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Item ID Number	05720 ☐ **Not Scanned**
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Report/Article Title	Memorandum: From Don Barnes, US EPA, to Fellow Members of the Agent Orange Working Group (AOWG) Science Panel Distribution List, regarding Trip Report on the Las Vegas "Dioxin" Meeting, dated November 13, 1987 with attachments
Journal/Book Title	
Year	1987
Month/Day	
Color	☐
Number of Images	28
Description Notes	Attachments include Trip Report, 7th International Symposium on Chlorinated Dioxins and Related Compounds, Las Vegas, Nevada, October 4-9, 1987; and Program from Dioxin '87: Seventh International Symposium on Chlorinated Dioxins and Related Compounds, University of Nevada, Las Vegas, Nevada, October 4-9, 1987

11/13/87

RE: Report on the Las Vegas "Dioxin" Meeting

TO: Fellow Members of the AOWG Science Panel Distribution List

FROM: Don Barnes, US EPA

As you may recall, I was appointed, commissioned, and otherwise ordained to be the representative of the AOWG Science Panel at the Seventh International Symposium on Chlorinated Dioxins and Related Compounds held in Las Vegas last month. As such, I recieved and duly exercised all rights and priveleges thereunto apertaining.

In order to steal -- and thereby limit -- my own thunder when I file my report at the meeting of the Science Panel on Nov. 24, I am sending you a copy of my trip report. Those of you who were at the meeting might want to examine this report as to its completeness and accuracy. Those of your who were not at the meeting might want to examine it as to its "originality, neatness and aptness of thought." In either case, I promise to limit the formal report on the 24th to less than one page.

Seriously, if there are topics/areas that any of you feel merit further atteniton, I would appreciate your input.

Famous last words overheard at the conference: "I never gamble when I come to Las Vegas. I keep all my assests in good sound stocks...and I am doing very well, thank you."

10/13/87

T R I P R E P O R T
7th International Symposium on Chlorinated Dioxins and Related Compounds
Las Vegas, NV October 4-9, 1987

Donald G. Barnes

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I. HIGHLIGHTS

A. Hazard Identification

1. Human Data
   a. No new human data were presented that would increase our concern.
   b. On the other side of the ledger, the 1986 CDC Missouri "immunotoxicity anomaly" did not hold up under closer scrutiny.
   c. The NIOSH study remains the "last, best hope" of getting something semi-definitive. It should be reported on at the meeting next year.
2. Animal Data
   a. Low level administration of OCDD for 13 weeks elicits effects that begin to look like initial stages of "dioxin" toxicity. Longer term studies need to be conducted to verify the true "dioxin" toxicity; e.g., weight loss, thymic atrophy, etc.
   b. High levels of some "non-toxic" CDD/CDF-like compounds appear to act as antagonists for "dioxin" toxicity. This may be a blessing for public health (God is good) and a curse for risk assessment (How can this be taken into account?).
   c. The Univ. of Wisc. monkey data was presented with something of a flurry, including press reports. Some effects (behavioral effects and learning deficits) were reported. However, I think there was less there than met the eye.
      On the other hand, the analysis of the reproductive effects in these animals suggests a NOEL of .05 ng/kg-d; cf. the Murray NOEL of 1 ng/kg-d.
   d. 2,3,7,8-TBrDD and 2,3,7,8-TBrDF are 100x less toxic than their chlorinated counterparts as measured in rabbit ear and LD50 studies. There are implications of these data for our mandated limit of detection for the TSCA Section 4 D/F test rule.
3. Ancillary Data
   a. No TCDD-induced DNA adducts were observed at a detection of about 1 in 10**11.
   b. More information was generated on metabolism of CDDs/CDFs.
   c. The receptor-mediated model of toxicity remains generally accepted.

B. Dose-Response Assessment

1. The 3 order of magnitude spred in "the window" describing international views of the potency of 2,3,7,8-TCDD remains.
2. Relatively little research is being directed at examining this issue. Each jurisdiction is basically maintaining its position.
3. Some additional subchronic, whole animal data have been

generated. The TEFs generally agree with these results, with some exceptions which should probably be addressed. A previous detractor of the TEF approach has generated data that supports the approach. However, vitamin A reduction does not seem to follow the TEF predictions.

4. The NATO CCMS group is proposing a common set of TEFs for everyone.

C. Exposure Assessment

The majority of papers at the conference were devoted to analytical chemistry, sources, fate and transport, and levels.

1. Analytical chemistry

a. Biological analyses (Safe, Gierthy, and Poland) are viewed as competing favorably with GC/MS in detection limits. In addition, these approaches seem to integrate the total biological response; i.e., these approaches might even be better for analysis of mixtures. Gierthy's analysis has been effective in assessing fish, following a cleanup which is less extensive than that needed for GC/MS.

The Agency should move smartly to complete the mission of developing these approaches to replace the TEF method.

b. The waxy cuticles of some leaves contain CDDs/CDFs which may reflect airborne levels of these compounds. This could be important both for monitoring purposes and for risk assessment -- if those leaves happen to be cabbage leaves, etc.

c. The detection limit of .1 ppb for the HDD/F TSCA rule is unattainable (according to some folks) due to the overlap of the BrDDs with the brominated diphenylethers in the clean up.

d. Infrared techniques can now detect and determine structure of CDDs/CDFs in the ng range.

2. Sources

a. Sources receiving more than usual attention this year included
Pulp
Paper (1986 German work found 2,3,7,8-TCDD in tissue, coffee filter, and newsprint)
Automobiles -- CDDs/Fs, BrDDs/Fs, and mixed C/BrDDs/Fs. (In all, there are more than 5000 chemicals in this family -- which is enough to make even the best analytical chemist blanch...and then get a gleam in his eye!)
Burning of straw -- which has .1% natural Cl
Home heating

b. We are likely to find other sources. However, we probably has found most of the "biggies". There is a need to put these sources into relative perspective.

c. De novo synthesis of CDDs/Fs from C, Cl and "clean"

flyash has apparently been shown.

3. Transport and Fate
   a. Evidence is being gathered that supports the notion of long range transport of CDDs/CDFs by air.
   b. Hutzinger believes that aerial deposition/collection in leafy plants is a more important exposure route to humans by direct ingestion or via grazing animals than is soil uptake by plants or sediment/water uptake by fish.
   c. Consensus is emerging on the half life of 2,3,7,8-TCDD at 5-8 years. (Yes, Dr. Poiger was there again looking fit and, theoretically, with only 750 pg of his original 1000 pg of labelled 2,3,7,8-TCDD. He says he has not acquired a taste for more.)
   d. 2,3,7,8-TCDD may be a major sink for the photodegradation of OCDD.
   e. Freeman and Schroy now agree with Yanders on the volatilization of 2,3,7,8-TCDD from soil; about 100X less than they had previously said.
   f. Vapor phase 2,3,7,8-TCDD photodegrades in solar wavelength UV.
   g. There is a lack of correlation between modeled dispersion and environmental samples of CDD/F emissions from a "dirty" MWC. This raises questions about the model or the sampling.
   h. Chloranil is apparently a source of CDDs and CDFs. Therefore, are we addressing chloranil?

4. Levels

   a. There is growing consensus that there is a background level of CDDs/CDFs in humans, which in Western societies is roughly 10 ppt on a lipid weight basis.
   b. This body burden is probably being maintained via a dietary intake of 1 pg TEQs/kg-d. Ingestion of beef is viewed by many as being the principal component in the diet
   c. High body burdens of CDDs/CDFs are markers for high exposures; in some cases, for exposure which occurred long ago.
   d. A limited review of pooled samples of human milk in Sweden suggests that levels of TEQs have dropped by 60% over the last decade. (Note that Sweden has eliminated use of chlorophenols during this time.)

D. Risk Characterization
   1. Only a few true risk characterizations were presented: human milk, pulp and paper sludge use in land reclamation, and occupational exposure limit.
   2. It is clear that more needs to be done in dealing with uncertainties. The human milk effort was notable in that it used three approaches: extrapolation from animals, comparison to human data (Yusho/Yucheng patients), and

background levels.

3. There is a real need to link up the analytical chemists and the exposure modellers in order to validate some of the remarkable predicitons which can now be made.
4. Kay Jones's analysis suggests that the difference between "worst case" homologue-specific assumption (all congeners are 2378-substituted) and the isomer-specific data is more than 10X.

E. Control Technology

I did not attend these sessions; however, it is my impression that USEPA is in the lead in these matters.

F. Risk Management

Success has been met in some instances; e.g., portions of the Missouri problem. Good risk management does not happen without good risk communication.

G. Other

Some folks are using "congener" where we use "homologue". Oh, goodness!

II. TECHNICAL PRESENTATIONS

See attached list of papers and posters.
Full abstracts are available.
Papers will be published in Chemosphere next year.

A. Plenary

1. Hazard Identification -- Steve Safe (Texas A and M)
   The 5000X spread seen in LD50 in different species is larger than that seen for other toxic endpoints.
   The AHH induction assay correlates well with subchronic responses. (Isn't it time that we began to move away from TEFs and toward this approach?)
   Good antagonists are those which have high binding affinity to the receptor, but do not turn on the responses; e.g., AHH. These include Aroclor 1254, PCBs and 1,3,6,8-TCDF.
   Antagonism also seen in immunotoxicity endpoint; i.e., sheep red blood cell (SRBC) plaque-forming assay.
2. Dose-Response -- Bob Scheuplein (FDA)
   Listed all the difficulties with modeling.
   Sielken makes some good points; cf., Food Cosmet. Tox. article. Wee need to address this substantively.
   The Moolgavkar model makes testable predictions. The LMS does not.
3. Exposure (Analytical and environmental levels) -- Christopher Rappe (Sweden)
   There are 5020 chlorinated and brominated Ds/Fs and they are being found in the environment.
   We need to look at "homologue profiles" (e.g., histograms of TCDDs vs. PeCDDs vs. HxCDDs, etc.) and "isomer patterns" (e.g., histograms of 2,3,7,8-TCDD vs. 1,3,6,8-TCDD vs. 1,2,3,4-TCDD, etc.) The patterns may be more indicative of source than are the profiles.
   The emission patterns from a number of combustion sources are essentially the same; e.g., MWC, automobiles, and PVC pyrolysis.
   Tissue levels
     Essentially the same in US, Sweden, Germany, Japan and South Vietnam
     Relatively lower in Chian and North Vietnam
     In general for CDDs: T Pe Hx Hp O
     In general for CDFs: T Pe = Hx Hp O
   Special cases
     Spanish family who ingested contaminated olive oil
     Level in the blood for OCDD is about 1 ppm!
     A girl who died at 6 days old had levels just a bit below background.
   Routes/sources of exposure
     Dermal: Low
     Inhalation: .1 pg TEQs/kg-d
     Ingestion: 1 pg TEQs/kg-d
     Nursing: 70 pg TEQs/kg-d

4. Exposure (Sources, fate and transport) -- Otto Hutzinger (FRG)

Home heating and automobiles have been underestimated as sources of CDDs/CDFs. He suggested that home heating emission would be about equal to the MWC fly ash.

84 chlorinated chemicals have been analyzed for Ds/Fs in Germany. Their analytical criteria were "under 2 ppb for 2,3,7,8-TCDD and under 5 ppb for all of the other toxic CDDs/Fs". OCDD was found in many places, including aliphatic compounds. He suggested a "Trace Chemistry of Cl" theory for predicting the presence of OCDD; i.e., wherever you have Cl, you are likely to find OCDD.

Possible sources include

a. Chloroaromatic precursors
b. Chloroaliphatic precursors
c. Pyrolysis of natural precursors; cf. lignon and Cl
d. Formation from ethylene units; cf. organics and Cl

Combustion air could be source of feed materials leading to CDD/F emissions. See ES and T article.

The amount of CDDs/Fs (mostly OCDD) in the needles of a Christmas tree is about 1 ug, or 3 kg in all of the trees in Germany. This compares to 3-4 gm TEQs/yr from automobiles.

5. Risk Characterization -- Barnes

Hard copy of slides available. (Golly, they're swell!)

6. Control Options -- Tom Hauser

Has a good one page summary of approaches. See des Rosiers.

7. Risk Management -- Boddington

Ontario has a different SF for assessing 2,3,7,8-TCDD in air compared to fish. [Note: I would express this as saying that the risk managers had decided to accept different margins of exposure (MOEs) in the two cases in reaching their "regulatory dose (RgD)".]

We do more <u>crisis</u> management than we do <u>risk</u> management. In fact, we should be doing more <u>issue</u> management, since we really don't know what the risk is. [This is a good thought which should be more fully developed and discussed.]

We all know that funding does not support low profiles projects.

B. Hazard Identification (Papers in Epidemiology, Mechanism-of-Action, Animal Toxicology, and selected posters)

1. <u>Human data</u>

a. EP01 -- "Use of Phenoxy Acid Herbicides in Sweden"
A attempt to correlate production and use figures. Only a small portion was contaminated with 2,3,7,8-TCDD.

b. EP02 -- "Risk of STS, Hodgkin's Disease and NHL Among Swedish Licensed Pesticide Applicators"

Small study of about 300 out of a cohort of 20,000. Mean follow-up time was 12 yrs. Relative risk:
For STS, 6 (obs)/6.4 (exptd) = .94
For HD, 11 (obs)/9.1 (exptd) = 1.20
For NHL, 21(obs)/20.8(exptd) = 1.01
For the latter two, there was a non-significant increase with time since license.

c. EP03 -- "NHL Among Phenoxy Acetic Acid Herbicide-Exposed Farm Workers in WA State"
Some "blips", particularly for other chemical exposures; e.g., chlordane, DDT, organic solvents and arsenic compounds. Nothing sticks out for 2,4-D and 2,4,5-T. There was adequate power to detect 1.3-fold excess risk. (Shiela Hoare commented that her study had detected 1.4-fold excess risk to those who had been exposed for more than 10 yrs.) "These results...do not implicate phenoxy acetic acid herbicides or their contaminants alone as sufficient causes for NHL..." The author noted that there could be a promoter or a co-carcinogen active here.

d. EP04 -- "NHL, STS and Exposure to Phenoxy Herbicides in New Zealand: Relationship to Frequency and Duration of Exposure"
Withdrawn

e. EP05 -- "Follow-up of Chloracne Cases Following TCDD Exposure at Seveso"
The abstract states: "...shows the absence of effects other than chloracne and its whole reversibility in the Seveso population."

f. EP06 -- "... Seveso...and Ten Year Mortality Experience"
The abstract states: "...suggestions of an increased risk of cardiovascular diseases shortly after the accident and of a later possibly increased mortality from liver cancer in selected subgroups were obtained."

g. EP07 -- "Spontaneous Abortion and Exposure to Emissions from Waste Electrical Products Combustion"
I didn't hear this paper from Taiwan, but the abstract states that "...former study shoes that pregnant women in polluted area have higher prevalence rate of spontaneous abortion." The abstract promises more info in the presentation.

h. EP08 -- "Medical Follow-up of the Health Effects of Long-Term Exposure to 2,3,7,8-TCDD"
Failure to confirm the original (flawed?) study which reported immuntox effects in exposed folks in Missouri. Includes a somewhat generous discussion of what might have gone wrong and suggests additional study. (Heaven help us!)

i. PS12 -- "Case-Control Study of Women Giving Birth to Deformed Babies and on Patients Having Hydatiform Mole in the Obstetrical and Gynecologic Hospital of Ho Chi Minh city in South of Vietnam"

j. PS 13 -- "Retrospective Study on the Incidence of Birth

Defects and Other Reproductive Anomalies in the Obstetrical and Gynecological Hospital of Ho Chi Minh City"

k. PS23 -- "Surveys on Incidence of Reproductive Anomalies in Herbicide-Sprayed and Non-Herbicide-Sprayed Villages in the South of Vietnam"

l. PS28 -- "Lab Assessment of Immune System in Individuals Occupationally Exposed to 2,3,7,8-TCDD: Test Selection, Assay Methods, and QC in a Cross-Sectional Epi Study"

Preliminary work to determine the variation in the endpoints. It is going to be hard to interpret, given the intra-individual variations in these endpoints.

m. MA06 -- "Humans Exposed to PCBs and PCDFs Exhibit Increase SCE Frequencies in Lymphocytes When Incubated with a-Naphthoflavone (ANF)"

The presence of ANF increases the frequency of observed SCE in a blood cultures. True for smokers vs non-smokers. True for Yucheng patients in which the blood concentrations were 15 ppb for PCBs and 14 ppt for PCDFs. It is thought that the pollutants result in greater metabolism of ANF to genotoxic metabolites. Note TCDD receptor concentrations: rat lymphocyte -- 2 fmol/mg; human lymphocyte -- .3 fmol/mg. ANF assay also active with Chinese hamster ovary cells and TCDD induced liver microsomes. ANF-DNA adduct detected (but see lack of adducts noted by Safe). Phenobarbitol is not effective, even with ANF. P-450c is implicated.

n. MA15 -- "Placental Markers of Human Exposure to PCBs and PCDFs"

Placentas obtained 4 years after exposure in Taiwan. Marked AHH activity (10-100X). Significantly lower EGF stimulated receptor autophosphorylation, correlating with decreased birthweight. However, no alteration in EGF receptor binding kinetics. Only very low concentration of specific binding sites. This suggests "...that the potent AHH induction can occur in humans in the absence of high concentrations of the Ah receptor" (cf., similar receptor concentrations in species of differing susceptibility). Marked elevation of some PCBs and PCDFs in the placental tissue; e.g., 2,3,4,7,8-PeCDF (120 ppt) 1,2,3,4,7,8-HxCDF (380 ppt), and some PCB isomers at a ppb. "Total PCB values might be better predictors of response than PCDF values following human exposure..."

2. <u>Animal data</u>

a. ATO1 -- "Interleukin 1 Responsiveness and Production in 2,3,7,8-TCDF-Treated Mice"

Response of thymocytes to IL-1 used as a probe for 2,3,7,8-TCDF treated mice. Perinatally exposed

animals compared to postnatally exposed. Some effects seen.

b. AT02 -- "Behavioral Effects in Monkeys Exposed to 2,3,7,8-TCDD Transmitted Maternally during Gestation and for Four Months of Nursing"

This is a follow-up of the Allen work. "There were no observed physiological indications of toxicity in either the mother monkey or their offspring..." Fat data are reported: e.g., offspring of the 5 ppt moms had about 350 ppt, while those from 25 ppt moms had 370, 760 and 1350 ppt.

Certain social behavioral differences are noted; e.g., "...increased maternal care of the [treated] offspring..." A series of "learning data" were present allegedly showing some sort of D-R relationship. This is strange stuff, however; e.g., for some effects the trend is up with dose, for other effects the trend is down with dose. In nearly every case, the responses of the treated animals <u>are within the range of the controls</u>! During the Qs and As, the author (Bowman) characterized the learning data as "Not strong, subtle".

For me, this is some interesting, preliminary data for "... a relatively new area of research in which the methodology is still at an early stage of development". It certainly doesn't seem to be the type of thing that would support a story in a San Francisco newspaper.

c. AT03 -- "Chronic Dietary Intake of 2,3,7,8-TCDD...: Kinetics and Dose-Effect Estimate of Reproductive Toxicity"

The abstract indicates that a lot of tissue (fat/milk from mom/kids) have been gathered. Mom's fat conc of 2,3,7,8-TCDD is less than that of the kid. However, the moms' levels are proportional to the kids'. There is a 20-30% transfer of mom's body burden to the kid via nursing. Begin to see toxic effects in animals having a fat level of 200 ppt. Apparent half-life is a bit over a year. An index of reproductive success was developed. (Comment, anyone?) Extrapolation suggests a NOEL of about .05 ng/kg-d. (This is 20X lower than the Murray NOEL of 1 ng/kg-d or the level used by CDC, derived from these same monkeys at an earlier date.)

d. AT04 -- "Increase in Epidermal Langerhans Cells in Mouse Skin Following Treatment with TCDD"

Langerhans cells are strange little fellas, being immunologic cells of skin. Contrary to expectation, these cells increased 3-4X following treatment of the skin with 2,3,7,8-TCDD. In vitro, both the haired and the hairless mice responded to the chloracnegen. In vivo, however, only the hairless responded. Note that phorbol esters also stimulate Langerhans cell proliferation in hairless mice.

e. AT05 -- "Metabolism of Some PCDFs in the Rat"
Chemicals studied: 1,3,7,8-TCDF
2,3,6,8-TCDF
1,2,3,4,8-PeCDF
1,2,3,7,8-PeCDF
2,3,4,7,8-PeCDF
1,2,3,6,7,8-HxCDF
1,2,3,4,6,7,8-HpCDF

Breaking the ether linkage is tough. Metabolism of the persistent 2,3,4,7,8-PeCDF is complex. The Hx- and Hp- are not metabolized very much.

f. AT06 -- "Subchronic Toxicity of some PCDDs/PCDFs in the Rat"

Compounds studied:		TEFs:
	2,3,7,8-TCDD	1.0
	1,2,3,7,8-PeCDD	.4
	1,2,3,4,8-PeCDF	.001
	1,2,3,7,8-PeCDF	.01
	2,3,4,7,8-PeCDF	.4
	1,2,3,6,7,8-HxCDF	.1

13 week studies. A variety of endpoints examined. Differences were noted in the manner in which the compounds distributed in the body.

Tests with mixtures showed that additivity holds. (Safe predicts that antagonism would be seen if non-toxic congeners were also used.)

g. AT07 -- "Relay Toxicity Study with a Mixture of PCDDs and PCDFs"

This was presented as a poster session, which I did not see. However, the abstract talks about feeding flyash to rabbits, excising the liver, extracting the CDDs/CDFs from the liver, analyzing by GC/MS, and feeding the extract to rats. The toxicity predicted by their TEFs were in good agreement with the results of the rat study.

This marks a change in the Suter-Hoffman stance in that she now finds that the TEF approach (using factors somewhat different from EPA's) works rather well, thank you. The toxic effects of the CDDs/CDFs in mixtures appear to be additive.

h. AT08 -- "Effects of Combinations of PCDDs/PCDFs Given to Rats"

Vitamin A reduction does not seem to follow the TEF predictions very well.

i. AT09 -- "Is the TCDD-Induced Embryotoxicity in Rats Due to Maternal Toxicity?"

Yes, and principally.

j. AT10 -- "2,3,7,8-TCDD Induced Alterations in Vitamin A Homeostasis and in the EROD Activity in Rats and Guinea Pigs"

Lots of data, including the fact that rats can recover when guinea pigs can't.

k. AT11 -- "Distribution of 2,3,7,8-TCDD between Parenchymal and Non-Parenchymal Rat Hepatic Cells and Its Effect on the Vitamin A Content of These Cells"

"...long-lasting inhibition of the normal vitamin A accumulation occuring in the stellate cells..."

l. AT12 -- "Immune Abnormalities Associated with Chronic TCDD Exposure in Rhesus"
"...these immunological alterations are not global, nor are they of such severity that an immunodeficient state of major clinical significant is apparent...[T]here have been no clinical manifestations consistent with major loss of a host defense system...[T]he observed immunological alterations occurred quite consistently in the mother carrying the higher levels of TCDD in fat for years after the exposure."

m. PS44 -- "Two-dimensional Polyacrylamide Gel Electrophoresis of Liver Microsomal Proteins from Rats Treated with 2,3,7,8-TCDD, 3-Methylcholanthrene, and Phenobarbital"
Characteristic patterns developed.

n. PS69 -- "Response of Murine Skin to 2,3,7,8-TCDD: Comparison of Haired and Hairless Genotypes"
Response of newborn mouse skin to TCDD was similar to that of adults. Also looked at some skin enzyme and added treatment with other chemicals.

o. SE16 -- "Investigation of Comparative Toxicity of [Flame Retardant Treated Plastics] Using 2,3,7,8-TBrDD and 2,3,7,8-TBrCF as Positive Controls"
By the LD50 test and by the rabbit ear test, the TEFs for the brominated analogs are .01 or less. There is some question about portions of the experiment, but apparently BrDDs are formed a non-detectable levels. However, the BrDFs have a pretty good yield; e.g., .1%.

3. Ancillary Data (Mechanism-of-Action and selected posters, including information on physical properties)

a. MA01 -- "Evidence for the Formation of Chlorinated Biphenyls, Phenoxyphenols and "Dioxins" Upon Metabolism of 2,4,5-TCP in Rat Liver S9-Fractions"
Apparently, a hydoxylated HxCDD has been detected in this in vitro experiment which is not observed in the absence of the S9 fraction. Previously, this biochemistry had only been observed in a fungus.

b. MA02 -- "A Physiologically Based Pharmacokinetic Model for 2,3,7,8-TCDD"
The model was developed using data from the C57B6 vs the DBA vs rat. It estimates tissue distributions in terms of organs volumes, blood flow rates, etc. It postulates two groups of liver binding proteins; cf. cytosolic receptor and the microsomal monooxygenase system.

c. MA03 -- "Elimination and Pharmacokinetic Interaction of some PCDFs in the Liver the Rat"
Half lives: 1,2,3,7,8-PeCDF 3.3 days
2,3,4,7,8-PeCDF 108 days; longer than

2,3,7,8-TCDD
1,2,3,6,7,8-HxCDF 61 days; " " " "
First order kinetics. No interaction in binary mix.

d. MA04 -- "Selective Retention of PCDDs and PCDFs in Mammals: A Multiple Cause Problem"
Posits that more than differential metabolism is involved. Suggests "...primary involvement of carrier-protein(s)...". Similar thoughts as MA02.

e. MA05 -- "Liver DNA Alterations by 2,3,7,8-TCDD and 1,2,3,7,8-PeCDD in Female Rat"
This was an investigation of "I-spots", liver alterations which have been suggested as sites of spontaneous initiation. Both the TCDD and PeCDD dosing led to a decrease in the concentration of these I-spots. Hmmm.

f. MA07 -- "Effects of 2,3,7,8-TCDD on Enzyme-Altered Foci, Hepatocellular Tumors, and Estradiol Metabolism in a Two-Stage Hepatocarcinogenesis Model"
Ovarectomized rats did not yield the Pitot-like promotion of DEN-initiated rats. Studies with whole rats indicated that DEN potentiates estradiol metabolic effects of 2,3,7,8-TCDD. There is some confusion about what this means regarding the promotion behavior of the compound.

g. MA08 -- "Potentiation of the Toxic Action of 2,3,7,8-TCDD by Some PCBs, as Assessed by E.I.S-bioassy"
Extension of bioassay using fish to examine TEFs. Several unanswered questions about the study remain.

h. MA09 -- " Inhibitory Effect of Methylsulphonyl PCBs on AHH Activity"
These chemicals are human metabolites of PCBs found in Yusho patients. Surprisingly, they reduced AHH activity.

i. MA10 -- "Aroclor 1254 as a 2,3,7,8-TCDD Antagonist in Mice"
Safe's report of his work. Effects noted in immuno-assay. Also development of cleft palate.

j. MA11 -- "Effect of TCDD and Comparable Inducers Upon Synthesis and Mono-oxygenase Molecular Forms P-450c and P-450d Among Mammals"
The first Russian contribution at these conferences. TCDD microsomes have a low content of P-450c but have much higher benzpyrene-hydroxylase activity than the enzymes induced by other "MC-type" inducers.

k. MA12 -- "Dynamic Comparision of Rat Liver Benzpyrene-Hydroxylase Induction by 2,3,7,8-TCDD and 3-MC"
A kinetic investigation of the previous paper.

l. MA13 -- "Kinetic Models for Association of 2,3,7,8-TCDD with the Ah Receptor"
Usual determinations of dissociation constant of the receptor complex assume equilibrium. Due to the rapid dissociation of the unbound receptor, this

assumption is likely to be incorrect. An alternative method of determining the dissociation constant leads to values for Kd which are two orders of magnitude less than previous values; i.e., the complex is "tighter".

m. MA14 -- "TCDD Mediated Decrease in Dexamethasone Binding to the Hepatic Glucocorticoid Receptor is Not Accompanied by a Decrease in Immunodetectable Glucocorticoid Receptor Concentrations: The Role of Adrenal Steroids"

Adrenalectomized rats are more sensitive; therefore, "...circulating adrenal hormones may exert a protective role in modifying the animal's response to TCDD..." TCDD reduced the number of glucocorticoid receptor binding sites, but did not affect the concentration of the protein itself. The role of the Ah receptor is being investigated in "susceptible" and "unsusceptible" mice. The hypothesis is that this effect is separate from the Ah receptor mechanism.

n. MA16 -- "Comparative Effects of 2,3,7,8-TCDD and Progesterone on Estrogenic Responses in Rats"

Both lead to decrease in estrogen receptor. 2,3,7,8-TCDD also reduces the level of progesterone receptor. The pathway of 2,3,7,8-TCDD activity is different from that of progesterone.

o. PS08 -- "Prediction of Vapor Pressures, Boiling Points and Enthalpies of Fusion for 29 HDDs and 53 HDFs by Vapor Pressure Correlation Method"

Data from 14 CDDs/CDFs led to predictions for a number of other Cl-, Br-, I-, F- and mixed DDs and 53 CDFs.

p. PS41 -- "Solubility Studies Using a Generator Column for 2,3,7,8-TCDD"

Solubility of 13 ppt determined at 4 degrees C. Solubility at 25 degrees was much higher (over 100 ppt?) These values differ from the 1986 value of Marple, which EPA is citing these days. This wide variation in the literature cautions against our using any value as a "real number".

q. PS48 -- "Molecular Geometry Approximations for CDDs by Fourier Transform IR Spectroscopy"

Vapor spectra of 14 different CDDs. Calculated COC bond angles to determine geometry. Ranged from nearly planar for lateral substitution with high electron withdrawing groups to tetrahedral for low electron withdrawing groups. Attempts to correlate with toxicity; cf., polarizability (Safe's earlier work), geometry, and steric interactions; cf. PS51.

r. PS 51 -- "Calculated Molecular Geometrics and Energies of All CDDs Using MMP2"

A "non-quanatum mechanical technique", involving "forcefield calculatiohs", leads to estimates of geometry. The COC bond angles are determined from IR and correlated with toxicity; cf. PS48.

s. PS55 -- "Thermal Decomposition Curves of HDDs by Scanned Flow Tube Pyrolysis Method with On-line Ion Trap MS Detection"

t. PS59 -- "An Evaluation of the Molecular Structure of OCDF" (and HpBrDF)

Planar. Low energy UV preferentially removes Cl at the 9 position. Higher energy UV is less selective. Apparently OBrDF cannot be made due to steric hindrance; you get decabromodiphenyloxide instead. Further, only one HpBrDF forms (9 position vacant), while four HpCDFs form.

u. PS68 -- "Receptor Binding Characteristics of 2378-CDF Radioligands"

Kds: 2,3,7,8-TCDF = 1.3 nM
1,2,3,7,8-PeCDF= 5.9 nM
1,2,3,6,7,8-HxCDF= 2.3 nm

These values did not correlate with competitive displacement of tritiated 2,3,7,8-TCDD of in vitro AHH induction. Apparently the oral presentation discussed the implication of these results.

C. Dose-Response Assessment (Dose-Response, some Risk Characterization, and selected posters dealing with TEFs)

1. Direct Potency
   a. DR01 -- "Reexamination of the D-R Relationship for Induction of the Hepatic Monooxygenase System by 2,3,7,8-TCDD"
      Looked at enzyme induction at near lethal doses. An unanticipated "super induction" is seen.
   b. RC08 -- "Cancer Risk Characterization of HxCDD"
      The CAG potency calculation. Reference to a Moolgavkar model result that is two orders of magnitude lower.
   c. PS42 -- "Quantitative Model for the Tumor Promoting Activity of 2,3,7,8-TCDD"
      The application of Moolgavkar to 2,3,7,8-TCDD gives results that are two orders of magnitude lower than the UCL of the LMS. "More than half of this difference can be attributed to the differences between the higher background female rat liver tumor and lower human liver cancer rates. The second major factor is the difference between the MLE of the M-V-K model vs the UCL used with the LMS."

2. TEF related data
   a. DR02 -- "Effect of Pharmacodynamic Considerations on TEFs for CDDs and CDFs"
      Might want to consider changing values for 2,3,4,7,8-PeCDF, 1,3,4,7,8-PeCDF, and OCDD.
   b. DR03 -- "Applications of the In Vtiro AHH Induction Bioassay for Determining TEQs: Pyrolyzed Flame Retardant Mixtures"
      Good correlation between in vitro AHH induction and in vivo AHH and EROD induction, body weight loss and thymic atrophy.
   c. DR04 -- "Subchronic Effect of Exposure to OCDD"
      Linear absorption of small doses over time. Final levels: liver, 222 ppt; adipose, 165 ppt; skin 160 ppt; and blood 70 ppt. (Cf. Rappe's Spaniard with 900,000 ppt OCDD in the blood.) Increased liver/body wt, but no change in thymus. EROD and P-450 show D-R. Analyzed both the chemical and the liver for all CDDs/CDFs. Non-OCDD contaminant levels cannot account for observations. TEF = .009. Commenters noted that "classical dioxin-like activity" (e.g., thymus and body wt decrease) have not yet been seen. Response: The response seen here is like that seen at low doses of 2,3,7,8-TCDD. More work is underway.
   d. PS11 -- "TEFs for 2378-CDDs/CDFs by the E.L.S. Bioassay"
      This is a fish fry assay, which has not yet been generally accepted. However, the TEFs are as follows:
      2,3,4,7,8-PeCDF = .4
      1,2,3,7,8-PeCDF = .04

1,2,3,6,7,8-HxCDF = .04

e. PS43 -- "Validation of the AHH Induction Bioassay for the Determination of TEQs"

Safe's work for CDDs, CDFs, and PCBs. Demonstrated log-log linearity between in vitro AHH induction and in vivo AHH induction, body wt loss and thymus atrophy.

F. PS45 -- "Relative Toxicity of CDDs and CDFs Measured by Thymus Weight and Liver Enzyme Induction in Perinatally Dose Rats: 2,3,7,8-TCDD, 2,3,4,7,8-PeCDF, and 1,2,3,7,8-PeCDD"

TEFs 1,2,3,7,8-PeCDD = .16
2,3,4,7,8-PeCDF = .05 (Ed. note: Strange)

In utero exposure is about equal to exposure via milk.

D. Exposure

1. Analytical

These are found in the 32 papers of the Analytical Methods section and several of the Poster papers. There were too many to discuss here. Some were somewhat arcane, but generally lead to lower detection limits in various media. Others were related to new methods, both instrumental and biologically based.

2. Sources (MWCs) (Source Emissions, Levels/Trends, and selected Posters)

a. SE09 -- "On the De Novo Synthesis of PCDD/PCDF on Fly Ash of MWCs"

Baking "clean" fly ash, carbon and inorganic Cl at 300 degrees C for two hours apparently can give rise to CDDs/CDFs in the ppb range; hence, "de novo" synthesis. The presence of a little copper as a catalyst is helpful.

b. SE10 -- "Joint German/Swedish Program on Testing Sampling Equipment for MWC Incinerators"

c. SE11 -- "Chemistry for Formation and Fate of PCDD/PCDF in MWC Process"

Only minor amounts of CDDs/CDFs in the fire box (650 - 700 degrees C). CDDs/CDFs formed on the way to the heat exchanger (250 - 350 degrees), as well as Cl-phenols formed. CDDs formed on fly ash. Amount of CDDs/CDFs varies 10x depending on operating conditions.

d. SE12 -- "Air Emission Characterization of a Two Stage MWC"

Results of testing at one NY facility.

e. LT05 -- "CDDs/CDFs in Fly Ash from MWC"

Tests the hypothesis that (effectively) total TEQs can be estimated from values for TCDD. Finds that "...fly ash does not have a very useful characteristic profile (fingerprint)."

f. LT06 -- "Complete Analysis of Organic Compounds in MWC Flyash using HPLC and GC/MS"

Levels of CDDs/Fs from Machida incinerator (Japan) are significantly lower than most other MWCs.

Insert a barrier in order to generate more turbulence.

g. PS50 -- "Determination of PBrDDs and PBrDFs and Mixed Br/Cl Ds/Fs in Environmental Samples"

Data have been obtained from MWCs and automobiles.

h. PS54 -- "Theoretical and Experimental Analysis of the Post-combustion Region of an Incinerator"

i. PS61 -- "Evidence for Post-Furnace Formation of PCDDs and PCDFs: Implications for Control"

Review of studies in the literature

3. Sources (Other Than MWCs)

a. SE01 -- "Emissions of PCDDs and PCDFs from the Swedish

Pulp and Paper Industry"
Found it in the emissions from combustion of "black liquor". (EPA's Tier 4 did not find any here.) Found it in wastewater in low ppqd. The suspended solids had it in the ppt range.

b. SE02 -- "Occurrence and Fate of CDDs and CDFs in Bleached Draft Papermaking Processes"
EPA's tale.

c. SE03 -- "CDDs and CDFs in Effluents and Sludges from Pulp and Paper Mills"
Comparable to EPA data.

d. SE04 -- "Emissions of CDDs and CDFs in Gasoline and Diesel Fueled Cars"
Actually looked for and found Cl, Br and mixed Cl/Br dioxins and furans. Estimated total in Sweden is 3-7 g/yr, which is about 10% of the amount generated by MWCs in that country. Most the TEQs are from TCDF.

e. SE05 -- "Levels of Bioactive CDFs in Used and Unused PCB Dielectric Fluids"
Concludes that "...normal usage of capacitors and transformers does not result in the conversation (sic!) of PCBs to PCDFs."

f. SE06 -- "Environmental Behavior of Monomehtyl-Substituted Polychlorinated Diphenylmethanes (Me-PCDMs) in Comparison with PCBs"
These Me-PCDMs are PCBs substitutes in the electrical and hydraulic situations. It appears that they have similar problems, however. For example, pyrolysis yields up to ppthousand levels of CDFs, and fish bioaccumulation is likely to be similar to PCBs.

g. SE07 -- "Search for Industrial Sources PCDDs/PCDFs: IV. Phthalocyanine Dyes"
CDDs/CDFs in the ppt/ppb range. In general, "...chlorobenzene with catalytic amounts of CuCl2 or the chlorophenols which are present in most commercial chlorobenzenes cause the PCDD/PCDF formation. Some experiments were made for prevention of PCDD/PCDF formation."

h. SE08 -- "Pyrolysis of Dibenzodioxin, Dibenzofuran and 1,2,3,4-TBrDD with Different Chlorine-donors and Catalyts"

i. SE13 -- "Ash Data from Combustion Sources"
EPA's Tier 4 results. Flyash levels of CDDs/CDFs are a qualitative, not quantitative, indicator of CDD/CDF stack emissions.

j. SE14 -- "Production of CDDs and CDFs During the Thermal Treatment of Soils Contaminated with Hexachlorohexane"
CDDs are ND around 750 degrees C; CDFs ND about 1100.

k. SE15 -- "PBrDD and PBrCDF from Brominated Flame Retardants"
The German study. Higher Br compounds photodegrade. A .1 ppb detection limit is not realistic since the brominated diphenyl ethers cannot be separated from

the BrDDs, due to the lower solubility of BrDDs compared to CDDs. This makes EPA's detection limit of .1 ppb seem unrealistic.

l. SE16 -- The American Study of the BrDDs/Fs from retardants.

m. PS09 -- "Tetrachlorobenzoquinones as Source of PCDD/PCDF"
O-Chloranil and p-chloranil have levels of CDDs/CDFs in the ppb range, up to ppm for OCDD/F.

n. PS27 -- "CDD/CDF and Chlorinated Phenols in Wood Preservation Formulations for Household Use"
Levels are reported in PCP paints produced in the 1965-85 timeframe in Germany. In addition, levels in dust from homes are also reported.

o. PS56 -- "CDDs and CDFs in the Environment as a Result of Outdoor Chemical Waste Burning"
Uncontrolled burning of wastes, including 2,4,5-T still bottoms.

p. PS57 -- "Emissions of PCDDs and PCDFs from the PVC Fire in Holmsund, Sweden. A Case Report"
A January, 1987 plastic carpet company fire was investigated. "Surprisingly low levels of dioxins were detected" inside the building. Levels were found in the snow downwind.

q. PS58 -- "Monitoring New Source of PCDD and PCDF: Automotive and Straw Firing"
Automotive data are compared with MWC. Straw is regularly burned in Denmark to clear fields and for residential and community heating. Straw contains .1% Cl. Home firing exceeds Swedish MWC standard of .1 ng TEQ/m3. Community firing is at .1 ng/m3 level.

4. <u>Transport and Fate</u> (Transport and Fate, Levels and Trends, and selected posters)

a. TF01 -- "Geometrical Description of the TCDD Contamination in Times Beach"
Used the Univ of Pavia method for depicting the spatial distribution of the surface contamination.

b. TF02 -- "Bioavailability of Grain and Soil Borne Tritiated 2,3,7,8-TCDD Administered to Lactating Holstein Cows"
Bioavailable equally from crushed grain and "aged" soil. 50% is excreted in the feces, 40% stays in the fat. 15% comes out in the milk, mostly within 4 days. No receptor found in cows. No adverse effects are seen in 14 days.

c. TF03 -- "PCDD and PCDF Bioaccumulation by Lake Ontario Fish: Sediment to Fish Residue Relationships"
For slimy sculpins, the ratio ranged from 1:1 to 1:.1. More data were presented and compared with predictions.

d. TF04 -- "Bioavailability of CDDs from Contaminated Soils"

	TCDD	HpCDD	OCDD

Newark:	1.6%	6.4%	2.6%
Times Beach:	30%	8.7%	3.6%

e. TF05 -- "Sorption of Dioxins to Soils"
Log Koc, normalized on fraction of organic content in the soil, for some 2378-CDDs. There were more hydrophobic than predicted.

	Log Koc
2,3,7,8-TCDD	6.6
1,2,3,4,7,8-PeCDD	5.1
1,2,3,4,7,8-HxCDD	7.5
1,2,3,4,6,7,8-HpCDD	6.9
OCDD	7.9

f. TF06 -- "Photolysis of OCDD on Soils: Production of 2,3,7,8-TCDD"
In solution, the lateral Cls seem most likely to be removed. On soil, the reverse is true and 2,3,7,8-TCDD is a sink. [This is similar to what the Wizards observed some time ago.]

g. TF07 -- "Measurement of the Photoinduced Loss of Vapor Phase TCDD by MS"
Lab study suggests a half life of 11 min for 355 nm high pressure light. Quantum yield of .02. [Use this with the characteristics of the solar spectrum to predict environmental half life.]

h. TF08 -- "Long-Range Transport of PCDD, PCDF, Cl-PAH and PCP on Airborne Particles"
Slight variation in congener (sic -- we would say "homologues") profiles. Isomeric patterns within a homologue are similar and are characteristic of auto and MWC emissions.

i. TF09 -- "Studies of Physico-Chemical Parameters Affecting Translocation of PCDDs in Soil"
Mobility highest in the presence of organic solvents, but not directly related to solubility. Examines effects of surface area, pore size distribution, organic matter content, type of soil organics and the potential for micelle formation.

j. TF10 -- "Field and Lab Studies on the Movement and Fate of TCDDs in Soil"
Simulates the conditions at Times Beach. "The results obtained indicate that both the migration and loss of TCDD from soil through such processes as volatilization and subsequent photolytic degradations or through direct surface photolysis of soil-bound TCDD are substantially lower than the values previously reported in the literature."

k. TF11 -- "Comparison of the Rate of TCDD Transport at Times Beach and at Eglin AFB"
Freeman and Schroy revise approach to fit Yanders' data.

l. TF12 and PS02-- "Estimating Exposure to 2,3,7,8-TCDD"
EPA's "Fat Document"

m. LT15 -- "Human Exposure to 2,3,7,8-TCDD"
ORNL global modelling of 2,3,7,8-TCDD, leading to an

estimated intake of about 1 pg/kg-d, 98% of which comes from food, especially meat and dairy products, not contaminated fish and shellfish.

5. Environmental Levels (Levels and Trends and selected posters)

a. LT01 -- "PCDD/PCDF in Dutch Inland Waters"
Investigation of waste water discharges, sediments and fish. Sediments had differing profiles. "..[i]n only a few cases could (the profiles) be related to the sources."

b. LT02 -- "CDDs/CDFs in Selected Estuarine Sediments"
Samples from New Bedford Harbor, Bridgeport and upper Narragansett Bay. PCDFs decreased with distance less rapidly in NBH than did the PCBs. [Additional sources? Differential decomposition?]

c. LT03 -- Plant Surfaces: A Sampling System for Atmospheric PCDDs and PCDFs
"Since uptake PCDDs and PCDFs via roots can be excluded..." [Basis?] Hutzinger's work on pine needles: ppt levels of all CDDs/Fs. Profiles typical of combustion sources. [How does this correlate with Crosby's finding of short half life on leaves?]

d. LT04 -- "Surface Contamination of PCDD and PCDFs in PCB Disposal Facility"
Three facilities examined for air and surface levels of PCBs, employee blood levels for PCBs, and surface levels of CDDs/CDFs.

e. LT07 -- "PCDDs and PCDFs in the Dust of the Working Atmosphere of a MWC"
The dust is "quite different from the flyash samples usually collected in incinerators." All of the CDDs/CDFs are present. 2,3,7,8-TCDD was found a about .9 ppb and 6 pg/m3.

f. LT08 -- "Ambient Air and Incinerator Testing for CDDs/CDFs by LR-MS"
Hites showed 1 pg/m3 per congener group or lower in ambient air around Bloomington, IN. This study also shows about 1-2 pg/m3 for the most abundant congener groups. "TCDFs predominate in some samples, while OCDD predominates in others."

g. LT09 -- "CDDs/CDFs in the Ambient Atmosphere: A Baseline Study"
Bloomington, IN data of Hites

	Vapor	Particle		Vapor	Part.
TCDDs	20 fg/m3	ND	TCDFs	350	20
PeCDDs	50	25 fg/m3	PeCDFs	65	35
HxCDDs	20	120	HxCDFs	20	75
HpCDDs	25	280	HpCDFs	6	100
OCDD	150	750	OCDF	9	40

h. LT10 -- "Background Concentrations of CDDs and CDFs in Commercial Office Buildings"
Two "clean" buildings in Boston and three in Santa Fe. 2,3,7,8-TCDF found in 36/41 of wipe samples; only in

4/28 air samples. 2,3,7,8-TCDD was ND (100 pg/m2 and 1 pg/m3). Predominant presence of OCDD and OCDF. Compared to levels in PCB-fire buildings.

i. LT11 -- "Pattern Analysis of PCDDs and PCDFs in Environmental Samples as an Approach to a Source/Occurrence Correlation"
Looks at homologue profiles, D/F homologue ratios, isomer patterns, 2378-patterns. [This might be helpful.]

j. LT12 -- "PCDD and PCDF in Indoor Air of Kindergartens in N. FRG"
Examines indoor and outdoor air of facilities treated with PCP or Lindane. Levels in wood also presented. TEQs generated. See RM02.

k. LT13 -- "CDD/CDF Levels in Food Samples Collected in 1984-87 in N and S Vietnam"
Data from pork, duck, beef, chicken, turtles, fish and snakes.

l. LT16 -- "Environmental Levels of CDDs and CDFs"
General survey by Rappe. Sediments reflect long range airborne transport and local sources. Baltic Sea fish are about the same as 15-20 years ago. Levels of TCDF are found in deep (150 cm) sediment. This may be due to formation 300 years ago, or it may be due to some sort of "leaching" into the sediment.

m. PS04 -- "PCDD and PCDF in Mice and Insects from Various Environments"
Levels in insects are similar to the soil. Levels in mice (including skin), excised of the liver, are similar to soil. Liver is not like the soil. "...[H]erbivoran mice showed PCDD/PCDF levels much lower than carnivoran mice."

n. PS05 -- "Survey of Background Levels of PCDDs and PCDFs in UK Soil"
100 points of a 50 km grid. No data in the abstract.

o. PS06 -- "CDD and CDF Levels from Soil Collected in 1984-86 in N and S Vietnam"
An advertisement, not an abstract.

p. PS07 -- "Monitoring Ds/Fs in Precipitation Samples"
Samples obtained from a remote area. TCDD and TCDF are ND at 4-30 ppqd. OCDD was found 120-620 ppqd. Lower levels of HpCDD and HpCDF.

q. PS24 -- "Analysis of Fog Samples for PCDD and PCDF"
OCDD predominates, although HpCDD is the only other CDD present. T- thru OCDF found at lower levels.

r. PS25 -- "Concentrations of PCDD and PCDF in Soil from Vicinity of a Large Refuse Incinerator in Hamilton, Ontario"
Lack of correlation between predicted locations of high CDD/F levels and analytical results. And this at one of the highest "dirtiest" emitters. Hmmm.

s. PS26 -- "Mathematical Approach to the Data Analysis in Environmental Science. The Lecture Learnt from Seveso"

A critique of past reports.

t. PS49 -- "Recoveries of the Early Analytical Procedures to Detect 2,3,7,8-TCDD in Soil Samples in Seveso"
Makes the point that maps drawn before 1979 "...were characterized by underestimation, sometimes remarkable, of TCDD levels -- especially where contamination was higher."

u. PS56 -- "PCDD and PCDFs in the Environment as a Result of Outdoor Chemical Waste Burning"
Top soil concentrations of 2,3,7,8-TCDD up to 50 ppb. Total of 159 ppt TEQs. Sediments at nearby shoreline showed Pe- thru O- CDD/CDFs; no detectable 2,3,7,8-TCDD. Eel had only 82 ppb HpCDD; no other CDDs/CDFs. Rabbit livers had up to 500 ppt TEQs; 2,3,4,7,8-PeCDF accounting for 90% of the TEQs.

v. PS64 -- "Planar and Coplanar Polychloroaromatic Compounds in Baltic Salmon and White-Tailed Eagle"
PCBs, DDTs, and Chlordane present in the ppm range. Some PCDDs/PCDFs seen near 1000 ppt.

w. PS65 -- "Survey of 2,3,7,8-TCDD in Selected Plants and Animals from Times Beach"
Advertisement, not an abstract. [Should be interesting, though.]

6. Human Tissue Levels

There were 24 papers and 6 posters (PS-31, -46, -47, -53, -62, and -63) addressing this subject. Basically, they re-enforced previous information; e.g., background levels in fat and blood, half life values, industrial vs. non-industrial nations, 1 pg/kg-d intake, and higher levels in occupationally exposed folks.

Consult abstracts for details and some more interesting information (which is not to imply that this information is "uninteresting"!).

E. Risk Characterization (Risk Characterization section)

Risk characterizations were presented for

Human milk (RC02 - WHO)

Occupational setting (RC04 - Paustenbach)

Land application of paper mill sludge (RC07 - Envirologic Data).

Other papers raised questions about and pointed to the need for improvements in the exposure assessment of any RC:

RC01 - need for greater attention to dealing with uncertainty

RC05 - need to reassess exposure due to application to agricultural soils

RC06 - need for validation of air dispersion modeling.

Commenters noted that consumption of fish should yield 40 ppt in fat of people. Generally this is not seen. However, see paper by CA DOH: HT11.

F. Control Options (Control Technology section and selected posters)

MWCs, landfills, photochem, etc. treated.

G. Risk Management (Risk Management section)

Abstracts are self-explanatory. Particularly noteworthy is

RM02 -- "Indoor Air Pollution by Dioxins in Day-Nurseries: RA and RM". Cf. LT16.

III. Scuttlebutt

A. SOme researchers are examining coffee (thru paper filter) and tea (thru paper bag) for the presence of 2,3,7,8-TCDD and 2,3,7,8-TCDF.

B. The materials associated with the briefing of the Ad Hoc "Dioxin" Group to the Administrator on the "window" of 2,3,7,8-TCDD toxicity was released to EDF as a part of the discovery action related to the EDF-EPA law suit.

C. In Sweden, these sources are approximately equal contributors of CDDs/Fs into the environment:
MWCs, automobiles, steel plants, and pulp and paper plants

D. The CDC 1 ppb risk assessment assumed that a cow would ingest up to 2 kg of soil while grazing. This is an outer limit. A more realistic number would be .3 kg. (There is a question, however, as to whether the grass itself would have CDDs/Fs in/on the blades.)

E. I understand that concerns originally raised by the Battelle report on the collection efficiency of the EPA air sampler has been resolved; cf. Haggenmeir. Therefore, the Tier 4 data are probably firm. (We had thought that they might be as much as 10x too low.)

F. At the Univ of NV at Las Vegas 90% of the students are from in-state. (All but the basketball team?!) The University will give a 4-year "free ride" to the valedictorian of any NV high school.

G. There were more than 500 participants from 18 countries. Next year the conference will be in Sweden. The bidding has already begun for the following year. More people are questioning the need for an annual conclave.

H. Al Young didn't make it this year.

I. The Wizards may be able to help unravel the Battelle report.

J. 2,3,7,8-TCDD volatilizes off glass at 40 degrees C.

K. Harless is getting a new instrument, which will put him back into the hunt.

L. Not everyone agrees with Greenpeace's assessment of Barnes!

M. NTP is testing phorbol esters for carcinogenicity. The result could shed light on the question of TCDD's promotor/initiator status.

N. NTP is conducting skin promotion studies with PeCDFs.

O. The proposed TEFs for the NATO CCMS are as follows:

2,3,7,8-TCDD	1	2,3,7,8-TCDF	.1
2378-PeCDF	.5	2,3,4,7,8PeCDF	.5
		Other 2378-PeCDFs	.01
2378-HxCDD	.1	2378-HxCDFs	.1
2378-HpCDD	.01	2378-HpCDF	.01
OCDD	001 or .0001	OCDF	.001 or .0001

All non-2378-TCDDs are ignored since they do not show up in biological tissue and their collective impact is small in the current scheme. The guiding principal of this proposal is simplicity.

IV. Personal Activities

1. Presented plenary lecture on Risk Characterization. Paper is to be published.
2. Participated in a press conference with Eric Bretthauer, Gary Amendola, Alec McBride, Chris Rappe, and Ray Clement.
3. Participated in a conference call on levels in residue from incineration of 2,4,5-T and Silvex. I am on the point for discussing the model, which I strongly suspect as not being applicable in this case. Consider total 2,3,7,8-TCDD and the impact of local dispersion in the environment.
4. Chaired the session on Dose-Response Assessment.
5. With Judy Bellin, co-authored a paper on TEFs with Jim Olson of SUNY. Jim is the lead author and will prepare it for publication.
6. Chaired the session on Risk Characterization.
7. Presented final plenary lecture on Risk Characterization. Summary to be published.
8. Chaired a rump session of the NATO CCMS group addressing TEFs which led to a proposed "common set of TEFs" for consideration by member countries. I have to prepare this for more formal circulation to all participants and eventual publication.
9. I introduced myself to two young women from Greenpeace. We had a pleasant discussion about regulatory actions and inactions regarding "dioxin". I asked them to send me their views on the biggest dioxin problems facing us and their recommendations for action.
10. I met a woman from Ohio who is concerned about land application of 2,3,7,8-containing sludge to old stripmined lands. We have chatted on the phone a few times in the past. She had become aware of my recent notoriety. I put her in touch with folks from EDF and Greenpeace, as well as discussing some of her concerns with her.

One of them is Renate K. — not Kimbrough, but Kroesa,