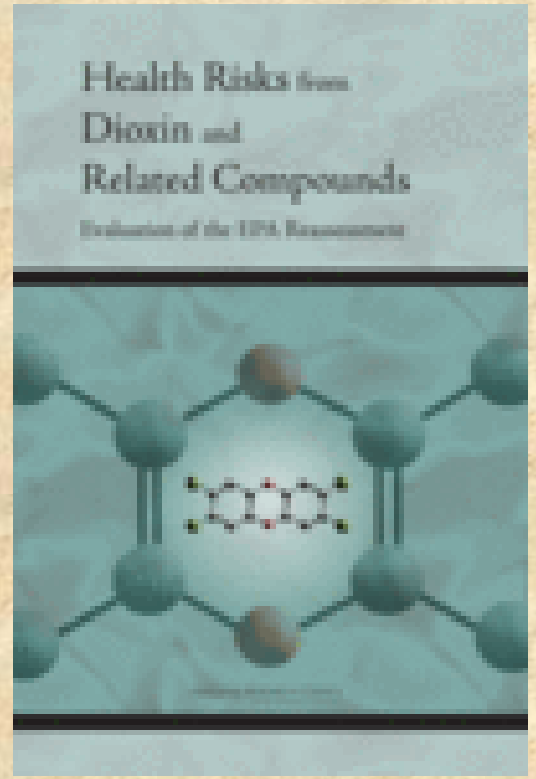
NAS/NRC Report: Health Risks from Dioxin and Related Compounds



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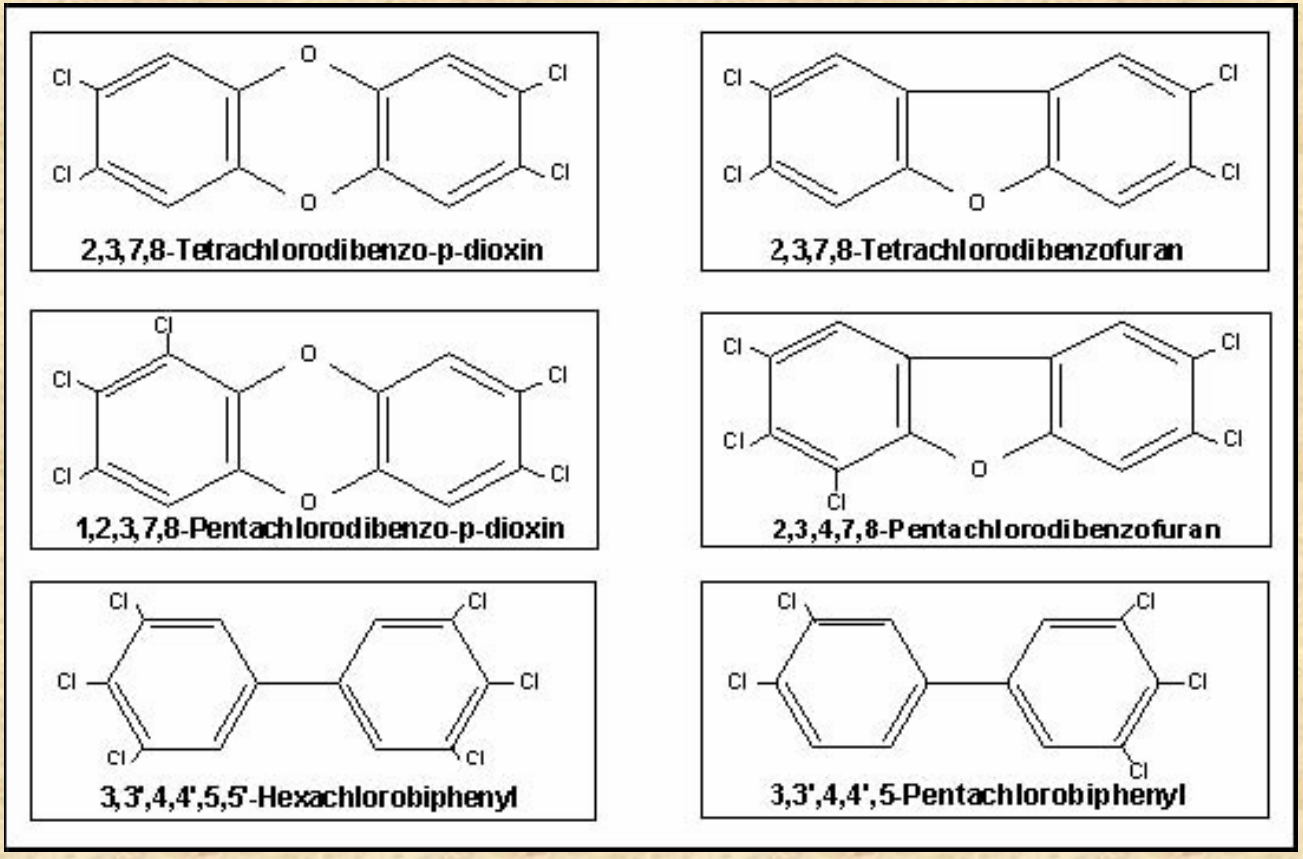
Dioxins, Dibenzofurans and PCBs

- Chlorinated Dioxins represent a class of compounds, of which 7 are included in EPA regulations (Contaminants – no commercial use)
- Chlorinated dibenzofurans include 10 congeners (formed from PCBs)
- Certain ‘planer’ PCBs also have ‘dioxin-like’ activity, and are included.



Toxicology of ‘Dioxin’ in 1 Minute

- Very toxic, both acute and chronic
  - LD50 0.6 ug/kg in sensitive species
  - Chronic – birth defects, immunotoxicity, cancer, chloracne, reproductive effects, liver, CNS
  - Large species differences
- Mechanism of all (or nearly all) toxic effects is by binding to the Ah Receptor
  - Transcriptional activation of numerous genes, especially CYP1A1
  - Toxic effects are result of ‘downstream’ events that follow Ah Receptor activation
- Very fat soluble, resistant to degradation
  - Persistent in the environment
  - Bioaccumulate
  - Long biological half life (about 6-7 yrs in humans)

Known Effects of TCDD in Humans

Before Dioxin poisoning

After Dioxin poisoning

Yuschenko's blood TCDD concentration was ~ 100,000 pg / g lipid

Known Effects of DLCs in Humans

- Yusho and Yu-Cheng Disease in Japan
  - Massive PCB/PCDF exposures via diet
- Michigan nursing mothers exposed to PCBs via fish consumption
- Dutch cohort of off-spring of moderately exposed mothers (DLCs, mostly PCBs)
  - Developmental abnormalities following in utero exposures, mostly neurobehavioral and cognitive deficits
  - Some evidence of immune system dysfunction
  - Some evidence of hormonal/endocrine dysfunction
- Occupational exposures – possible increase in cancers

Dioxins, Diobenzofurans & PCBs Toxic Equivalence Factors (TEFs)

Table 1-3. The toxic equivalency factor (TEF) scheme for TEQ_{DFP}-WHO_{98}[a]

Dioxin congener	TEF	Furan congener	TEF	Dioxin-like PCB	TEF
2,3,7,8-TCDD	1.0	2,3,7,8-TCDF	0.1	PCB-77	0.0001
1,2,3,7,8-PeCDD	1.0	1,2,3,7,8-PeCDF	0.05	PCB-81	0.0001
1,2,3,4,7,8-HxCDD	0.1	2,3,4,7,8-PeCDF	0.5	PCB-126	0.1
1,2,3,6,7,8-HxCDD	0.1	1,2,3,4,7,8-HxCDF	0.1	PCB-169	0.01
1,2,3,7,8,9-HxCDD	0.1	1,2,3,6,7,8-HxCDF	0.1	PCB-105	0.0001
1,2,3,4,6,7,8-HpCDD	0.01	1,2,3,7,8,9-HxCDF	0.1	PCB-118	0.0001
1,2,3,4,6,7,8,9-OCDD	0.0001	2,3,4,6,7,8-HxCDF	0.1	PCB-123	0.0001
		1,2,3,4,6,7,8-HpCDF	0.01	PCB-156	0.0005
		1,2,3,4,7,8,9-HpCDF	0.01	PCB-157	0.0005
		1,2,3,4,6,7,8,9-OCDF	0.0001	PCB-167	0.00001
				PCB-114	0.0005
				PCB-189	0.0001

History – EPA Dioxin Risk Assessment

- 1985 – Completed first risk assessment of Dioxin
  - Classified as potent, 'likely' human carcinogen
  - linear extrapolation, mg/kg-d dose metric
  - Controversial assumptions
- 1991 – EPA announces that it will Reassess Dioxin Risks
- 1994 – First draft of Reassessment released
  - Basically supported findings of 1985 assessment
  - Used body burden as dose metric
  - Extensive peer review and public comments raised concerns about models and assumptions
- 2000 –Revision of 1994 Reassessment released
  - Additional Peer review and SAB comments
  - Questions about linear model for cancer
- 2003 – Revised 'Near Final' Reassessment
  - Requested the National Academies to do review of Reassessment
- 2004 – NAS/NRC appoints panel, begins review (Nov, 04)
- 2006 – NAS/NRC report released (July)

Why is this important?
Policy implications

- Many industries, (pulp and paper, chemical manufacturers, incinerators, etc), have dioxin emissions that are regulated
  - Emissions standards will be based on risk
- Many state and federal (Superfund) hazardous waste sites contain dioxins/DLCs
  - clean-up standards will be based on risk
  - State agencies can set own standards (if stricter than feds)
- Draft Reassessment suggested that there was potentially unacceptable cancer risks at <u>current</u> <u>background</u> levels
  - Implications for regulation of foods, especially meat and dairy
- Some regulations have been ‘on hold’ pending acceptance of a final EPA Reassessment

The NAS/NRC Process
Selecting the Committee

- Committee of ‘highly respected’ scientists, with all relevant areas of expertise represented
  - Not necessarily ‘experts’ on dioxin, but have high level of credibility in their discipline
- Full disclosure of potential conflicts and biases
- Not involved in preparation or review of the EPA Reassessment
- Committee selected by the NAS leadership, following detailed ‘vetting’ of information on nominees
- Tentative Committee becomes ‘final’ committee after 1st meeting, when bias and conflict of interest are discussed

End Result – Committee of 18

- Dave Eaton, PhD, UW (Chair)
- Dennis Bier, MD, Baylor
- Joshua Cohen, PhD, Harvard (now at Tufts)
- Mike Dennison, PhD, UC-Davis
- Rich DiGiulio, PhD, Duke
- Norb Kaminski, PhD, Mich. St.
- Nancy Kim, PhD, NY St DoH
- Djien Liem, PhD, European Food Safety Authority, Italy
- Tom McKone, PhD, UC-B
- Malcolm Pike, MD, USC
- Alvaro Puga, PhD, U Cinn.
- Andy Renwick, PhD, Univ. Southampton, UK
- David Savitz, PhD, UNC (now at Mt. Sinai)
- Allen Silverstone, PhD, SUNY-Upstate (Syracuse)
- Paul Terranova, MD, KUMC
- Kim Thompson, PhD, Harvard (now at MIT)
- Gary Williams, MD, NYMC
- Yilang Zhu, PhD, U. S. Florida

Members highlighted in blue have spent much of their careers on dioxin toxicology

Committee Charge

The NAS/NRC will convene an expert committee that will review EPA's 2003 draft Reassessment….to assess whether:

- Risk estimates are scientifically robust
- There is a clear delineation of all substantial uncertainties and variability
- To the extent possible, focus on:
  - Modeling assumptions (shape of D-R curve, points of departure, dose ranges for likely human health outcomes)
  - EPA's quantitative uncertainty analysis
  - EPA's selection of studies as a basis for its assessments
- Also address:
  - Scientific evidence classifying TCDD as a human carcinogen
  - Validity of the non-threshold, linear D-R model and slope factors
  - Usefulness of TEF/TEQ approach

Other Conditions / Limitations

- Complete the review in 18 months
  - including peer review, revisions, and final editing
- Solicit public input prior to writing report
- Strive for a ‘Consensus’ report
- Have no more than 5-6 meetings
- Draft report subject to extensive peer review
- Final report must include consideration of all peer review comments
- We focused our on review Part III of the Reassessment
  - “Integrated Summary and Risk Characterization”
  - ~200 page summary of the several thousand page report
- Considered ‘new information’ only if it was critical to key assumptions, and likely to change the RA

Process

- 2 meetings with invited presentations
  - Heard from 16 different ‘interested parties’
  - Received piles of solicited and unsolicited information
- Organized the report to include 8 chapters:
  1) Introduction
  2) General Considerations of Uncertainty and Variability, Selection of Dose Metric, and Dose-Response Modeling
  3) Toxic Equivalency Factors
  4) Exposure Assessment
  5) Cancer
  6) Non-Cancer Endpoints (Immune, Repro/Development, Other)
  7) Risk Characterization
  8) Conclusions and Recommendations
- Chapter review assignments made by Chair, based on areas of expertise – 3-4 members per topic
- 2 meetings to discuss recommendations
- 1 meeting to finalize report conclusions and consensus
- Most of the real work was done by e-mail

Process (cont)

- Consensus draft report completed in December, 2005
- Edited by staff then sent to 15 different peer reviewers
- 3 months later, received ~120 pages of comments
- Made numerous changes (requiring approval of all committee members) and submitted revised to NAS/NRC Study Monitor
  - along with detailed list of how document was changed in response to comment, and if not, why not
- After approval of Study Monitory, Final draft report sent to NRC staff for editing and printing
- Congressional Briefings and Press Conference held on day before report was released (July 11, 2006).

Key Findings of The Committee

- “3 areas that require substantial improvement in describing the scientific basis for EPA’s dioxin risk assessment”
  - Justification of approaches to D-R modeling
  - Transparency and clarity in selection of key data sets
  - Transparency, thoroughness and clarity in quantitative uncertainty analysis
- Classification of TCDD as known vs. likely human carcinogen
  - Use the new definitions in 2005 CAG
- TEFs continue to be best approach for assessing mixtures
- Encouraged EPA to calculate RfD and MOE scenarios

Key issue – Qualitative Assessment of TCDD carcinogenicity to humans

- Seems to be a ‘big deal’ to lots of people
- Committee felt that it really was not important, as TCDD will (and should) be regulated as if it is carcinogenic to humans regardless of what label it is given
- Better off spending time on more critical uncertainties that will affect the quantitative risk estimations
- Guidelines and definitions changed in 2005 – EPA should use new guidelines, then justify their decision
- Committee was ‘split’ on whether the available data met the criteria of the new guidelines
  - Full agreement that it was at least likely to be carcinogenic in humans
  - Other DLC congeners ‘Likely’ to be carcinogenic to humans

"Carcinogenic to Humans"
EPA Carcinogen Assessment Guidelines 2005

This descriptor indicates strong evidence of human carcinogenicity.

This descriptor is appropriate when there is convincing epidemiologic evidence of a causal association between human exposure and cancer.

Exceptionally, this descriptor may be equally appropriate with a lesser weight of epidemiologic evidence that is strengthened by other lines of evidence. It can be used when all of the following conditions are met:

(a) there is strong evidence of an association between human exposure and either cancer or the key precursor events of the agent's mode of action but not enough for a causal association, **and**

(b) There is extensive evidence of carcinogenicity in animals, **and**

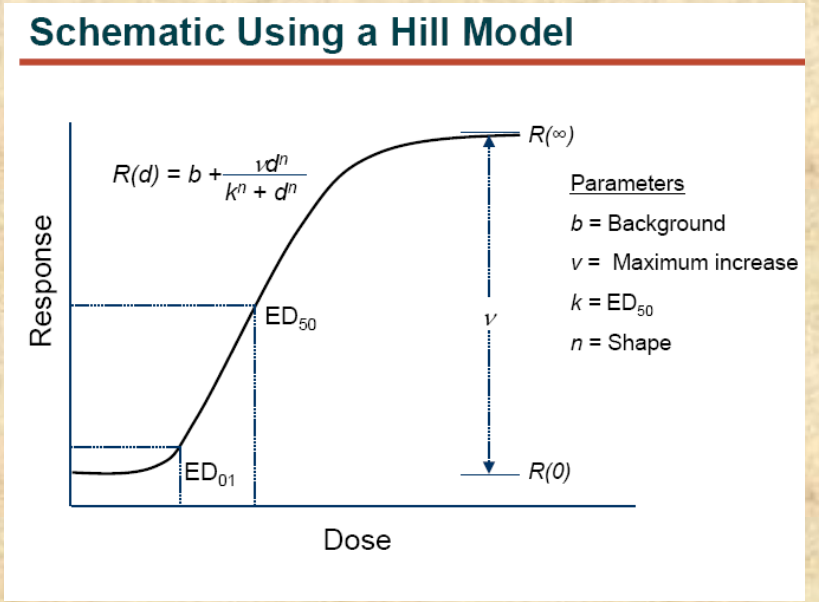
(c) the mode(s) of carcinogenic action and associated key precursor events have been identified in animals, **and**

(d) there is strong evidence that the key precursor events that precede the cancer response in animals are anticipated to occur in humans and progress to tumors, based on available biological information.

Epidemiological Evidence

- Four occupational cohorts with substantial TCDD exposure
  - Ott & Zober, 1996 – 1953 accidental exposure (N=243, 13 cancer deaths)
  - Becher et al 1998 – Pesticide production cohort (N=1189; 124 Ca deaths)
  - Fingerhut et al (’90, 91) – 12 manufacturing facilities (N=5172; 377 deaths)
  - Steenland et al (2001) – update on Fingerhut cohort (N=3538; 256 deaths)
  - De Mesquita et al (1993) – Phenoxy production (N=2310, 31 cancer deaths)
- Most, but not all, found significant increase in all cancers, but no consistent increase in any specific tumor type
- Committee conclusions:
  - Overall, the committee concurs with the value of conducting analyses of total cancers given the potential for dioxin to affect multiple types of cancer
  - “It was the Committee’s impression that EPA’s narrative tended to focus on positive findings without fully considering the strengths and limitations of both positive and negative findings.”

Key Issue – Shape of the D-R Curve



- Mode of action – Receptor-mediated for all end points
- Non-genotoxic
- Evidence of tumor promotion
- Binding to Ah receptor is necessary, but not sufficient, to cause cancer
- Existing animal and human epi data provide little guidance as to the shape of the D-R at response levels below 5-10%

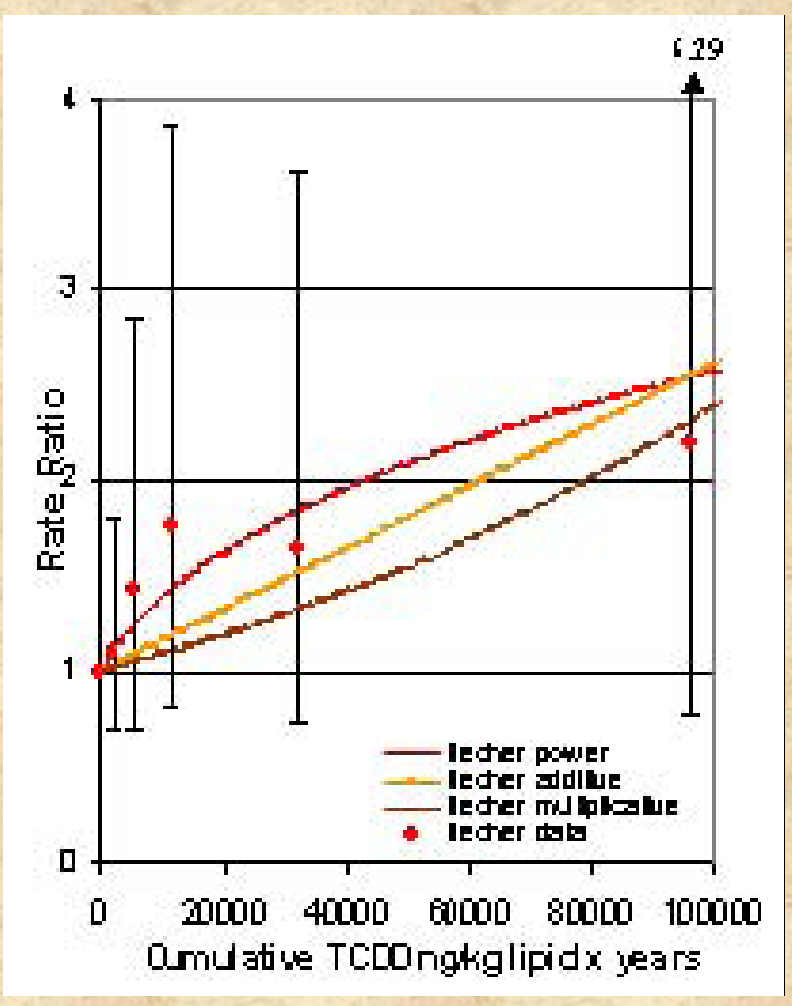
Rationale for EPA's Choice of Linear, non-threshold model

- *"At this time, the knowledge of the mechanism of action of dioxin, receptor theory, and the available dose-response data do not firmly establish a scientific basis for replacing a linear procedure for estimating cancer potency."*
- *"The linear default is selected on the basis of the agent's mode of action when the linear model cannot be rejected and there is insufficient evidence to support an assumption of non-linearity"*
- Committee disagreed with using the 'default' assumption, given the enormous amount of data on dioxin mode of action, and noted that EPA had used non-linear modeling for other receptor-mediated carcinogens (thyroid carcinogens, estrogens, etc.)
- Recommended that they do BOTH, to illustrate the importance of this assumption
- If they choose to use the linear estimates out of 'precaution', that would be a policy decision, with the implications clearly described

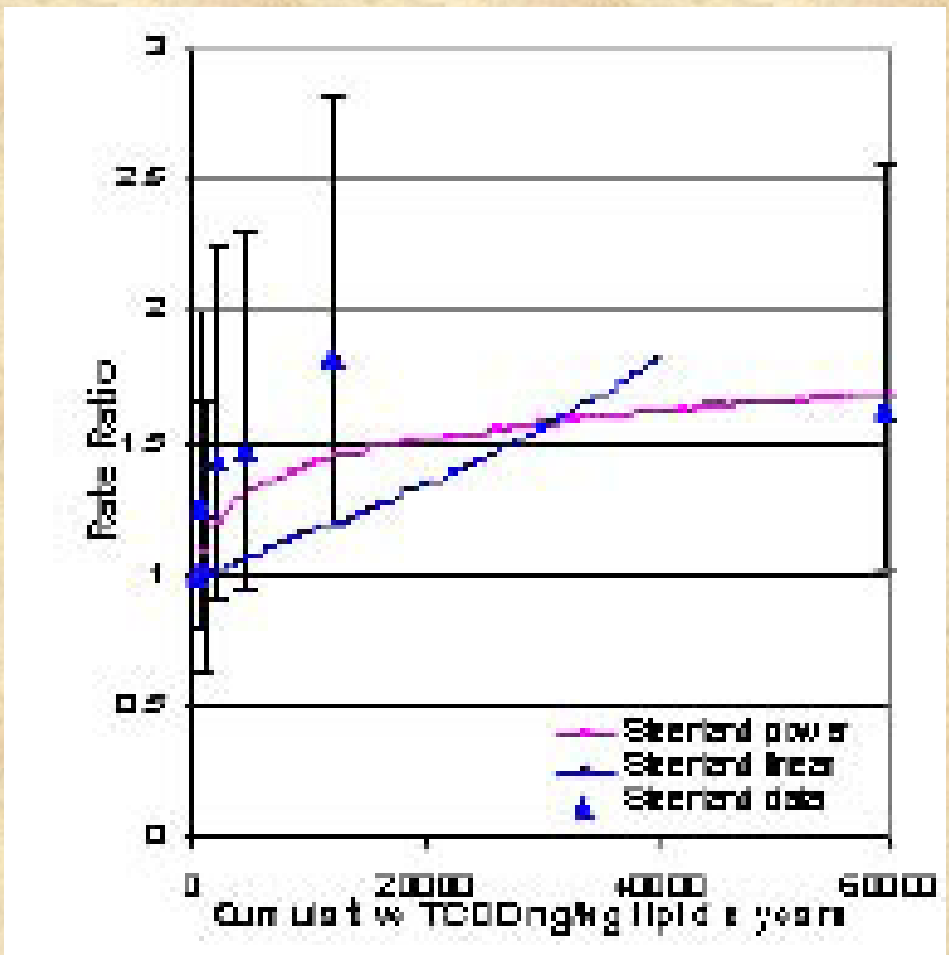
Key Issue – Point of Departure

- EPA used a POD of 1%: *"The curve-fitting procedure is used to determine a POD, generally at the 10% response level, but when more sensitive data are available, a lower point for linear extrapolation can be used to improve the assessment (e.g., 1% response for dioxin, ED01).*
- They calculated 1% PODs from the epidemiology data, using various models
- Use of ED05 would greatly expand the Confidence Limits around the central estimate (from which the Slope factors are derived)
- "It is evident that the choice of POD can have a substantial impact on the uncertainty of the final risk estimate – importance of this assumption is not readily evident in the Reassessment""

EPA Modeling of Cancer Data for ED01

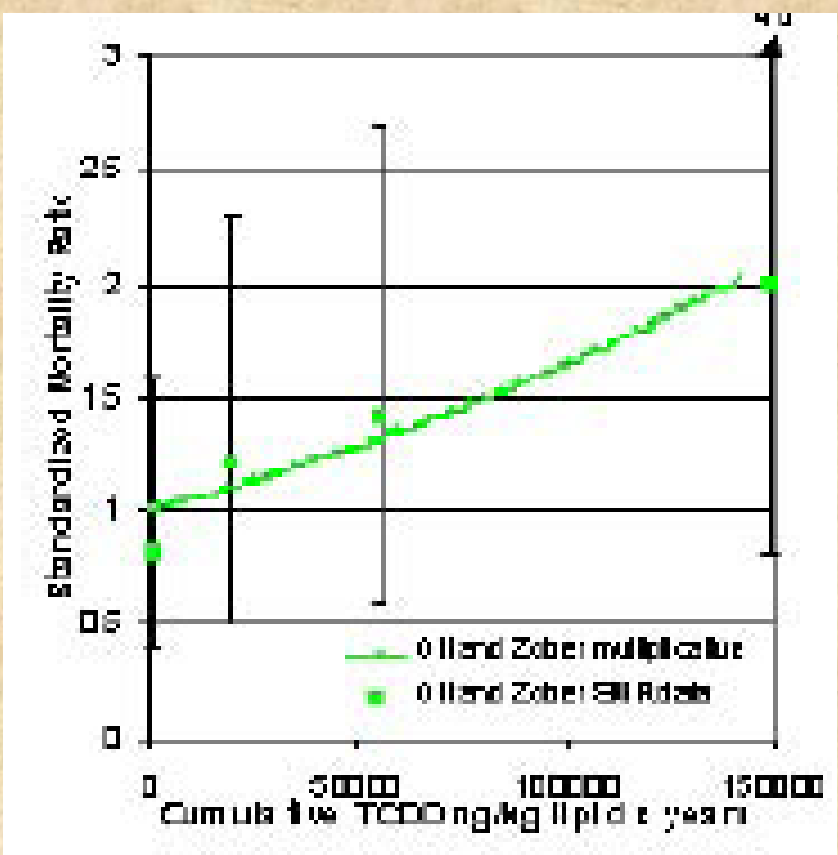


Mean Serum TCDD: 507 (2 – 6397 ppt)



Mean Serum TCDD: 2000 (2 – 32000 ppt)

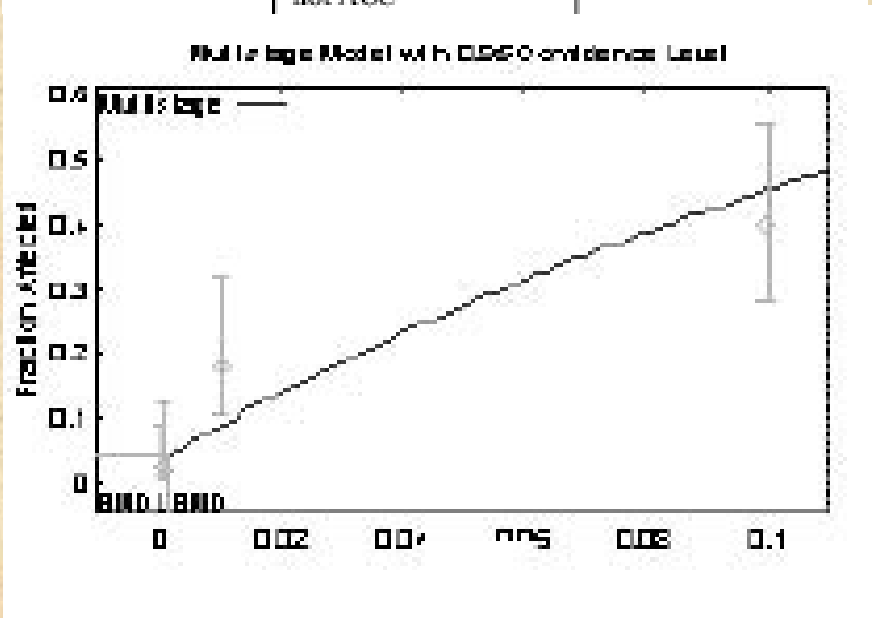
EPA Modeling of Cancer Data for ED01



BASF cohort

Mean Serum TCDD: 1008 (20 – 13360 ppt)

Exposure groups	Central estimate of range (ng/kg fat x years)[a]	All cancer deaths observed (latency)
0	0	2/86 Tumors
0.001	540	1/50
0.01	1700	9/50
0.1	8100	18/45
µg/kg/day	ng/kg lipid, not AUC	



Male Sprague-Dawley Rat data

Cancer Slope Factors Derived from ED01 modeling

Table 5-3. All cancer risk in humans through age 75[a]

Study	Model and Sex	ED_{01}	95% CI (lower, upper)	Unit excess risk for 1 ppt body burden above background
Steenland et al. (2001)	power male	1.38	0.71, 8.95	0.0079 (0.0027, 0.0132)
	power female	1.84	0.92, 14.9	0.0064 (0.0022, 0.0107)
	piecewise linear male	18.6	11.5, 48.3	0.00052 (0.00020, 0.00084)
	piecewise linear female	23.1	14.3, 59.8	0.00042 (0.00016, 0.00067)
Becher et al. (1998)	power-male	5.971		0.0018
	power-female	7.58		0.0014
	additive-male	18.22		0.00055
	additive-female	22.75		0.00044
	multiplicative-male	32.16		0.0003
	multiplicative-female	39.82		0.00024
Ott and Zober (1996)	multiplicative-male	50.9	25.0, ∞	0.00019 (0, 0.00039)
	multiplicative-female	62.1	30.5, ∞	0.00015 (0, 0.00032)

[a] Units are constant body burden in ng/kg not adjusted for lipid: see Part III, Chapter 8, Table 8-2, for details.

EPA Conclusions for Cancer Slope Factors

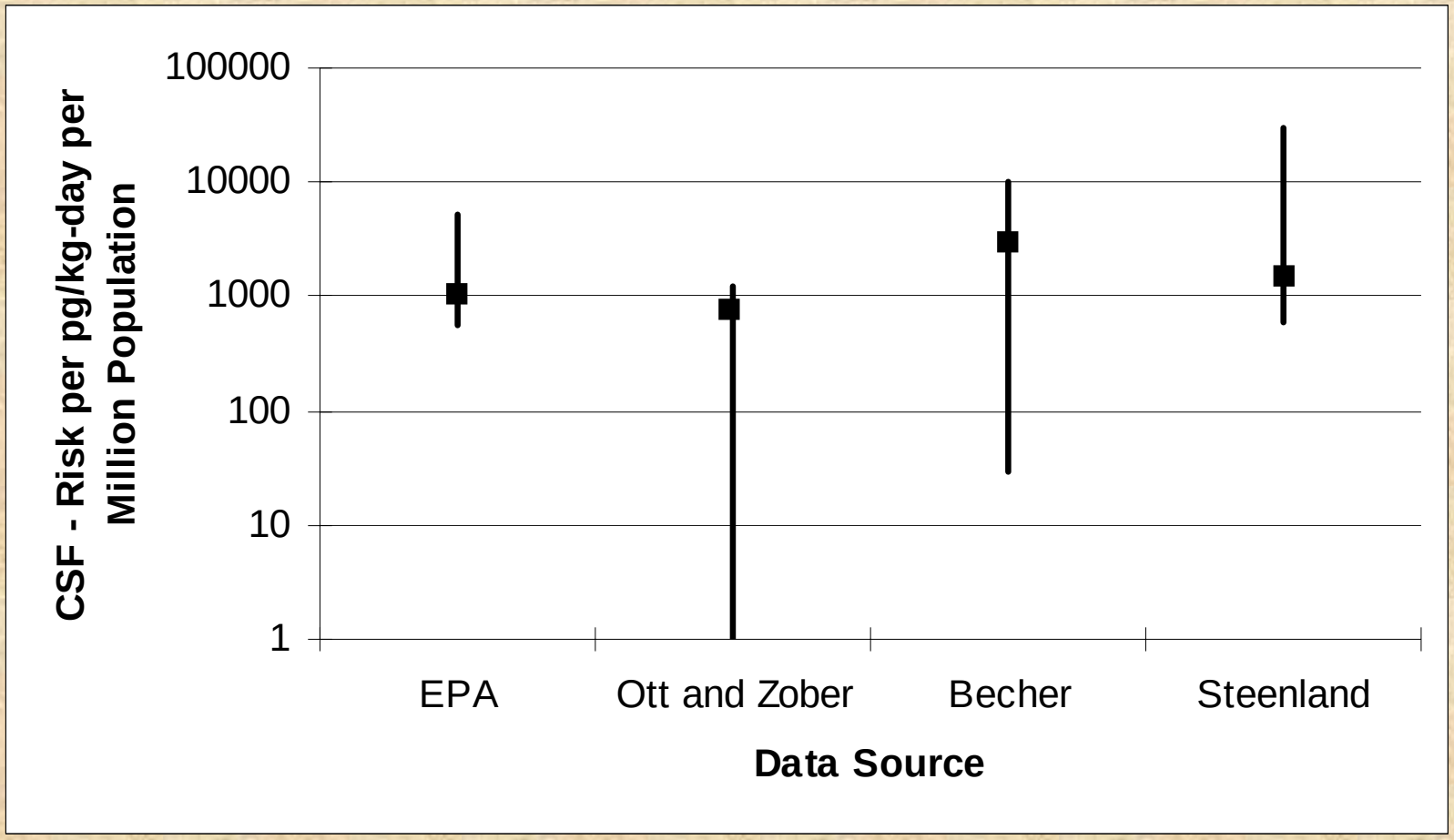
Table 5-4. Summary of all site cancer ED_{01} and slope factor calculations

Study	ED_{01} (LED_{01}) (ng/kg)	Cancer slope factor for 1 pg/kg/day above background[a] (UCL)
Hamburg cohort, Becher et al. (1998), power	6	5.1 E-3
Hamburg cohort, Becher et al. (1998), additive	18.2	1.6 E-3
Hamburg cohort, Becher et al. (1998), multiplicative	32.2	0.89 E-3
NIOSH cohort, Steenland et al. (2001), piecewise linear[b]	18.6 (11.5)	1.5 E-3 (2.5 E-3)
BASF cohort, from Ott and Zober (1996), multiplicative	50.9 (25.0)	0.57 E-3 (1.2 E-3)
Sprague-Dawley rats, Kociba et al. (1978); Goodman and Sauer (1992), pathology	31.9 (22)[f] BMD dose 38 (27.5) BMD adipose	0.97 E-3 (1.4 E-3) 0.8 E-3 (1.1 E-3)

Main concern of Committee

- Significance of ED01 vs. ED05 for POD
- Alternative, biologically plausible, dose-response functions
- Final cancer slope factor estimates from Epi studies ranged from 0.9×10^{-3} to 5.1×10^{-3} (6-fold) and compared with two estimates from rats data of 0.8×10^{-3} and 0.97×10^{-3}. (all within a factor of 10).
- Committee felt that the range of uncertainty is greater than indicated in Reassessment

NRC Report
Range of Plausible CSF Values –
Consideration of Parameter Confidence Intervals Only



Key Issue – Dose Metric

- EPA used ‘body burden’ rather than daily intake rate (pg/kg/day)
- Makes a very substantial difference (~280-fold) in cancer risk estimates from animal studies because of species differences in half-lives (~100-fold) and body fat composition, and thus the daily dose that yields a particular dioxin body burden at steady state
- Committee agreed with EPA that body burden, although not perfect, is the best dose metric to use for TCDD and DLCs, given their long half-lives and bioaccumulation in adipose tissue.

Key Issue – Use of TEF/TEQ

- Use new TEFs from WHO
- Encourage development of stronger scientific basis for individual congener TEFs, esp. those that are ‘drivers’
- Background levels of dioxins in environment are declining- are body burdens also?
- Most of the body burden is a result of a few congeners, and little or no TCDD

- EPA Reassessment used a ‘peak’ TEQ value of 55 pg/g lipid (30 – 70 CLs) as median US Background, and 5.2 +/- 1.3 pg/g lipid for TCDD -- from 1990’s

Table 93. 2,3,7,8-Tetrachlorodibenzo-*p*-dioxin (TCDD) (lipid adjusted)

Geometric mean and selected percentiles of serum concentrations (in pg/g of lipid or parts per trillion on a lipid-weight basis) for the U.S. population, National Health and Nutrition Examination Survey (1999-2000, aged 12 years and older; 2001-2002, aged 20 years and older).

LOD = 5.8 pg/g lipid

	Survey years	Geometric mean (95% conf. interval)	Selected percentiles (95% confidence interval) 50th	75th	90th	95th	Sample size
Total, age 20 and older	99-00	*	< LOD	< LOD	< LOD	< LOD	1240
	01-02	*	< LOD	< LOD	< LOD	< LOD	1228
Age group							
12-19 years	99-00	*	< LOD	< LOD	< LOD	< LOD	658
	01-02	†	†	†	†	†	†
20 years and older	99-00	*	< LOD	< LOD	< LOD	< LOD	1240
	01-02	*	< LOD	< LOD	< LOD	< LOD	1228
Gender (20 years and older)							
Males	99-00	*	< LOD	< LOD	< LOD	< LOD	572
	01-02	*	< LOD	< LOD	< LOD	< LOD	559
Females	99-00	*	< LOD	< LOD	< LOD	< LOD	668
	01-02	*	< LOD	< LOD	< LOD	6.40 (<LOD-9.10)	669
Race/ethnicity (20 years and older)							
Mexican Americans	99-00	*	< LOD	< LOD	< LOD	< LOD	336
	01-02	*	< LOD	< LOD	< LOD	< LOD	262
Non-Hispanic blacks	99-00	*	< LOD	< LOD	< LOD	< LOD	222
	01-02	*	< LOD	< LOD	< LOD	7.40 (<LOD-10.0)	217
Non-Hispanic whites	99-00	*	< LOD	< LOD	< LOD	< LOD	567
	01-02	*	< LOD	< LOD	< LOD	< LOD	665

< LOD means less than the limit of detection, which may vary for some chemicals by year and by individual sample. See Appendix A for LODs.

* Not calculated. Proportion of results below limit of detection was too high to provide a valid result.

† Data not collected for this age group for these years.

Table 87. 1,2,3,6,7,8-Hexachlorodibenzo-*p*-dioxin (HxCDD) (lipid adjusted)

Geometric mean and selected percentiles of serum concentrations (in pg/g of lipid or parts per trillion on a lipid-weight basis) for the U.S. population, National Health and Nutrition Examination Survey (1999-2000, aged 12 years and older; 2001-2002, aged 20 years and older).

LOD = 9.1 pg/g lipid

	Survey years	Geometric mean (95% conf. interval)	Selected percentiles (95% confidence interval)				Sample size
			50th	75th	90th	95th	
Total, age 20 and older	99-00	*	< LOD	36.1 (31.5-40.5)	62.8 (53.6-69.1)	75.6 (70.5-84.2)	1237
	01-02	34.6 (29.6-40.6)	39.2 (32.7-44.7)	60.7 (50.3-74.2)	95.2 (76.2-120)	127 (99.4-153)	1234
Age group							
12-19 years	99-00	*	< LOD	< LOD	< LOD	26.7 (20.2-29.6)	648
	01-02	†	†	†	†	†	†
20 years and older	99-00	*	< LOD	36.1 (31.5-40.5)	62.8 (53.6-69.1)	75.6 (70.5-84.2)	1237
	01-02	34.6 (29.6-40.6)	39.2 (32.7-44.7)	60.7 (50.3-74.2)	95.2 (76.2-120)	127 (99.4-153)	1234
Gender							
(20 years and older)							
Males	99-00	*	< LOD	34.4 (27.2-40.5)	59.2 (47.1-68.5)	73.0 (64.4-81.9)	569
	01-02	34.1 (28.3-41.1)	38.8 (31.5-44.6)	61.3 (50.0-79.5)	94.7 (70.8-131)	128 (88.5-181)	564
Females	99-00	*	< LOD	37.9 (32.5-41.6)	65.6 (55.1-70.5)	82.8 (69.3-98.9)	668
	01-02	35.1 (29.9-41.2)	40.1 (32.4-46.3)	59.8 (49.8-72.3)	97.0 (77.1-114)	126 (108-142)	670
Race/ethnicity							
(20 years and older)							
Mexican Americans	99-00	*	< LOD	24.2 (<LOD-32.4)	46.8 (38.1-56.1)	59.5 (52.7-67.9)	332
	01-02	18.3 (15.6-21.4)	21.2 (19.4-25.0)	31.9 (27.0-40.3)	51.5 (40.3-69.9)	67.9 (48.0-111)	260
Non-Hispanic blacks	99-00	*	< LOD	35.1 (28.4-44.9)	62.8 (48.0-79.2)	84.9 (72.2-98.1)	223
	01-02	38.9 (33.6-45.0)	40.2 (33.5-47.3)	63.2 (54.6-76.9)	93.9 (78.5-132)	133 (92.6-185)	219
Non-Hispanic whites	99-00	*	< LOD	38.2 (34.4-42.0)	64.4 (54.3-69.3)	77.7 (69.9-84.9)	567
	01-02	37.8 (31.5-45.4)	42.6 (33.9-51.1)	65.0 (52.3-82.2)	99.6 (78.4-130)	130 (103-165)	671

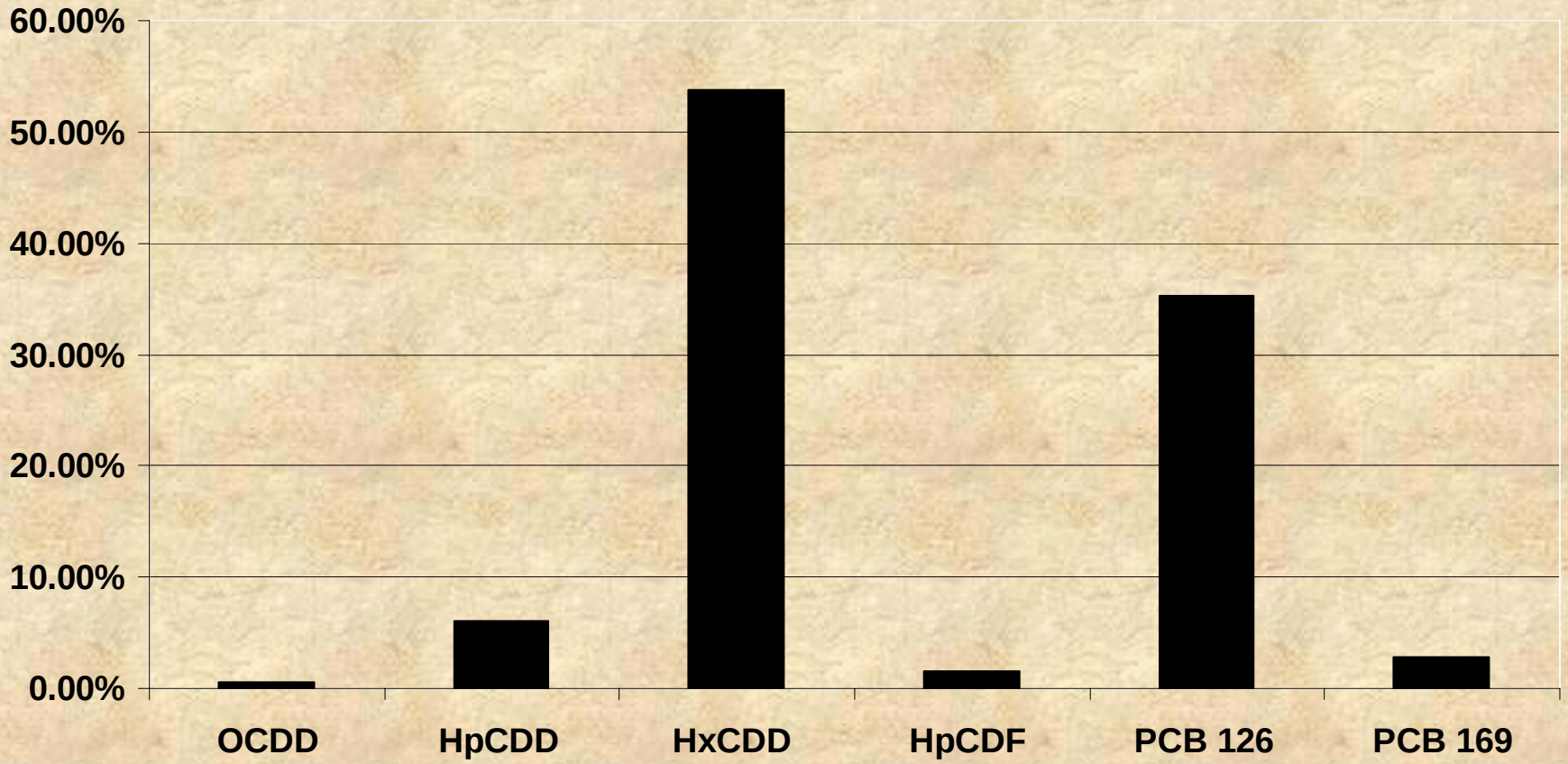
< LOD means less than the limit of detection, which may vary for some chemicals by year and by individual sample. See Appendix A for LODs.

* Not calculated. Proportion of results below limit of detection was too high to provide a valid result.

† Data not collected for this age group for these years.

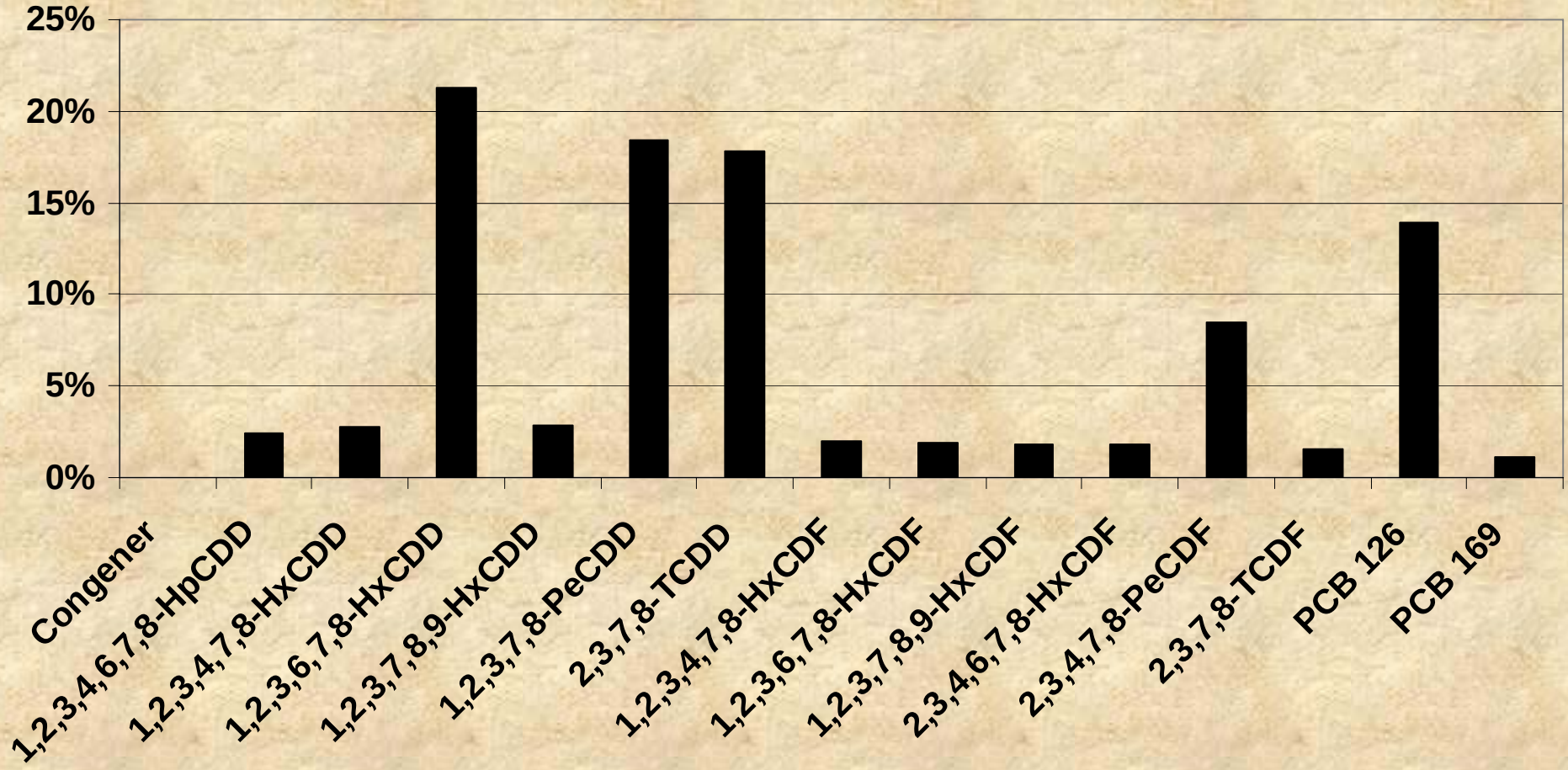
Background serum levels of Dioxin TEQs in NHANES II (US, 2001)

Assumes that non-detects are Zero
Sum of GM for all detects = 6.4 pg/g lipid



NHANES II Dioxin TEQ Data

Geometric Mean, using 0.5 x DL for non-detects
Sum of GM = 16.3 pg/gm lipid; Sum of 95% = 60.8 pg/gm lipid



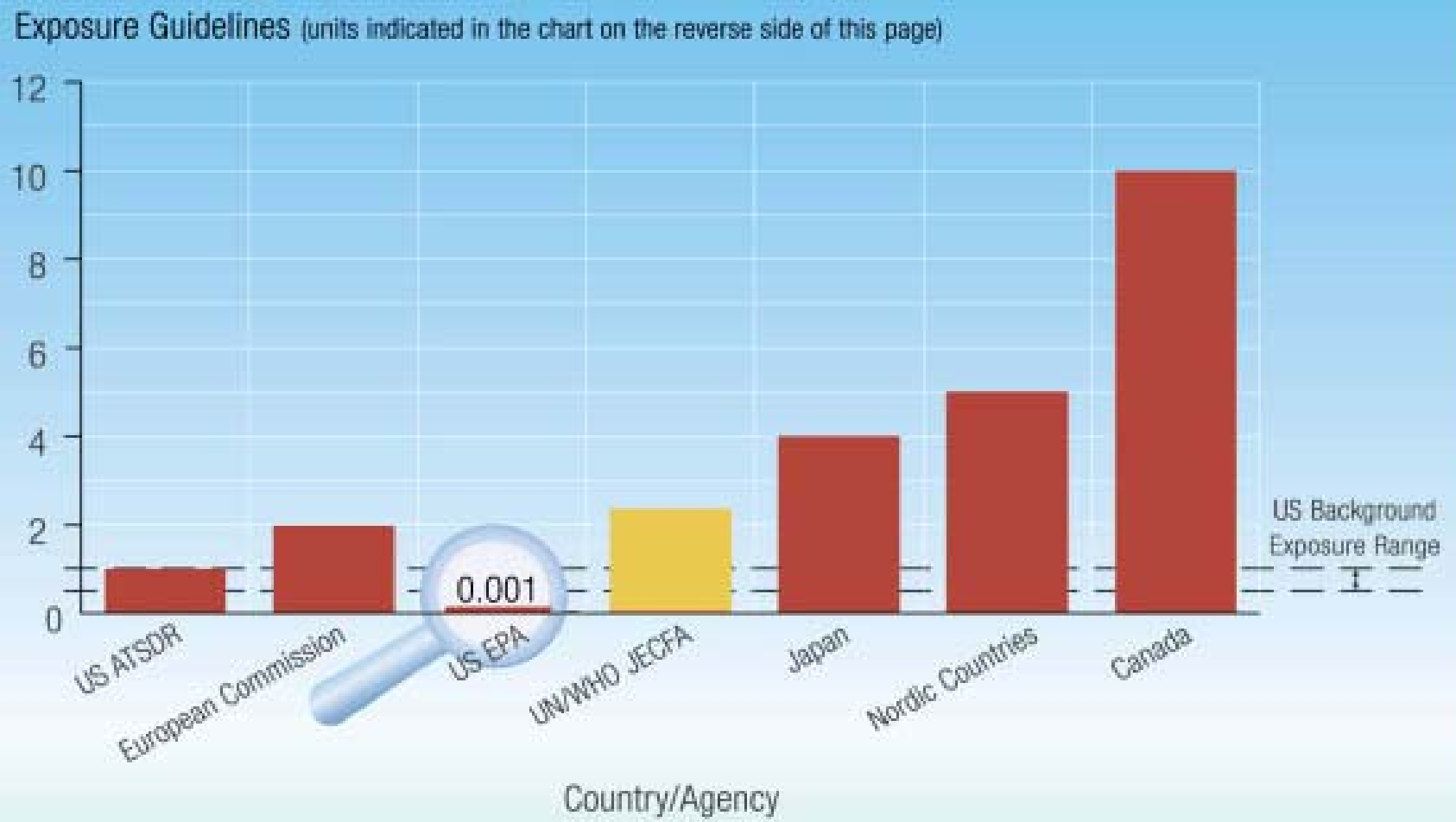
Key Issue- Calculation of RfD

- EPA chose not to calculate a Reference Dose or Margins of Exposure, primarily because their risk estimates would have resulted in low (or negative) MOEs.
- However, EPA's approach for RfD/MOE calculations use a number of uncertainty factors (such as a factor of 10 going from rodents to humans), which may not be necessary if Body Burden is used as dose metric
- WHO and many other countries utilize this approach

Bottom Line implications

- EPA's draft Reassessment arrived at a cancer slope factor of 1×10^{-3} per pg TEQ/kg/day
  - If 1 excess cancer per 100,000 was used as the 'acceptable risk' level, this would result in an 'Tolerable Daily Intake' rate of 1×10^{-5} pg TEQ/kg/d, or 0.01 pg TEQ/kg/d
  - If 1 excess cancer per 1 million was used, the TDI= 0.001 pg/kg/d
- These risk levels are based on the linear extrapolation assumption. Use of a non-linear risk estimates would use a Benchmark dose and Uncertainty factor approach

Dioxin Exposure Guidelines Set by Various Countries and Government Agencies*



*The US EPA has not set a reference dose for dioxin, but predicts that it would be 100-1,000 times lower than current background exposure levels. That theoretical reference dose is here represented as 0.001 pk/kg-body weight/day.

From Chlorine Chemistry Council

Next steps

- Although highly critical of a few key assumptions, overall the Committee endorsed much of what was done in the EPA Reassessment
- “Committee recognizes that iw will require a substantial amount of effort for EPA to incorporate all the changes recommended in the NRC report”
- “Nevertheless, the committee encourages EPA to finalize the current Reassessment quickly, efficiently, and concisely as possible after addressing the major recommendations in the report”

Midland Daily News
1/04/07

Dioxin bill signed by Granholm

Legislation allowing the state to begin recalculating the dioxin cleanup standards by incorporating the recommendations made by the National Academy of Sciences was signed into law on Dec. 31, 2006.

"This legislation calls for the best available science to better protect our health and our natural resources," Moolenaar said. "The Legislature and governor have come together to support using sound science for environmental cleanup, including the work conducted by the independent National Academy of Scientists, to lead to a more productive resolution."