
**Review of Overestimates
in the Risk Assessments Supporting
Standards for the Use or Disposal
of Sewage Sludge
Notice of Data Availability
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Association of Metropolitan Sewerage Agencies*

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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 monofill² 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

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

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, $M_{vol}(t)$ (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

$$t \sim \frac{x^2}{4D} \quad (3.1)$$

where D is the effective diffusivity in whatever medium the diffusion is occurring. For diffusion in air, $D \approx 4 \times 10^{-6} \text{ m}^2/\text{s}$ for most organic chemicals (see, for example, Appendix D of the TBD). Thus for a typical soil pore dimension of order $100 \text{ }\mu\text{m}$ 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 $20 \text{ }\mu\text{m}$ thickness or less, with a diffusivity in water of typically $4 \times 10^{-10} \text{ m}^2/\text{s}$, 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 $4 \times 10^{-15} \text{ m}^2/\text{s}$, 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 $100 \text{ }\mu\text{m}$, 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 $100 \text{ }\mu\text{m}$ of air; and the flux through the gas phase can be calculated from this. However, above the soil surface is much more than $100 \text{ }\mu\text{m}$ 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 $1 \text{ cm}/100 \text{ }\mu\text{m}$, 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

$$\frac{\partial Q}{\partial t} = D \frac{\partial^2 Q}{\partial z^2} \quad \frac{dZ}{dt} = -kZ \quad (3.2)$$

(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

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

Now if I define

$$C(x,t) = G(x - Vt, t) \quad (3.4)$$

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/PCBs 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 top 1 cm 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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