based limit or ceiling concentration using the 1×10^{-4} risk level, AMSA believes that all POTWs subject to the rule should be required to conduct an initial test to establish a baseline dioxins concentration. POTWs already monitoring for dioxins would be able to rely on existing data. If the baseline concentration is lower than the ceiling concentration, POTWs would be required to monitor dioxins concentrations once every five years. If the baseline concentration exceeds the ceiling concentration, AMSA would suggest a two-step approach. The initial step would consist of a confirmatory test. If the test confirms the elevated dioxins concentration, the POTW would be required to investigate the source of the dioxins using the methodology suggested by EPA in the *NODA*. In the event that a source can not be identified and controlled, and as long as the dioxins concentration remains above the limit, the POTW would not be permitted to land apply its biosolids.

EPA also requested comments on whether a monitoring requirement in lieu of a numeric limit should be considered. Unfortunately, a monitoring regiment without some sort of action level is

meaningless. Any requirement to monitor biosolids for elevated dioxins concentrations would have to be accompanied by some sort of guidance as to what levels are elevated, what levels are "safe", and at what level should land application be halted. Without this information, POTWs would be required to establish their own action levels, which would likely make the monitoring requirements more burdensome and potentially less effective than a national numeric limit.

11. Request for Comments
    Excluding small entities from the limits for dioxins in sewage sludge to be land applied.

    AMSA Response
    Given that the total amount of land applied biosolids generated by small POTWs accounts for less than eight percent of total biosolids land applied, and the extremely small probability that this small amount of biosolids could unreasonably increase the health risk for any individual, AMSA agrees with EPA that small entities could be subject to less stringent requirements or completely excluded without a measurable increase in risk. The apparent difference between dioxins concentrations at small and large facilities (small facilities having lower concentrations on the whole) observed in the EPA survey data, suggests that a different regulatory scheme may be warranted. EPA notes that the significance of these differences may be difficult to assess due to a variety of factors, including a small sample size, and the AMSA survey did not find a significant difference between these two groups (see response to Comment 1). Nevertheless, when the costs of regulating these small facilities are compared to the minimal risk posed by the small quantity of biosolids generated by these facilities, an exclusion appears warranted.

12. Request for Comments
    A methodology to assist communities in voluntarily identifying and reducing or eliminating sources of dioxins entering wastewater treatment plants that contribute to elevated levels of dioxins in sewage sludge.

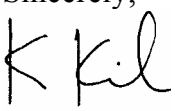
    AMSA Response
    EPA should develop guidance to provide additional details and explain how communities can utilize this voluntary methodology. Although eliminating the elevated concentrations of dioxins does not measurably change the risk to individuals exposed to land applied biosolids, it will be beneficial to have some means by which to track down the sources of those elevated levels. In addition, communities will be able to evaluate the potential for their biosolids to have elevated dioxin levels without having to conduct expensive tests.

EPA's Final Action

AMSA believes that given the information presented in the *NODA*, including the current data on dioxins in biosolids and the results of the Agency's new probabilistic risk assessment, a no action decision is warranted. As noted above, AMSA continues to believe that EPA's decision regarding dioxins in land applied biosolids must be based on sound science. Accordingly, AMSA strongly recommends that EPA strengthen the underlying foundation of the information presented in the *NODA* by further investigating the issues we have identified concerning the risk assessment and addressing them as appropriate as the Agency works toward final action.

We appreciate the opportunity to comment on the *NODA*, and look forward to continued discussions with the Agency on this matter. If you have any questions about our comments, please do not hesitate to contact me at 202/833-4653 or via email at kkirk@amsa-cleanwater.org.

Sincerely,



Ken Kirk
Executive Director

ATTACHMENT

Review of Overestimates in the Risk Assessments Supporting Standards for the Use or Disposal of Sewage Sludge Notice of Data Availability 67 Fed. Reg. 40554–40576 (Wednesday, June 12, 2002) Docket W–99–18 NODA

Prepared for:
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1 Introduction

1.1 The review

This review is based on an examination of the EPA Biosolids Risk Assessment, including:

- the Federal Register notice (FR, EPA, 2002a),
- the *Technical Background Document* and their appendices (TBD, EPA, 2002b, 2002c),
- the *Risk Characterization* (RC, EPA, 2001a),
- the *External Peer Review Comments* (EPRC, EPA, 2002d), and
- the *Population Risk Estimate* (PRE, EPA, 2001b),

I have examined these documents for methods and parameter values used by EPA and its contractors that produce overestimates of risk. The same examination also produced examples of methods and parameter values that may underestimate risk, or whose importance is not easy to determine because of lack of information (see Section 1.2). Such examples are listed in the Association of Metropolitan Sewerage Agencies comments on the NODA.

The approach taken by the EPA and its contractors is mixed. A Monte Carlo probabilistic approach is used to (partially) evaluate variability in exposures and risks of the sub-population examined, while uncertainty is treated by choosing estimates of some fixed parameters at extremes of their range. For the fixed parameters, therefore, it is unequivocally possible to determine immediately when the values used result in overestimates of risk. However, for the parameters incorporated in the probabilistic part of the analysis, whether risk is overstated may depend on the point on the variability distribution (or the metric derived from the probability distribution) that is ultimately chosen to "characterize" risk. Currently this is stated (in the *RC* but not the *FR*) to be the 50th percentile; but EPA (in the *FR*) requests comments on the selection. For example, overstatement of the variability of a distribution may have little effect on the 50th percentile of the result, but overestimate the upper percentiles and the mean.

I list separately overestimates due to scenario selection, incorrect models or methods, those due to fixed parameters, and those actually or potentially due to probabilistic parameters. Where possible, I attempt to semi-quantitatively characterize the size of the actual or potential overestimates, using the following scale:

Very large	Quite likely a factor of 5 or bigger,
Large	Quite likely a factor of 1.5 to 5,
Small	Probably less than a factor of 1.5, and
Trivial	Probably less than a factor of 1.1.

and where necessary and possible I distinguish between effects at the 50th percentile and at higher percentiles.

Since all the exposure pathways examined except the milk and beef pathways have a trivial (in these terms) effect on the ultimate risk estimates, and there are some very large effects that are common to all pathways, I examine separately:

Effects common to all pathways,
The major pathways (milk and beef), and
Other pathways.

EPA and its contractors have attempted to list their understanding of which of the fixed parameter values are overestimates (in the *RC* and *FR* at 40567). However, their lists are somewhat misleading, primarily because they do not examine the pathways separately, and there are such large differences in the contributions of the different pathways.

1.2 Impediments to review

Some comment is needed on difficulties encountered in this review that could readily be alleviated by EPA. These difficulties limit me to only rough estimates of the effects of the overestimates documented here. The principal difficulty was in determining exactly what models had been applied, since some of the documentation is confusing or self-contradictory. Intermediate results that would allow confirmation of the computations are largely undocumented. The effort involved in reproducing all the modeling from the documentation would have been far too substantial for this review, as well as impossible because of ambiguities in the documentation. The implementations of the models (including input data files, output data files, spreadsheets, and any specialized computer programs) were not made available to the public, and attempts to obtain them from EPA were unsuccessful.[1] The reader thus cannot tell, in some cases, exactly what models were intended (because of ambiguities in the documentation), and cannot evaluate whether what was implemented corresponds either to what was documented, or to a correct model.

[1] An e-mail on June 24, 2002 to the EPA contact listed in the Federal Register received no response. Subsequent contact with the EPA in August 2002 did not lead to the identification of anyone at EPA or elsewhere who could supply the implementations. Some of the models appear to be similar to, or identical with, those used in the "3MRA" modeling being developed for the Hazardous Waste Identification Rule. However, the implementation of even those models is not yet available (personal communication, Dr. Gerry Laniak, EPA).

2 Effects common to all pathways

2.1 Scenarios

2.1.1 Field versus farm size

In defining scenarios, it is assumed that a field and a farm are equivalent (TBD, Section 4.3.1), so that if biosolids are used on a field, they are implicitly used on all crop and pasture land on that farm. In particular for the major pathways, it is assumed that on every farm where biosolids are used, they are used on all pastures where cows or steers graze that supply milk or beef to the farm family, and on all fields where feed for such cows or steers are grown. In practice, it is unlikely that all farms that use biosolids will use them in such a fashion. I do not have data to evaluate degree of risk overestimation due to this assumption, but it is likely a small to large factor.

2.1.2 Period of biosolids use

It appears to be assumed that any farm that uses biosolids will continue to use them regularly for long periods, but it is not clear from the documentation what is assumed for the application period. Section 4.2.2 indicates that application continues for "up to" 40 years. Table C-1 lists a triangular distribution for a parameter "CutOffYr," given as an "operating life" for an agricultural field and monofill[2] which the reader might guess to be the period of application. A parameter with the same name is listed on page K-18 as having that meaning in the sensitivity analysis. However, CutOffYr is not mentioned elsewhere, so it is still unclear what is assumed for the period of biosolids use.

It is also not clear how the application period and exposure period interact, but it appears that they are (in the model) assumed to be almost independent — so farm management is treated as independent of the farmers who might come and go. There is an enigmatic "SY" parameter defined in Table C-1 (but nowhere explained) as the "start time exposure begins," assumed to be a uniform distribution "capped at the operating life of the unit." The exposure period (at least for adults) is taken from the distribution of time a farmer stays at one location, with most farmers staying on a single farm for short durations (Section 6.2.2.2). The effect appears to be that the application period (in the modeling) is highly likely to exceed the period of residence of the farmer who presumably determines whether or not such applications will occur at any particular location — an unlikely occurrence. It seems more likely that when farmers move, farm management practices will change.

[2] It is not clear why monofills and waste management units (WMU) are mentioned at various places throughout the TBD. Similarly, repeated reference to land application units (LAU) is confusing when agricultural fields are of interest.

This affects the distribution of results — it is likely that a larger farmer population is exposed than is modeled, but they are affected less on average, while the extremes of the distribution are less affected. Farmers who takes biosolids application practices with them to new farms will be less exposed because each farm is likely to start biosolid application afresh (only a small fraction of farms apply biosolids). More farmers may be affected because some farmers who do not use biosolids may arrive at a farm at which biosolids have been used before they got there. The extremes of the distribution will be driven by farmers who do not move and who use biosolids continuously for long periods. Given the small fraction of farms/farmers that use biosolids, it is unlikely that a farmer who uses biosolids will move to a farm where biosolids have been in long-term use.

Using the current approach, the 50th percentile estimate of risk could be overestimated by a large factor, but the effect on upper percentiles may be trivial. The effect on the estimates of total cancers would likely be trivial, because that is driven by the population total exposure (which is controlled mostly by the total amount of biosolids applied).

2.2 Methods and models

2.2.1 Method used to estimate the total number of cancers

The total number of cancers is estimated in both the PRE and in the FR (at 40568). In the former, the total population evaluated in the TBD is estimated, and this total population is multiplied by "an estimated lifetime risk of 6×10^{-6} from the TSD [sic]." The provenance of this risk estimate is unclear (for example, "TSD" is nowhere defined in any of the documents; and this risk estimate was apparently obtained prior to the last update of the TBD).

In the Federal Register, the methodology used in the PRE is also used to estimate the population evaluated in the TBD. Thereafter

> The number of lifetime cancer cases is estimated by multiplying the risk by the number of individuals in the modeled population. The estimated lifetime cancer cases for the modeled population is 0.224 if the 95th percentile adult risk from land application of sewage sludge (2×10^{-5}, see Table 5) is used for this calculation, and 0.112 using the 90th percentile adult risk (1×10^{-5}, see Table 5). The number of annual cases is estimated by dividing the lifetime cancer cases by 70 years of exposure. The estimated annual cancer cases is 0.006 if the 99th percentile adult risk is assumed, 0.003 if the 95th percentile adult risk is assumed, and 0.002 if the 90th percentile adult risk is assumed.

Thus the population risks have here been estimated by applying *to the whole population* the risk estimates corresponding to just the 90th, the 95th or the 99th percentile of that same population, since the distribution derived in the TBD (and reported in Table 5 of the FR) is the *variability*

distribution in the population evaluated. Such calculations are mathematically incorrect, being counter to the definition of a variability distribution.[3]

A cumulative variability distribution for risk, $F(R)$, is defined to be the fraction $F(R)$ of the population that has risks lower than R. The differential distribution $f(R)$ defined by

$$f(R) = \frac{dF(R)}{dR}$$

has the property that the fraction of the population with risks between R_1 and R_2 is

$$\int_{R_1}^{R_2} f(R)\,dR$$

The lifetime number of cancers in a population of size N is obtained by adding up the cancers in each fraction of the population subject to different risks. The number of persons that have risks in the range R to $R+dR$ is $Nf(R)\,dR$, so the lifetime number of cancers in that number of persons is $RNf(R)\,dR$. Adding up over all possible risks, to account for all members of the population, the lifetime total number n of cancers in the population of size N is

$$n = N\int_0^{\infty} Rf(R)\,dR = N\overline{R}$$

where the overscore represents "mean value," since the integral in this expression defines the mean value of risk. In words, the total number of cancers in a population of size N is the product of the population size and the mean value (the average over the variability distribution) of risk in that population.

Failure (as in the *FR* at 40568) to use the mean value of the variability distribution to calculate total numbers of cancers is simply a methodological error. Using the values from the upper end of the variability distribution, as given in the *FR*, gives a very large overestimate of expected number of cancers. The *FR* uses as examples the 90th, 95th and 99th percentiles of risk. While the TBD does not report the mean value of the risk distribution, I estimated it approximately from the percentiles given in Table 7.11 of the TBD (by fitting a lognormal distribution to those percentile values). The 99th percentile estimate is approximately 10 times the mean value, so the incorrect use of the upper percentiles in the FR leads to very large overestimates of the expected number of cancers.

[3] The procedure used in the FR could legitimately be used only if the distribution reported in Table 5 was an *uncertainty* distribution for the *mean value* of risk in the population.

2.2.2 The size of the population modeled

Neither the PRE nor the FR (at 40568), in their discussions of the size of the farmer population that is modeled in the TBD give any indication of where they obtain in the *Exposure Factors Handbook* (EPA, 1997) the statistics:

> Two percent are the "high end" modeled population that live on farms, raise their own crops and animals, and consume a significant portion of their annual diet from their farms. [PRE]

or

> ... 2 percent of the United States population are in farm families whose diets consist of 50 percent of products produced on their own farm (5.6 million people). [FR]

In the National Food Consumption Study analyzed in EPA (1997), an imputed population of 3,842,000 (Table 13-18) is estimated for households consuming home-produced meat (not only beef) among households in which at least one member operates a farm or ranch (Table 13-3), a number representing 2% of the imputed population of 188,019,000 covered in the study (Table 13-4).

For the major pathways evaluated in the TBD, however, what is required are the fractions of the population consuming home-grown beef or milk. For beef, Table 13-36 shows an imputed total population in farming households consuming home-grown beef of 2,850,000, only 38.89% of the imputed farm population covered by the survey and only 1.5% of the total population. For dairy products, Table 13-28 shows a total imputed population in farming households consuming home-produced dairy products of 1,020,000, a fraction of only 0.5% of the total population.

The population estimates in the PRE and FR are thus overstated by a factor of about 1.33 for beef, and 3.7 for milk; so the total cancer estimates will be overstated by an intermediate amount.

2.3 Fixed parameters

2.3.1 Carcinogenic potency

The carcinogenic potency of 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) is the basis for all the risk estimates. The value used in the TBD is based on observations in experiments on rats exposed to 2,3,7,8-TCDD in the laboratory, with an extrapolation to humans. At high doses 2,3,7,8-TCDD has been demonstrated to cause cancer in some rodents. It is much less certain whether, at the doses involved in the risk estimates of the TBD, 2,3,7,8-TCDD is either a rodent or a human carcinogen. The methodology used to extrapolate the observations at high doses in rodents to estimate risks at low doses in humans is designed to result in an overestimate of risks, possibly by a large factor (or even an infinite factor, if 2,3,7,8-TCDD is actually not a human carcinogen at low doses).

As noted in the FR, the EPA is in the process of re-estimating the carcinogenic potency of 2,3,7,8-TCDD, and may come out with a value that is about 6 times higher than the current estimate as representing a "best estimate," based on an interpretation of observations in humans exposed to relatively high doses of 2,3,7,8-TCDD at relatively high dose rates for relatively short

periods. However, even if the interpretations of human observations are correct, it is not clear that the same potency that EPA estimates is applicable to the low doses, low dose-rates, and long exposure periods included in the risk assessment of the TBD. If anything, the value obtained by EPA is likely to be an overestimate for such situations.

In addition, the TBD applies the potency of 2,3,7,8-TCDD, modified by various toxicity equivalency factors (TEFs), to all the other 2,3,7,8-substituted polychlorinated dibenzo dioxins and furans (PCDD/PCDF), and to certain planar PCBs. Except in a couple of instances, the TEFs are not based on observations of carcinogenic effects. Instead they are based on other measurements that may correlate to some degree with carcinogenicity. Thus major fractions of the risk estimates are not from PCDD/PCDF/PCBs for which there is any direct evidence for carcinogenicity (even in rodents), leading once again to a potential for a large overestimate of risk by imputing carcinogenicity to such compounds at the low doses and dose-rates estimated in the TBD.

2.3.2 Exposure frequency

All exposures are assumed to continue for 350 days/year. This may introduce an overestimate, since periods away from the farm are likely to exceed 2 weeks/yr. However, the overestimate is likely trivial, since farming households are unlikely to spend large fractions of the year off the farm.

2.4 Distributional parameters

2.4.1 Concentrations of dioxins in biosolids applied to land

In the TBD, the PCDD/PCDF/PCB concentration distributions measured in the biosolids survey have been treated as a variability distribution between fields for long-term average concentration of PCDD/PCDF/PCBs in the biosolids applied to the field. However, any single field treated more than once will not receive biosolids with identical PCDD/PCDF concentrations every time it is treated. It is likely to receive multiple "samples" of biosolids over a long period, possibly from different sources, so that any variation in PCDD/PCDF concentrations in such multiple samples will tend to average out.

The FR discusses the temporal variation in TEF concentrations of PCDD/PCDF/PCBs in biosolids in Section VIII (page 40559), and observes that there is evidence of variation over the long term in concentrations in biosolids even from the same source. However, the TBD did not take account of the few data on temporal variability that are available. The effect is to accentuate the extremes of the distribution of concentrations of PCDD/PCDF/PCBs applied to fields, and hence to accentuate the extreme values in the distribution of risks. Upper percentiles of long-term average TEF concentrations, and hence of risk, will be overestimated, while low percentiles will be understated. The mean and median estimates are unlikely to be much affected.

3 Major Exposure Pathways

3.1 Methods and models

3.1.1 Soil vapor emission model

The soil vapor emission model as described apparently has several major defects.

- the solution described is incorrect at the soil surface because it does not meet the boundary condition there,
- the boundary condition described is incorrect, and application of it will substantially overestimate emission rates of PCDD/PCDF/PCBs,
- the solution method fails to take adequate account of infiltration of rainwater, again resulting in an overestimate of vapor emission rates, and
- the solution method over-disperses material in the soil column.

Other defects of the soil modeling are relatively minor compared with these. Since the description of the soil column modeling is self-contradictory, and the implementation is not available, it is impossible to tell what model has been implemented. Thus it is currently impossible to tell from the available documentation the extent to which the soil model overestimates emission rates.

The TBD writes the correct differential equation for the given approximations at equation F-2-8. It is then stated that the solution used is equation F-2-16, which does not take account of rainwater infiltration or decay. Decay is correctly incorporated using F-2-23 (provided the decay rate is spatially invariant). However, the attempt to incorporate rainwater infiltration by a numerical technique fails to take account of infiltration of rainwater at short times after tilling, when the majority of emission occurs. It is thus likely to overestimate emissions.

It is stated at Section F-2.4.2.1 that "Zero concentration is assumed at the upper boundary of the soil column." However, the solution given, at equation F-2-8, does not satisfy this boundary condition anywhere (there is no location where equation F-2-8 is zero for all times). The description is thus self-contradictory.

It should also be noted that the statement "[t]he volatilization loss from the surface of the soil column, Mvol(t) (g/m2), is assumed to be due to gaseous phase diffusion only and is determined by . . ." on page F-10, and equation F-2-22, are both incorrect. It is not correct, as stated on page F-11, that "the contaminant mass in the gas phase volatilizes out the surface of the soil column, the contaminant mass in the aqueous phase is left behind." At the microscopic level, the entire assumption made in deriving equation F-2-8 is that there is equilibrium between vapor, dissolved phase, and adsorbed phase of the contaminant in the pore spaces of the soil. That equilibrium applies even at the soil surface, whatever the concentration in the gas phase (including zero concentration). It is fairly straightforward to show that this is a very good approximation.

Examination of equation F-2-16 and similar solutions of the diffusion equation F-2-8 shows that there is a characteristic time scale t associated with equilibration due to diffusion over distances x given by

$$t \sim \frac{x^2}{4D} \tag{3.1}$$

where D is the effective diffusivity in whatever medium the diffusion is occurring. For diffusion in air, $D \approx 4 \times 10^{-6}$ m^2/s for most organic chemicals (see, for example, Appendix D of the TBD). Thus for a typical soil pore dimension of order 100 μm or less, the characteristic time scale for diffusion equilibration is of order 1 ms or less. For the water film covering soil particles, typically of order 20 μm thickness or less, with a diffusivity in water of typically 4×10^{-10} m^2/s, the equilibration time is of order 0.1 s or less. The equilibration time for adsorption to the organic carbon on soil particles is also extremely fast, because the distances involved are molecular in scale. Thus, pore spaces come to diffusive equilibrium very rapidly.

On the other hand, for diffusion through the soil column (taking account of the equilibrium between soil water, organic carbon, and air), the effective diffusivity is given by Equation F-2-9a of the TBD. For example, with typical soil values, the effective diffusivity for 3,3',4,4',5,5'-HxCB is very low, about 4×10^{-15} m^2/s, because of the very high soil/water partition coefficient for this compound, giving a characteristic equilibration time across 1 cm thickness of soil of about 200 years. It follows that the soil pore spaces (air, water, organic carbon) can always be considered to be in equilibrium compared with the soil column concentrations, even at the soil surface. The material that diffuses to the surface in the pore water simply evaporates out to the gas phase, maintaining the equilibrium.

Equation 3.1 also allows a demonstration that application of a boundary condition of exactly zero concentration at the soil surface will substantially overestimate emission rates. Consider the same contaminant, 3,3',4,4',5,5'-HxCB, diffusing from soil that has just been turned over. After a few days, the depth to which 3,3',4,4',5,5'-HxCB will have evaporated is given approximately by the relation of equation 3.1. Inserting the value of effective diffusivity and a time of 30 days gives a depth of order 100 μm, around the thickness of one pore space. Now consider what is going on in the gas phase. The entire concentration difference (between equilibrium with the original soil concentration, and zero concentration in the air above the soil) is assumed to appear across 100 μm of air; and the flux through the gas phase can be calculated from this. However, above the soil surface is much more than 100 μm of air thickness — there is a stagnation layer probably centimeters thick (for pasture and cropland) through which the principal transport mechanism is diffusion. Omitting to take account of this stagnation layer results in overestimates of diffusion rates at short times (and here a "short" time may extend to months) of order 1 cm/100 μm, or 100-fold. Moreover, it is at such "short" times that the majority of emissions occur. Similar considerations apply for the other PCBs and the PCDD/PCDFs, since all have high values of soil-water partition coefficient and relatively low Henry's law constants.

The analytical "solutions" used by the TBD do not solve the required differential equation F-2-8 with the required boundary conditions. It is asserted in the TBD that separate solutions of the three equations F-2-13, F-2-14, and F-2-15 can be combined to obtain a solution of F-2-8. This is correct, up to a point. That is, suppose that $Q(z,t)$ solves equation F-2-13, and $Z(t)$ solves equation F-2-15, so that

$$\frac{\partial Q}{\partial t} = D\frac{\partial^2 Q}{\partial z^2} \qquad \frac{dZ}{dt} = -kZ \tag{3.2}$$

(subscripts on the various symbols appearing in Equation F-2-8 have been removed for ease of exposition). Then if $G(z,t)=Q(z,t)Z(t)$, it follows that

$$\frac{\partial G}{\partial t} = \frac{\partial Q}{\partial t}Z + Q\frac{dZ}{dt} = ZD\frac{\partial^2 Q}{\partial z^2} - kQZ = D\frac{\partial^2 G}{\partial z^2} - kG \tag{3.3}$$

Now if I define

$$C(x,t) = G(x - Vt, t) \tag{3.4}$$

then $C(x,t)$ will satisfy equation F-2-8.

However, this demonstration has ignored the boundary conditions under which F-2-8 has to be solved. The boundaries are stationary with respect to the soil column, so that even if the original solution F-2-16 satisfied the required boundary conditions, which it does not, the modified solution would not — a solution of the form given in equation 3.4 cannot represent a solution with a constant concentration at a boundary that is fixed in space. And the solution proposed in the TBD also does not satisfy the stated boundary condition of zero concentration at the surface, nor any other suitable boundary condition at the surface. In short, the solution is an incorrect representation of the physical situation of the soil column. This is unfortunate, because it is not difficult to write down solutions of F-2-8 that do satisfy physically realistic boundary conditions at the soil surface. Jury *et al.* (1983, 1984a, 1984b, 1984c, 1990, 1992) wrote down such a solution, including the physically realistic requirement (necessary in this application, as discussed above) for diffusion through a stagnant air boundary layer at the surface. Not surprisingly, the solutions they obtain do not have the form of equation 3.4. The failure of the TBD to use the published solutions at least for the surface component of the soil column model is troubling; what has been used, according to the TBD documentation, is not physically realistic.

The numerical implementation of the model also does not accurately represent the effect of infiltrating rainwater. The implementation estimates diffusion from the surface layer during the first time step (possibly several time steps) as if there is no infiltration at all. However, the effect of infiltration is to move the concentration down into the soil column, dramatically reducing the concentration gradient at the surface — and it is the concentration gradient at the surface that

controls diffusion emission rates. Omission of the infiltration rate term can have a dramatic effect on emission rate estimates. However, for the PCDD/PCDF/PCBs evaluated in the TBD, the effect is small, since the "effective" infiltration rate (equation F-2-10) is so small.

The numerical implementation of the diffusion equation is also incorrect in that it necessarily leads to overestimates of vertical dispersion in the soil column. The driving force for dispersion in the soil column is diffusion driven by the gradient of concentration, and the effect of diffusion is to reduce such concentration gradients, smoothing out fluctuations in concentration. The numerical method adopted has the effect of completely smoothing the concentration gradient (reducing it to zero) in each soil layer at the beginning of each time step, while imposing substantial gradients (steps) in concentration between soil layers at the beginning of each time step. The within-layer smoothing increases the effective dispersion within the layer, and the imposition of very high gradients (at the concentration steps) artificially increases dispersion between layers. For example, the concentration gradient estimated in the model at the junction between layers will always be higher than reality, because of the imposition of the step profile at the beginning of each time step. The model will thus always overestimate transport between layers.

3.2 Fixed parameters

3.2.1 Absorption of dioxins by a mother

The assumption of 100% absorption of PCDD/PDCF/PCBs by a mother ensures that the estimated dose to her child is overestimated. However, the effect is likely to be small, since dioxin absorption in foods is expected to be high. The exposure of a child is currently calculated as trivial in the TBD.[1] However, if correctly analyzed, the child exposure route might contribute a larger fraction to multipathway risk estimates.

3.2.2 Food preparation losses

Food preparation losses for beef are based on observed weight losses during cooking and post-cooking operations. Since PCDD/PCDF/PCBs are preferentially concentrated in the fat, the loss of PCDD/PCDF/PCBSs is likely to be more closely proportional to the fraction of fat lost during cooking and post-cooking operations, rather than the fraction of total weight loss. A substantial fraction of the losses during cooking of beef is due to loss of fat, and fat is likely to be preferentially trimmed from beef in post-cooking operations. Any such preferential loss of fat is not accounted for when using the observed total weight loss, as in the TBD, so that the TBD will overestimate PCDD/PCDF/PCB intake in beef. However, the overall effect on risk estimates is likely to be small.

[1] Approximate calculations using the model described in the TBD give lifetime risk estimates of about 1/4 to 1/3 the multipathway estimates obtained in the TBD, agreeing with literature estimates (Smith, 1987). Because I do not have access to the implementation of any of the models, I cannot determine why the TBD makes this error.

3.3 Distributional parameters

3.3.1 Food survey bias

The food intake distribution estimates are based on short-term (three to seven day) observations in individuals. What is required are distributions of long-term average intakes for individuals. Use of short-term measurements result in a bias, because short-term observations probably do not represent long-term averages. This bias is discussed in the Exposure Factors Handbook (EPA, 1997, page 13-3), and an approach designed to overcome some portion of the bias was applied for major food groups. However, the TBD has used unadjusted distributions for beef and milk consumption.

The effect of this bias is twofold. First, it results in an underestimate of the fraction of consumers of home grown material, since some consumers might be missed in the short survey interval. Second, the extremes of the distribution are accentuated — short-term fluctuations in individual eating habits are mixed up in the distribution with long-term average consumption rates. For example, a farm household may have eaten beef for every meal during the survey period, because an animal had just been slaughtered; but that may overstate the long-term average consumption of the individual who took part in the survey. Similarly, other families with irregular consumption of beef may have been caught by the survey at a time when no home-produced beef was available, resulting in an underestimate of their consumption.

For the risk estimates, mean and median estimates are unlikely to be substantially affected by this bias, but extremes of the variability distribution may be affected to a small to large degree.

3.3.2 Correlations between milk and beef consumption

In the TBD, particularly in the multipathway assessment (in which risk estimates for all pathways are summed), it is assumed that consumers of home-produced beef are also consumers of home-produced milk. This cannot be true for all farmers consuming home-produced beef, since while about 40% of farmers consume home-produced beef, only about 14% consume home-produced dairy products (EPA, 1997, Tables 13-28 and 13-36; see also Section 2.2.2). The principal effect of this failure to account for the correlation between milk and beef eating is on the shapes of the distributions of risk, rather than on average estimates of risk. In particular, the upper ends of the estimated risk distribution are likely to be increased by a small to trivial factor.

4 Minor exposure pathways

The total effect of all minor pathways on risk is trivial; so the estimated size (trivial, small, large, or very large) of the effect of parameter choice on risk in this section is scaled to represent the effect on the particular pathway mentioned.

4.1 Scenarios

4.1.1 Worst-case farm layout

The scenario adopted appears to correspond to approximately the worst possible case. The entire farm is assumed to use biosolids; all runoff from biosolids-using areas runs over areas (the buffer areas) that act as sources for the exposure pathways (*e.g.* over areas where chickens are raised, and where children are assumed to be exposed to soil), and all the runoff affects the same stream, which is used by the farmer for fishing. The physical setting thus always corresponds to the farm being laid out so that the buffer areas (where the farm is supposed to be located and all other farm activities take place) are downhill of all crops and pastures. The result for the minor exposure pathways is to maximize their potential effect, introducing overestimates that probably range from small to very large.

4.2 Methods and models

4.2.1 Soil erosion modeling

The discussion of soil erosion is in Appendix F of the TBD. The "Spatial implementation" in Section F-3.3.3 indicates that the analysis is limited to a hillside situation where the entire pasture and crop area lies above the farm and "buffer" area. The entire approach described in F-3.3.3 appears to be completely ad hoc — no references are provided to indicate that this has any support in the literature, and there is no indication of any verification of the approach. The "sediment delivery ratio" approach in particular attempts to apply to small areas (less than the size of a field) a concept that was developed for entire watersheds, and for which the empirical relation (equation F-3-21) has not been validated or tested on such a small scale. There is no provision in the erosion modeling described in Section F-3.3.3 for the situation where sheet flow stops at some sub-area, and then re-starts at a later one, as can happen with the methodology previously described for runoff in Section F-2.3.2 (and should happen physically). That is, there appears to be no connection between the modeling of water flow, and of the erosion caused by that water flow.

Subsequently, the various inconsistencies identified in the modeling are summarized in the TBD, and for my purpose I can just quote the conclusion "Contamination in a downslope buffer would be overestimated." Instead of fixing the problem, however, it is decided to adopt a simpler solution and simply re-define the "conceptual model" so that "The conceptual module corresponding to this approach is that the runoff water itself may be diverted by swales or

ditches, but the soil and chemical being eroded are maintained on the local watershed surface, to be transported downslope over time across the buffer and into the waterbody." In other words, it is simply assumed that anything eroded off the pasture or cropland of the farm must traverse the buffer area, no matter what the real physical situation. Indeed, the assumption is physically impossible, since it assumes that the same processes that moved the eroded material down to the buffer area suddenly stop operating when that eroded material reaches the buffer area.

Thus all the minor routes of exposure that have as their source the contamination of the buffer zone or the waterbody are necessarily overestimated, by factors that cannot be evaluated.

4.3 Fixed parameters

4.3.1 Contamination fraction in fish

The assumption that all home-caught fish consumed by the fishing farmer is contaminated results in a large to very large overestimate of risks by this (trivial) route, with the upper end of the distribution particularly affected. While there may be fishing farmers who only fish water bodies on or adjacent to their farms, it is highly unlikely that all do.

4.3.2 Contamination fraction for ingested soil

All soil ingestion by children is assumed to occur on the buffer zone (where soil is contaminated mainly by erosion from pasture and cropland), while all soil ingestion by adults is assumed to come from cropland to which biosolids have been applied. While there may be farmers to whom such conditions apply, they are unlikely to occur for all farmers modeled. The effect is a distortion of the distribution of risk estimates by this route, with the upper end of the risk distribution particularly affected. The effect is likely to be small to large, but on an exposure pathway that contributes trivially.

4.3.3 Soil ingestion rates

The standard regulatory assumptions of 100 mg/day for young children, and 50 mg/day for adults, are used. Evidence indicates that these regulatory assumptions overestimate actual average ingestion rates. The largest available study in children (Stanek and Calabrese, 2000) indicates an average in children of about 31 mg/day, with a long term average 95th percentile of about 106 mg/day. There are only two studies that have attempted to measure adult average soil ingestion rates (Calabrese *et al.*, 1990; and Stanek *et al.*, 1997), the former in six adults, the latter in ten. The U.S. EPA recommendation of 50 mg/day is based principally on Calabrese *et al.* (1990). The later study by Stanek *et al*. (1997) is said to "suggest lower levels of soil ingestion in adults than previous studies," based on 280 subject-days (10 subjects × 28 days) of evaluation, the largest amount of data available on soil ingestion in adults. Stanek *et al*. (1997) estimated that the average adult ingested 10 mg/day (although the uncertainty is large). The effect of the overestimate of average soil ingestion rate is likely to be small to large, but on a trivial route.

4.3.4 Food preparation losses

Food preparation losses are estimated from weight losses during food processing. However, for many vegetables (root vegetables in particular) PCDD/PCDF/PCBs tend to be sorbed to the surface. Preparation losses for such vegetables may be higher, particularly for those that are peeled or have surfaces removed. The losses will therefore be higher than indicated by weight loss alone, so the loss is underestimated and the risk overestimated. The effect is likely to be trivial to very large, depending on particular food involved.

4.3.5 Soil mixing depth

In the buffer area, all deposited contaminants are assumed to be mixed into the top1cm of soil. However, such a small mixing depth (particularly on time scales up to 70 years) would be characteristic only of dead, undisturbed soils. Even at the surface of dead soils, physical processes like rain splash, freeze-thaw cycles, and thermal cycling are likely to mix surface soil fairly effectively in any locations appropriate for farming. In such locations also, the untilled buffer areas are likely to be highly populated by small animals (*e.g.* worms, ants) that burrow, resulting in relatively high soil turnover rate that acts like a diffusivity, mixing soils to considerable depths. The assumption of such a small soil mixing depth results in an overestimate of risks that is small to large for several of the trivial exposure routes.

4.4 Distributional parameters

4.4.1 Correlations between food ingestion rates

As for beef and milk, other food ingestion rates are treated as uncorrelated for different home-produced foods — the estimates obtained for consumers of each food are assumed to apply independently to each farmer,[2] even though the fraction of farmers eating such home-produced foods differ, so it is impossible for all farmers to eat all such foods. The effect of the assumption is probably to overestimate of food consumption and hence risks by a trivial to small factor, but for pathways that are trivial.

[2] It appears that a "fraction home-produced" has been applied to the distributions of food intakes for consumers; but applying such a fraction is incorrect, and does not account for the correlations between different foods.

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