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United States Environmental Protection Agency
Hudson River PCBs Reassessment Remedial Investigation/Feasibility Study (RRI/FS)
Community Interaction Program

Hudson River PCBs Oversight Committee Meeting
March 8, 2000
Saratoga Springs, NY

On March 8, 2000, as part of its Community Interaction Program, the United States Environmental Protection Agency (EPA) held a meeting of the Hudson River PCBs Oversight Committee (HROC) at the Sheraton Saratoga Springs, Saratoga Springs, New York. The EPA committee members present were Mel Hauptman, Leader of EPA's Sediments/Caribbean Team, representing HROC Chairperson William McCabe, Deputy Director of Superfund Program for EPA Region 2; Alison Hess, Remedial Project Manager; Doug Tomchuk, Remedial Project Manager; and Ann Rychlenski, EPA public affairs specialist and Community Relations Coordinator for the site. Other EPA representatives of the Hudson River PCBs RRI/FS team were Marian Olsen, Environmental Scientist, and Doug Fisher, Attorney. Other members of HROC in attendance included the following:

- Tom Borden, Agricultural Liaison Group Chair
- Andy Carlson, New York State Health Department Bureau of Environmental Exposure Investigation (NYSDOH)
- Darryl Decker, Government Liaison Group Chair
- Walt Demick, New York State Department of Environmental Conservation (NYSDEC)
- John Haggard, GE Hudson River PCBs Program Manager
- John Santacrose, Environmental Liaison Group Chair
- Judy Schmidt-Dean, Citizen Liaison Group Chair

The two guest speakers were Renate Kimbrough, M.D., chief investigator for a recent mortality study for General Electric Company (GE) and Dr. V. James Cogliano, Ph.D., Chief of EPA's Quantitative Risk Methods Group at the agency's National Center for Environmental Assessment in Washington, DC. Dr. Kimbrough presented the findings of her mortality study of workers exposed to PCBs at GE's two manufacturing capacitor plants in Hudson Falls and Fort Edward, NY. Dr. Cogliano discussed cancer and non-cancer health effects of environmental exposure to PCBs. The program for the evening was arranged in response to specific requests, made at the October 26, 1999 Steering Committee meeting by several liaison group co-chairpeople, that both EPA's and GE's experts on PCB health effects be invited to make presentations.

Mr. Hauptman opened the meeting by inviting anyone who did not get a full set of handouts to see Ms. Rychlenski after the meeting. The handouts were 1) the Agenda for the meeting, 2) copies of overheads for Dr. Kimbrough's presentation, 3) copies of the paper by Dr. Kimbrough and others (1999),

entitled, "Mortality in Male and Female Capacitor Workers Exposed to Polychlorinated Biphenyls," published in Journal of Occupational & Environmental Medicine, 41:161-171, March 1999, as well as the letters to the editors and the authors' response to the letters, all published in the September 1999 issue of the same journal, 4) copies of overheads for Dr. Cogliano's presentation, 4) copies of Dr. Cogliano's paper, entitled, "Assessing the Cancer Risk from Environmental PCBs," published in Environmental Health Perspectives, 106:6, June 1998, 5) copies of a paper by Dr. Cogliano and others (Brouwer et al., 1999) entitled, "Characterization of Potential Endocrine-Related Health Effects at Low-Dose Levels of Exposure to PCBs," published in Environmental Health Perspectives, 107:Supplement 4, August 1999.

The agenda for the meeting is Attachment 1. Sign-in sheets are found in Attachment 2. Hard copies of Dr. Cogliano's and Dr. Kimbrough's overheads and published papers are in Attachment 3. The updated RRI/FS schedule is Attachment 4. The use of brackets - [ ] - indicates clarifications made by the writer in cases where otherwise the text would be unclear to those not at the meeting. Copies of the audio tapes recorded at the meeting are available on request.

Ms. Hess reported on the risk assessments for the RRI/FS. Two human health risk assessments (HHRAs) have been released in recent months. The August 1999 HHRA addressed human health risks in the upper Hudson River. The December 1999 HHRA addressed human health risks in the mid-Hudson River. Two ecological risk assessments (ERAs) were also released. The August 1999 ERA assessed current and future risks in the upper Hudson River and current risks in the lower Hudson River. The December 1999 ERA assessed future risks in the lower Hudson River. Responsiveness summaries are being prepared for the human health and ecological risk assessments and will be released in March. Peer review of the risk assessments is tentatively scheduled to begin with an informational meeting on March 22 and 23, 2000 (selection of peer reviewers is still going on). At this peer review kick-off meeting, the peer reviewers will hear presentations on the risk assessment reports and some background information, and will receive the charge that is the focus of their review. The actual peer review, at which the peer reviewers will discuss their reviews and respond to the charge questions, will occur in May.

Mr. Tomchuk reported on modeling. The Baseline Modeling Report (BMR) was released in May 1999. At that time, EPA stated that it intended to further refine the model. EPA has also reviewed the comments received from the public on the BMR as well as GE's model. EPA issued the resulting Revised BMR in January 2000. The peer review for the Revised BMR, which began with a kick-off meeting on January 12-13, 2000, is still underway. The peer review meeting is scheduled for March 27 and 28, 2000. In addition to the Revised BMR, EPA also issued a Responsiveness Summary containing responses to comments on the BMR and explaining where in the Revised BMR changes based on comments could be found. EPA has also issued a Response to Comments on the first peer review, which was on the modeling approach. EPA will soon issue a Response to Comments on the second peer review on the Data Evaluation and Interpretation Report (DEIR) and the Low Resolution Sediment Coring Report (LRC). Mr. Tomchuk emphasized that [all comments] are being taken into account as additional reports are prepared [even if the responses to comments have not yet been finalized]. Mr. Tomchuk reminded everyone that the Feasibility Study Report (FS) and the Proposed Plan will be released in December 2000. The FS and evaluation of alternatives are currently underway.

Mr. Hauptman invited additional agenda items; none were forthcoming.

EPA was asked what will happen during the seven months between May 2000 and release of the FS in December 2000, and at what point in time EPA will lay out the nine evaluation criteria. Mr. Tomchuk replied that during that period, EPA will receive the draft FS from the contractor, review it, and prepare the Proposed Plan. The FS will contain an evaluation of seven of the nine criteria; the last two, state acceptance and community acceptance, will occur after the Proposed Plan is developed. Also during this time, EPA will respond to the two ongoing peer reviews and go through its internal decision-making process.

Mr. Carlson announced that NYSDOH has issued the final health consultation relating to the 1996 survey of Hudson River anglers. Copies are on the handout table and are available by calling Mark van Deusen on 1-800-458-1158 ext. 27530. This survey compared 1996 data to data generated in 1991-1992. Generally, results indicated better compliance with the state's fish advisory in the upper Hudson (Catskill and north), and no statistical difference from below Catskill to the Battery.

Mr. Santacrose requested that someone from the Attorney General's office be invited to a future meeting to present a status on the lawsuit relating to PCBs in the Hudson, and what impacts there may be from that lawsuit on [the RRI/FS] process.

Ms. Hess introduced Dr. Kimbrough, formerly with the Centers for Disease Control (CDC) in Georgia and EPA in Washington, DC and the principal investigator of a mortality study of workers at the GE capacitor plants in upstate New York. Dr. Kimbrough presented the findings of the report. She explained that she was part of an early study at the CDC that identified liver tumors in rats caused by PCBs. That study was shared with the National Institute of Occupational Safety and Health (NIOSH) and the chemical industry, resulting in one of the first studies in the GE capacitor plants, conducted by Dr. David Brown and colleagues in the 1970s. Subsequent studies at other plants followed, some of which indicated incidence of liver tumors, others indicated skin tumors, and others showed nothing. In the aggregate, Dr. Kimbrough suggested that there was "no real message," because the studies didn't support each other. She stated that normally in studies of exposure to a particular chemical, findings would be similar in similar settings.

The recent mortality study of 7,075 capacitor workers at the Hudson Falls and Fort Edward plants began in the 1990s; those individuals studied had a much longer time period between their exposure and the study than was the case with other studies. Dr. Kimbrough stated this was important particularly with regard to cancer because of the latency period associated with the disease: it takes quite a while for cancer to develop. This study determined how many workers who worked in these plants have died, and compared the reasons for their deaths to [statistics for] the general population.

To assist in understanding the figures on the overheads, Dr. Kimbrough explained the calculation process. Observed numbers of cancers are divided by expected numbers of cancers and adjusted for age; results indicate whether the incidence of tumors is more or less than expected. Dividends are multiplied by 100; numbers 100 or more indicate more tumors than expected, and numbers less than 100 indicate fewer tumors than expected. Further, confidence intervals are calculated to determine the upper and lower ends of the span of statistical probabilities. A lower confidence limit number below 100 indicates that the number is not statistically significant. Dr. Kimbrough said the study did not find any statistically significantly elevated cancer rates and that other diseases such as heart and cardiovascular disease were lower in the studied population, resulting in an overall lower death rate in this population. Finally, she also

said the workers tested had higher exposure than the general population - inhalation, dermal exposure from direct contact with PCBs during work, and skin absorption resulting from contact with PCB deposition on surfaces. Hard copies of Dr. Kimbrough's overhead slides are in Attachment 3.

Ms. Hess then introduced Dr. V. James Cogliano, Chief of EPA's Quantitative Risk Methods Group at EPA's National Center for Environmental Assessment, whose presentation was on health effects of PCBs and the reasons for EPA's concern.

Dr. Cogliano provided a brief overview of the difference between PCBs found in the environment and those used in commerce. Environmental PCBs are altered. In some cases this increases the persistence of toxicity, in others it decreases the persistence, and it allows scientists to focus on PCB exposures of greatest concern. He reviewed the chemical structure of PCB molecules, and discussed the various degrees of toxicity associated with the number of chlorines attached to the PCB molecule (up to ten).

Dr. Cogliano discussed some common terminology:

- PCB congener refers to configurations of chlorines at various positions on the PCB molecule;
- Homologues are congeners with the same number of chlorines; and
- Aroclors are mixtures of different congeners, with varying percentages of chlorine that gave the mixtures different electrical properties.

The fate of PCBs in the environment can be loosely related to the chlorine content. PCBs are not very volatile in general, but volatility is quite low for heavily chlorinated congeners and somewhat higher for less chlorinated congeners. PCB solubility in water is extremely low for heavily chlorinated congeners and increases as chlorines decrease. The highly chlorinated congeners, however, do adsorb, or stick to, soil and sediment very well, particularly soils and sediments with a higher carbon content. Further, higher chlorinated PCBs are more persistent in the environment than those with lower chlorinated content.

A PCB mixture released into the environment begins to separate; in the air and in the water there will be a higher proportion of the lower chlorinated congeners compared to what was originally released. In the soil and sediment the opposite is true. Differences also appear in living organisms. When living organisms take in PCBs, they try to metabolize them. Metabolism is easier if there are two adjacent positions on a PCB molecule without chlorines; the higher the chlorination, the less chance there is of having two adjacent unchlorinated positions. The result is that the congeners with higher chlorination tend to be retained more; this kind of bioaccumulation increases up the food chain. This leads EPA to focus on exposure pathways of greatest concern; the greatest concern is for bioaccumulated mixtures - PCBs found in fish or in birds that eat the fish. Another pathway of concern is contaminated sediment.

Dr. Cogliano discussed health effects of PCBs. He cited several studies that found evidence of various types of cancer, but the data are less compelling because of those differing types of cancer. Therefore, animal data are used in concert with human data to provide a good understanding of effects. With regard to whether just Aroclor 1260 or all PCB congeners were carcinogenic, Dr. Cogliano cited a study led by GE's Dr. Brian Mayes, study investigator, testing four Aroclor mixtures in parallel (1260, 1254, 1242, and 1016 - spanning the range of congeners). Dr. Cogliano stated it was a very good study in that it showed that all four congeners caused significant increases of liver cancer in rats. The potency differs for these four mixtures. Aroclor 1254 turned out to be the most highly carcinogenic in this test,

approximately 30 times more potent than Aroclor 1016, and Aroclor 1260 was next highest. This enables EPA to ascribe potencies to these Aroclors in the environment.

Dr. Cogliano's presentation covered not only cancer risks, but significant adverse effects attributed to PCBs other than cancer, including learning deficits, neurological effects, immune system dysfunction, thyroid effects, and hormonal effects. He reviewed several recent epidemiological studies that have raised concern about environmental exposure with regard to non-cancer hazards. These studies are supported by results of animal studies. Hard copies of Dr. Cogliano's presentation are found in Attachment 3.

A question and answer period followed the presentations, beginning with HROC members.

1. Question: Mr. Decker. Dr. Cogliano, do species higher on the food chain tend to metabolize higher chlorinated PCBs?

Response: There are metabolism differences across species; it is not a generalization that species higher on the food chain would be metabolizing the higher chlorinated PCBs any better. Some congeners are very strongly bioaccumulated as you go up the food chain, which would tend to indicate that metabolism does not become more efficient.

2. Follow-up question: Is there any indication that fat levels in animals have any corresponding relationship to PCB accumulations?

Response: Fat levels should have a relationship to PCB accumulation; PCBs are stored primarily in the fat.

3. Question: Mr. Haggard. Dr. Kimbrough, would you comment on the non-cancer issues you have worked on, and comment on the fish-eating issues that Dr. Cogliano raised?

Response: Dr. Kimbrough mentioned a number of clinical studies from her tenure at the CDC that showed no adverse health effects. She said that the studies Dr. Cogliano cited involving children of mothers who ate contaminated fish have been criticized. Although not definitive, concerns that have been raised include issues with exposure, where in some of the mothers, PCBs either were not measured or were below the limit of detection. Dr. Kimbrough stated that people in whom immunological effects, effects on birth weight, and effects on thyroid have been observed are not sick with a clinical disease; these are variations within the range of the distribution of what is normal, with the exception that some mothers have higher PCB levels. In the Dutch studies, children who were breast-fed, getting PCBs from their mothers' milk, "did much better" than the children who were bottle-fed, indicating there were other factors. Further, Dr. Kimbrough contended that the "startle" effects and the effects on memory in early infancy cited by Dr. Cogliano in his discussion of some of the studies are not predictive of what will happen later on in the children's lives.

4. Question: Ed Valentine, who worked in the Hudson River with a construction company prior to any PCB advisories. In 1974, his company was contracted to remove sediments from the river at Fort Edward, unaware that the sediment "was loaded with PCBs." The gentleman stated he now has a blood disorder, myelodysplasia, that his doctors say is "petroleum-related."He views the responsibility for initial removal of the dam and for subsequent activity as a combined federal and state responsibility, and inquired what

the state or federal government would do for people who worked there and were so exposed. He stated the people who worked on that job were not taken care of by either the state, federal government, or the contractor.

Response: Mr. Carlson, NYSDOH, said the answers this gentleman had received from his physicians probably accurately characterized the state's ability to say anything about any one individual and exposures. The studies discussed were aggregate studies. Mr. Carlson offered to provide the speaker with a telephone number at the DOH where he could reach someone to discuss his circumstances and get additional advice. Dr. Kimbrough stated that what the speaker described is unrelated to PCBs.

5. Question: Andy Revkin, *The New York Times.* Dr. Kimbrough, do you feel the EPA is overestimating the risk to humans from PCBs in the environment?

Response: Some of this is a policy decision based on certain methodologies the Agency is using, as [Dr. Cogliano] described. Dr. Kimbrough stated that she feels [EPA is] overinterpreting the "non-cancer" health effects described in children.

Follow-up Question: Do you think you can make an inference from human occupational exposure study to general risk in the environment to people who have a dietary exposure route?

Response: Dr. Kimbrough. The mortality study does not look at cancer incidence, which includes people who are cured. For most cancers there is a relationship between the number of people who die of cancer and the number of people who are cured. The relationship is "pretty consistent" and therefore even though the study does not include incidence, it provides "some degree of comfort" that PCBs are not a "smoking gun." Her study did not address what she called the "subtle effects" in infants that Dr. Cogliano described.

Dr. Cogliano. To use human occupational studies to estimate the risk of human environmental exposure, both a good estimate of the incidence or the mortality from and a good quantitative estimate of the exposure are needed. This is what is lacking in all occupational studies. PCB mixtures differ between the inhalation and food chain exposure pathways; further, we do not have a good estimate of what/how much workers were exposed to in the occupational study, so it is difficult to come up with a good dose-response curve. That is why EPA bases its dose-response curves on animal studies. The studies of children of mothers who had eaten PCB-contaminated fish were qualitative, indicating what can be expected -- learning deficits and neurological effects -- and were not used to set safe doses. Animal studies were used to determine a level without those effects; these studies differ from human studies in that there is a very precise dose given and a specific response.

Dr. Kimbrough commented that one cannot voluntarily expose humans to PCBs, but the dilemma is that one does not know whether the animals always respond as people do.

6. Question: Dr. Andi Weiss Bartczak. Dr. Kimbrough, what was the age range of the workers in the study?

Response: The total age range was from 20 to 70.

Follow-on: Based on that number, why did you choose cancer mortality rather than morbidity, when it is known that death certificates are quite inaccurate? Did you go back and check the certificates? Assuming most of the workers were still alive, given today's life expectancy, you ignored most of the workers and the damage that could have been done to them to go to an easier method of study.

Response: 1) By the time we got there, there was another lag of about 20 years, so it made more sense to conduct a mortality study. An incidence study would have been more difficult, and the comparison data is not as good. 2) No, there is greater uncertainty with rare diseases than with the garden variety of diseases we found. 3) I conducted the mortality study to get some idea of what was going on in the work force, and what we found was that nothing much was going on. The inaccuracies are not as great as you describe them.

7. Question: Dr. Richard Mansfield. I gather there has been no long term studies of the people who have been exposed the longest period of time.

Response: Dr. Kimbrough. When I was at CDC, they and NIOSH asked Monsanto about looking at their workers. Monsanto officials said they had very few workers exposed to PCBs, and suggested CDC and NIOSH go to the user. That is how David Brown ended up studying the GE plants in Fort Edward and Hudson Falls. There you had larger numbers of workers with higher exposures. In response to a follow-on regarding other users, Dr. Kimbrough said the capacitor plants really had the highest exposures; in the capacitor plants the PCBs were heated, "which greatly increases...." the exposures of those workers.

Follow-on: So you feel workers in the capacitor plants got the maximum dose anyone in the industry would have gotten.

Response: There may have been individuals in some industries that may have had high exposures, but if you want a large population and sufficient [statistical] power, you go to a capacitor plant.

8. Question: Bobbi Orsi, Registered Nurse from Pittsfield, MA. When Dr. Kimbrough's study came out, GE said they could take this study and apply it to the situation in Pittsfield: people in the plants have less mortality than you do [here in New York], with less exposure in the neighborhoods than in the plants, so "you really have nothing to worry about." Can you comment on that "huge leap that I think they took?"

Response: Dr. Kimbrough stated she could not comment because she would have to evaluate the situation in Pittsfield. She asked about the PCB levels in blood in the population that the State of Massachusetts tested.

Follow-on: In response to Dr. Kimbrough's comment on levels of PCBs in blood, what the state told the community was that along the Housatonic River the blood [PCB] levels were well within the background range, but there were problems with that study as well. The speaker's concern with Dr. Kimbrough's study was that it looked at job titles and length of employment as opposed to looking at blood [PCB] levels as a correlation to exposure.

Response: Dr. Kimbrough. There were some workers in some of those jobs with PCB levels. If you look at those blood [PCB] levels and the jobs used to classify exposure, they correlated very well. The problem was that [in total] we had about 200 blood [PCB] levels, and [the study involved] 7,075 workers; they did

not really represent the work force. [Study investigators] are satisfied that they were not inconsistent with what was done *vis-a-vis* job classifications.

Follow-on for Dr. Cogliano: Could you comment on the difference between a secretary exposed to Aroclor 1016 and a woman eating contaminated fish from the Housatonic River or Goodrich Pond.

Response: Dr. Cogliano: "I think there is a leap that has been made in lots of cases in applying this study to environmental exposure." He reiterated that PCB mixtures differ between inhalation and consumption of contaminated fish; the Brian Mayes [GE] study showed those differences in potency to be approximately thirtyfold.

Further, Dr. Cogliano took issue with the characterization of Dr. Kimbrough's study as a "study of capacitor workers," explaining that although there were capacitor workers included, the study would more appropriately be characterized as a study of people who worked in a capacitor plant. He referenced Table 3 of Dr. Kimbrough's presentation. The table shows people who worked in areas where PCBs were used, and it is a minority of the total cohort. The comparison he would like to see is what is the cancer experience among the people who really worked with PCBs and had the highest exposure. Although Table 7 and Table 8 address this, Dr. Cogliano observed that the numbers were small. Table 8 has mortalities from cancer among people who had worked with PCBs a year or more. The number of deaths in that category was 29, and Dr. Cogliano stated it is difficult to draw firm conclusions from that small number of deaths. He feels it is a very good study in terms of follow-up and diligence in tracking people down, but most of the workers were not capacitor workers and did not have high documented exposures to PCBs. Dr. Cogliano said it is "statements that we hear around the study that [are] not made directly in the study that have taken it beyond where it should go."

9. Question: Michael Rivlin, *Amicus Journal*. Mr. Rivlin acknowledged Dr. Kimbrough's reputation as a "fine and cautious scientist." He asked Dr. Kimbrough if she thought the results of her study may have been misrepresented or exaggerated? Citing a headline indicating that Dr. Kimbrough's study showed no link between PCBs and cancer, Mr. Rivlin pointed out that the study only addressed inhalation and dermal exposure, not the primary pathway for human risk from PCBs, which is ingestion. This is the problem the EPA is looking at.

Response: Dr. Kimbrough stated nothing is ever black and white; "it doesn't really matter whether you take PCBs up through the stomach or through the lung or through the skin." She said that once PCBs get into the body they have the same effect, so in that respect inhalation and dermal studies are relevant.

Follow-on: 1) So you would disagree with EPA, which has said inhalation is not an important exposure pathway? 2) Mr. Rivlin restated his original question about Dr. Kimbrough's results being misrepresented in some of the materials not part of her original study, or in comments about her study.

Response: 1) Dr. Kimbrough acknowledged EPA's position that PCBs ingested by eating contaminated fish are the more chlorinated and persistent congeners. The mixture of PCBs from fish would have been slightly different than what the workers might have gotten, but what the workers retained, when studied later on, "will look quite the same as what you see in people that have eaten fish." 2) Dr. Kimbrough said some reports were pretty accurate, some may have been exaggerated, but that she did not pay attention and did not know the answer.

10. Question: Roger Gray. Dr. Kimbrough, based on your studies, would you say we could ignore the NYSDOH fish advisory? In terms of my health, can I ignore the advisory?

Response: Dr. Kimbrough said she does not know anything about the levels in the fish, and that [the fish advisories] are a policy decision. "My study shows no increased mortality, so if you are concerned about your mortality, I guess you don't need to be concerned." Dr. Kimbrough stated all the levels including FDA levels are based on animal data, not on human data, which does not seem to support [the health risk]. In response to prodding, Dr. Kimbrough stated she did not want to get into policy decisions or make comments against what the NYSDOH does.

Ms. Hess stated that EPA encourages everyone to follow the fish consumption advisories put out by the NYSDOH.

11. Question: Pete Sheehan. Dr. Kimbrough, how did you decide on the cut-off of three months when you based your study on people who had worked at the plant three months or more?

Response: Dr. Kimbrough replied that the advisory board working with her felt that after three months, study subjects would have a sufficient [PCB] body burden to be meaningful. It is unknown when people go into a "steady state" with regard to blood [PCB] levels; with reference to other studies using one day or one week of exposure, Dr. Kimbrough said with that short a time, there is not enough body burden.

Follow-on: Do you know how many workers there would have been if you counted those with less than three months?

Response: Dr. Kimbrough said there were probably another 2,000, for whom there was data available, but it was not included.

Follow-on for Dr. Cogliano: Do you have any idea of how long an average person working in a capacitor plant would take to accumulate PCBs to where they would be a health danger to that individual?

Response: Dr. Cogliano said there have been studies of how much PCBs people would retain, and what the half-life elimination would be for various congeners. In predicting how long it would take to reach a steady state [in the body], in general he feels it would take more years for the more highly chlorinated [PCBs], more persistent congeners and a lesser number of years for the lower chlorinated congeners. Dr. Cogliano drew a distinction between how long it would take someone working in a capacitor plant to reach a steady state, and how long it would take someone highly exposed to PCBs. Someone who just works in a capacitor plant may not be exposed enough to build up much of a body burden over a couple of months or even years. He said it is really more a question of exposure: the fact that individuals are exposed, to what, and how much.

12. Question: Dr. John Brown of GE. Dr. Brown has been working on PCBs in GS's research laboratory since 1975. He stated the capacitor workers exposed to Aroclors 1016 and 1242 accumulated a great deal more PCBs than those exposed to Aroclors 1254 or 1260 because of the much higher volatility [of the former]; in fact there were workers in the directly exposed capacitor group who accumulated more PCBs than many of the rats in the rat study.

He asked Dr. Cogliano to explain his "selective use of the data that came from the Mayes [GE] study." This study was done by a conventional maximum tolerated dose chronic bioassay, a procedure that has given positive results for more than half of all chemicals tested. Therefore, finding a positive result is not necessarily an indication that PCBs are any more risky than "everything else in our present environment or on our dinner plate." Also, although the rats showed a higher incidence of liver tumors, they showed a lower incidence of total tumors, due to reduction in the incidence of mammary cancers and fibromas in those rats. Finally, female rats in the dose groups lived considerably longer than in the controls. Dr. Brown suggested that if one took the rat data at face value, PCBs had a greater anti-cancer effect than carcinogenic effect. On this basis, he asked, how do you go about calculating a cancer risk for them?

Response: In response to the comment about half of the chemicals tested being carcinogenic, Dr. Cogliano pointed out that chemicals are not tested at random; chemicals are tested for cancer when there is reason to suspect that they may cause cancer. Of the approximately two or three thousand of the hundreds of thousands of chemicals in commerce that have been tested, EPA only considers about 200 to be potential carcinogens. PCBs have caused liver cancer in earlier studies, so there is a reason to look for that.

With respect to the observation of increased liver tumors but larger decreases in mammary tumors in the rats, Dr. Cogliano said EPA has looked at cancer on a site-by-site basis, not on an overall basis, and would "not be willing to claim that there is a protective effect of PCBs on mammary tumors." EPA does see increases in liver cancer, sometimes accompanied by shifting patterns of mortality. Regarding increases in lifespan of dosed animals, Dr. Cogliano said it is true that cancer does increase with increasing longevity. What EPA saw in the controls was one percent; what EPA saw in the dosed animals (e.g., with Aroclor 1254) was 40 to 60 percent. The difference in longevity would not be able to explain that magnitude of an increase of cancer risk; therefore EPA does feel that the liver cancer increase is real, and the cancer risk estimate is based on that finding.

Follow-on: Dr. Brown said it is his understanding that most of the testing is based on a national policy to screen chemicals on the basis of their volume of production rather than risk; further, results of evaluation have concluded that even with correction for selection bias, maximum tolerated dose testing does give positive findings more than half the time, even on randomly selected chemicals.

Response: Dr. Cogliano. The National Toxicology Program [part of the National Institutes of Environmental Health Sciences of the National Institutes of Health] has begun high production volume testing as a matter of foresight, but by and large the chemicals that have been tested are those that short-term tests have revealed to have adverse health effects. A lot of chemicals are tested because they are suspected of being carcinogenic. EPA does not test only at maximum tolerated dose; with PCBs and other chemicals as well, cancer was seen at all doses tested with some of the Aroclors. No testing goes down to levels of PCBs encountered in the environment. The chronic bioassay in animals for carcinogenicity testing is state-of-the-art, used by the National Toxicology Program, and EPA does pay attention to those results.

13. Question: Robert Henshaw, Hudson River Environmental Society, Inc. Questions for clarification. 1) For Dr. Kimbrough. Is it correct that you said that when you received effect numbers less than 100, you

lost statistical accuracy? 2) For Dr. Cogliano. In the Lake Michigan and Lake Ontario fish consumption study, were the children of mothers who consumed PCB-contaminated fish exposed during their post-nursing lifetime or was there only exposure during breast-feeding?

Response: Dr. Kimbrough. 1) It does not mean [the numbers are] not accurate; it means [they] would not be statistically significant. It means that whatever you find in the exposed population is comparable to the general population. We ran statistics on everything [for the mortality study]; items in the tables with asterisks are statistically significantly different and explanations are in the footnotes. Items without those asterisks indicate values comparable to the general population.

Dr. Cogliano. 2) The children were selected because the mothers consumed fish, and they did so during family dinners. Because it is assumed this continued, it can be assumed that some of the children continued to be exposed directly through diet as well. A lot of those effects are ascribed to the mothers' consumption because the effects were observed early, during infancy when the biggest concentration would have been from the mother. Those children are still showing some effects as late as 11 years later, indicating the effects could be persistent. It is very difficult to separate the effects of pre-natal exposure, PCBs in breast milk, and later exposure, but effects are being observed.

14. Question: Irwin Sperber. Dr. Kimbrough, what is your view of the part of Dr. Cogliano's presentation on the nature of PCBs under actual field conditions such as those in the Hudson River? The speaker quoted Dr. Cogliano as saying PCBs "become substantially more hazardous, more likely...[to result in] carcinogenicity and potentially immunosuppressive effects," and asked if this is consistent with her own knowledge and understanding.

Response: Dr. Kimbrough. PCBs are changed in field conditions but also are changed in the body. Even though workers are exposed to commercial mixtures, after they cease having exposure to commercial mixtures, their body burdens will look quite similar to what you would find in the fish-eater. The sources and type of materials taken in may be somewhat different between a capacitor worker and a fish-eater, but what happens in the body is about the same, eventually resulting in the same body burden. What is of concern is the persistence and build-up of body burden of these types of congeners. Other congeners that are rapidly metabolized may also contribute, but we have not seen evidence.

Follow-on: Mr. Sperber said Dr. Cogliano was calling attention to particular changes in PCBs that pose substantially greater risks; he quoted Dr. Cogliano as having referred to risks thirty times more serious than original exposure to capacitor workers. He asked Dr. Kimbrough if she "believed" her research findings based on the types of PCBs being documented, and whether it is a "contribution to good public health science and good research." He said he felt there had been no good faith efforts, based on publications about the study and various GE announcements and public relations releases, to distinguish between the PCBs facing capacitor workers and the "significantly greater risks" posed by PCBs having undergone changes "under field conditions" and winding up in the food chain, ultimately to be ingested by people.

Response: Dr. Kimbrough, addressing Mr. Sperber's reference to the thirtyfold increase in risk, pointed out that Dr. Cogliano was referring to the difference in potency of PCB mixtures in the rat studies. With Aroclor 1016, fewer cancers were observed than when rats were given 1254. She said workers in the 1950s were working with Aroclor 1254 and were still working with Aroclor 1254 in some later years;

Aroclor 1016 was not really used until 1971, and in 1977, its use was stopped. Therefore, many of those workers had exposure to the more toxic PCB mixture. As to what people are exposed to when they eat fish, Dr. Kimbrough said some fish break down PCBs better than others, and only store the more persistent congeners, so that the mixture looks different. The mixture looks similar to what the body burdens would look like in the studied worker population if you looked at them down the road. Those workers also had environmental exposure; they lived here, so they also probably ate fish and were exposed to other sources of PCBs in the 1970s and 1980s.

Follow on: Mr. Sperber asked Dr. Cogliano if the PCBs absorbed through the skin by the capacitor workers would eventually wind up having the same molecular constitution and risks as the PCBs that are in the sediments, end up in striped bass, and are ingested.

Response: Dr. Cogliano. It is a complicated question. Different levels of the food chain and humans do what is called "filtering," selectively retaining some congeners more than others. It is true that over a long period of time a person retains some more than others of whatever PCBs are being taken in - whether by ingestion, inhalation, or dermal contact; over time the person will attain a body burden of the more persistent congeners. A person exposed to Aroclor 1016, getting only congeners with one to four chlorines, is not going to retain congeners with seven chlorines from that. More of the "fours" than the "threes" will be retained, more of the "threes" than the "twos" will be retained, perhaps. While that is what happens to the body burdens down the road, it does not negate the results in the rat study: rats that ingested Aroclor 1016 all their lives did get liver tumors, but fewer liver tumors than the rats that ingested [the more toxic] Aroclor 1254 all their lives. Even though eventually the body burdens might look similar from different exposures, the toxicological consequences are different depending on which mixture constitutes the primary exposure.

15. Question: Peter Tarana. Dr. Cogliano, why has EPA not sponsored an epidemiological study--is relying on epidemiological evidence to assess human health risk not a part of policy-making?

Response: One reason for not sponsoring that type of study is that it is difficult to characterize a group to study that would have high exposure to PCBs and not to other chemicals, so that the study would really be definitive. EPA looks at studies that exist in literature and at the animal experimentation, and makes the best judgment possible about what chemicals cause the greatest risks..

Follow-on: Do you think you could use and improve on the Kimbrough study using the original GE cohorts? That is a population that is massively exposed to PCBs and also probably fished and ate fish.

Response: Dr. Cogliano said he did not know that EPA could do a better epidemiological study now. Tables 3 and 8 in Dr. Kimbrough's study show the number of people who had high exposure to PCBs who have died and whose death certificates saying "cancer" contributed to the study (a total of 29). At the end of the study, the average age was 57 among all those workers, still relatively young with regard to the age where cancer develops. Twenty years from now more will be known about what happened to the individuals in those cohorts. Dr. Cogliano would like to see an incidence study, but EPA is looking at environmental exposures, not just workers. It would be difficult to do an epidemiological study to address all the concerns: PCBs from fish with different responses than the Aroclors to which workers are exposed; the general population's being exposed to other things; children's issues; people who have other health problems, etc.

16. Question. Dr. Jay Silkworth of GE. Question for Dr. Cogliano. The effects on the human cohorts that have been discussed are caused by a number of different chemical classes, including but not limited to PCBs; the same is true for effects on animals in animal studies. He asked how certain can Dr. Cogliano be of policies that are derived from epidemiological cohorts when only PCBs are measured and co-contaminants are not?

Response: Dr. Cogliano. This is what EPA wrestles with all the time in setting standards. Dr. Cogliano repeated what he had earlier stated, that the fish eaten by the mothers [in the Great Lakes study] had PCBs but also other contaminants, and effects of PCBs cannot be sorted out from effects of other persistent compounds. To set standards, you want a good estimate of the effect and a good estimate of dose. This is why EPA sets its non-cancer reference doses and its cancer risk estimates based on animal studies where effects analogous to effects in humans have been observed, but in relation to exposure to PCBs alone. Going to animal studies introduces some uncertainties that humans respond in the same way. Qualitative evidence from human studies exists that humans could respond in the same way, but quantitatively that is not totally nailed down.

Dr. Cogliano said it would seem "we should be able to get better data to precisely measure these effects," but the difficulty is in finding the cohorts to study - people with exposure only to PCBs. The question for EPA is what to do before ideal data become available. EPA has chosen to take action to be protective in the meantime; "EPA takes a protective approach in the face of uncertainty."

Follow-on: Dr. Silkworth [GE] said he thought EPA should demand full assessment in these studies of the other contaminants likely to have similar activities in these cohorts as part of study design.

Response: Dr. Cogliano said that is a very good standard to hold researchers to - to try to characterize the whole profile of contaminants present. Most past studies do not meet that standard, and again, EPA is faced with the question of what to do with information that is incomplete but still suggests there might be a risk.

Follow-on: Dr. Silkworth: "One of the things you do is scare a lot of people when statements are made relative to risks of exposure when you don't know with any high degree of certainty at all whether it is due to the agent - or any agent - in question."

Response: Dr. Cogliano stated that he hopes EPA does not scare people but rather provides information about what exposures might be potentially risky and what might not be. "Total ignorance is also scary," he said, and he feels a lot of people would like to know of potential risks.

17. Comments. Jim Regan of NYSDEC. For Dr. Cogliano. In doing an environmental study, it is impossible to eliminate the other possibilities and factors; this is an inherent limitation in doing environmental studies. This is why you go to animal studies where you can focus in on one contaminant.

Response: Dr. Cogliano: "I think you made the point better than I did. People are not exposed cleanly to one substance....It is very difficult to separate out all these other exposures people have."

Follow-on: Would you comment briefly on the investigations at the St. Lawrence River at Massena [New York] - fish study, wildlife study based on consumption, and breast milk study. Also, could you briefly discuss EPA's recent reevaluation of risks from PCBs?

Response: Dr. Cogliano was not familiar with the Massena studies [led by Dr. Ed Fitzgerald of NYSDOH]. The slope factor for the cancer risk assessment was released by EPA in 1996, and Dr. Cogliano wrote an article for Environmental Health Perspective in 1998 - these are the most recent evaluations of the cancer risks associated with PCBs. EPA is currently addressing non-cancer health hazards in response to some of the studies of women who consumed PCB-contaminated fish to see whether EPA should reevaluate the non-cancer reference doses for PCBs calculated from the monkey studies. That draft report will not be ready before 2001.

Follow-on: Was there much change in EPA's evaluation of cancer risk based on the 1996 reevaluation?

Response: Before 1996, there was a controversy over whether only Aroclor 1260 could be carcinogenic, or whether other PCB mixtures could also pose a cancer risk. Because Aroclor 1260 was the only Aroclor that had been tested well, EPA only had one slope factor and treated all PCBs as if they were as potent as Aroclor 1260. What changed in the 1996 cancer risk evaluation was that EPA had better information that all PCB mixtures could cause cancer, but also that different mixtures had different potencies. This enabled EPA to treat some mixtures in the environment as more potent, and make some sensible distinction among environmental mixtures. Qualitatively, EPA has better information that PCBs could cause cancer; quantitatively EPA has information indicating that some PCBs are quite a bit less potent than the old number based only on Aroclor 1260.

19. Question. David Mathis. Due to PCBs in the upper Hudson River, is there an increase in health risk, yes or no?

Response: Dr. Cogliano: I think there is an increase in health risk.

The meeting was adjourned.

ATTACHMENT 1

U.S. EPA H.R.O.C. Meeting

Hudson River PCBs Superfund Site Reassessment
Community Interaction Program

Hudson River PCBs Oversight Committee
Wednesday, March 8, 2000 - 7:30 p.m.
Saratoga Springs, New York

AGENDA

Welcome & Introduction	Mel Hauptman, Acting Chair HROC, USEPA
Committee Report-Outs (20 minutes)	Mel Hauptman, Acting Chair
Guest Speakers on PCB Toxicity	Renate Kimbrough, M.D. (30 minutes)
	Jim Cogliano, Ph.D. U.S. EPA (30 minutes)

Questions & Answers

ATTACHMENT 2, 1-6

US ENVIRONMENTAL PROTECTION AGENCY
HUDSON RIVER PCBs REASSESSMENT
REMEDIAL INVESTIGATION/FEASIBILITY STUDY

Community Interaction Program
Hudson River PCBs Oversight Committee Meeting
Saratiga Springs, NY
March 8, 2000

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Alan Belensz	NYS DOL	
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US ENVIRONMENTAL PROTECTION AGENCY
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Community Interaction Program
Hudson River PCBs Oversight Committee Meeting
Saratiga Springs, NY
March 8, 2000

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Richard Manfield Ph.D.	26 F Congress St #148 Saratoga Spgs NY 0866	Saratoga Chemical, Inc.
E Kamm	104 Walla Ln Hamden CT 06514	GE
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Bob Edwards	50 wolf Rd ALBANY	NYSDEC

ATTACHMENT 2, 2-6

US ENVIRONMENTAL PROTECTION AGENCY
HUDSON RIVER PCBs REASSESSMENT
REMEDIAL INVESTIGATION/FEASIBILITY STUDY

Community Interaction Program
Hudson River PCBs Oversight Committee Meeting
Saratiga Springs, NY
March 8, 2000

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Marygrace Brown	48 Ventura Ave Pittsfield	413 442 7733 Residents Env. action League Cof R.E.A.L.

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ATTACHMENT 2, 3-6

US ENVIRONMENTAL PROTECTION AGENCY
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Lee Coleman	Daily Gazette 376 Broadway Saratoga Springs NY 12866	Daily Gazette
J. Miller	c/o 640 Bay Rd. Queensbury NY	—

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Andy Revkin	NY Times 229 W43rd NY NY 10036	914 424 3530

ATTACHMENT 2, 4-6

US ENVIRONMENTAL PROTECTION AGENCY
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BRIAN MAYES		GE
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Ed Fitzgerald	Fla. Jan Sq, Troy 12180	NYSDOT 518-402-7960
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US ENVIRONMENTAL PROTECTION AGENCY
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Andy Carlson	NYS DOH ~~Albany~~ Troy	NYS DOH
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ATTACHMENT 2, 5-1

US ENVIRONMENTAL PROTECTION AGENCY
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Community Interaction Program
Hudson River PCBs Oversight Committee Meeting
Saratiga Springs, NY
March 8, 2000

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Jim Musial	"	"
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March 8, 2000

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ATTACHMENT 2, 6-1

US ENVIRONMENTAL PROTECTION AGENCY
HUDSON RIVER PCBs REASSESSMENT
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March 8, 2000

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GE Study of 7,075 Capacitor Workers *

- Renate D. Kimbrough, M.D.
  Principal Investigator
- Martha Doemland, Ph.D.
- Maurice LeVois, Ph.D.

*Worked for at least 3 months between January 1, 1946 to June 15, 1977

ATTACHMENT 3
1-23

Other Mortality Studies in Capacitor Workers

(2)

Reference	Workers	All Cause	All Cancer	Specific Causes
Bertazzi et al. 1987	2100 total 544 Males	Males O/E 30/27/8 SMR=108	Males O/E 14/5.5 SMR=253 95% C.I. (144-415)	Males Carcinoma GI Tract O/E 6/1.7 SMR=346 95% C.I. (141-721)
(The increases in cancer were no longer observed in a recent follow up)	1556 Females	Females O/E 34/16.5 SMR (local) = 206	Females O/E 12/7.7 (5.3 local) SMR=156 (N.S.) SMR (local) = 226 95% C.I. (123-385)	Females Hematological O/E (local) 4/1.1 SMR=377 95% C.I. (115-877)
Brown & Jones 1981	2567 1258 Males 1309 Females	Male and Female O/E 163/182 SMR=89 95% C.I. (76-104)	Male and Female O/E 39/44 SMR=89 95% C.I. (63-122)	Male and Female Rectal Carcinoma O/E 4/1.19 SMR=336 95% C.I. (92-860) Total Liver Carcinoma O/E 3/1.1 SMR=280 95% C.I. (58-820)

Referen.	Workers	All Cause	All Cancer	Specific Causes
Brown, 1987 Follow-up of Brown & Jones	2588 1270 Males 1318 Females	Male and Female O/E 295/318 SMR=93	Male and Female O/E 62/80 SMR=78	Male and Female Rectal Carcinoma 4/1.9 SMR=211 (n.s) Total Liver O/E 5/1.9 SMR=280 (p<0.05)
Sinks, et al 1992	3588 2742 Males 846 Females	Male and Female O/E 192/283 SMR=70 (p<0.01) 95% C.I. (60-80)	Male and Female O/E 54/64 SMR=80 95% C.I. (60-110)	Total Melanocarcinoma O/E 8/2 SMR=400 (p<0.01) 95% C.I. (180-800) Total Brain, Nervous System 0/E 5/2.8 SMR=180 95% C.I. (60-420)
Taylor, 1988	6292 3601 Males 2691 Females	O/E Males 355/430 SMR=83 95% C.I. (74-92) O/E Females 155/185 SMR=84 95% C.I. (71-98)	O/E Males 69/84 SMR=83 95% C.I. (64-105) O/E Females 67/61 SMR=110 95% C.I. (85-140)	None were significant

(2)

ATTACHMENT 3
2-23

10.9354

IEHR Advisory Committee on the Mortality Study of General Electric Capacitor Workers

- **Arthur C. Upton, M.D.**
  Environmental and Occupational Health Sciences Institute (UMDNJ-RWJMS and Rutgers University), Piscataway, NJ
  (Former Director of the National Cancer Institute)

- **Gilbert W. Beebe, Ph.D.**
  National Cancer Institute, Bethesda, MD

- **Jack S. Mandel, Ph.D., M.P.H.**
  University of Minnesota, Minneapolis

- **John E. Vena, Ph.D.**
  State University of New York at Buffalo

- **Roy E. Shore, Ph.D.**
  New York University Medical Center

Strengths of Study

- Largest cohort (7,075 workers)
- Highest number of person years (212,778)
- Only 1.3% of workers lost to follow-up
- Largest number of deaths with 1,195 death certificates
- Advisory committee of experts

Determining Death

- Equifax: Social Security mortality tapes
- National Death Index since 1979
- Obituaries tapes
- Tapes of the Veterans Administration
- Private investigator

Completeness of Cohort

- Review of pension records
- Review of Social Security Administration 941 Forms = quarterly earning reports
- Review of GE employee profile data base

Demographic Characteristics*

Characteristic	Hourly Male	Hourly Female	Salary Male	Salary Female	TOTAL
Number of workers	2984	2544	1078	469	7075
Number of deaths	586	380	177	52	1195 (17%)
Number lost to follow-up	33	52	7	3	95 (1.3%)
Mean years worked	6.2	5.8	5.7	4.8	
Number ever highly exposed	1268	352	87	10	1717
Number ever undefinably exposed	2031	133	407	9	2580
Number ever low exposed	2343	2468	831	459	6101
Mean years of follow-up	28	30	32	34	
Percent attended college	15	7	73	26	

*Inclusion criteria: must have worked for at least 3 months between 01/01/46 - 06/15/77.
Total person years 212,778

ATTACHMENT 3
5-23

USAGE OF PCBs

1946 - 1954	Aroclor 1254
1955	Aroclor 1242
1956 - 1971	Aroclor 1254, 1242
After 1971	Aroclor 1016
June 30, 1977	All PCB Usage Discontinued

(9)

GE Data, October 1975
Area Air Survey

Plant	H.F. Air Levels $\mu g/m^3$	F.E. Air Levels $\mu g/m^3$
Treat area	295 - 911	270 - 570
Manifold area	260 - 1040	230 - 370
Repair area	600 - 1000	227
Heat sink	600 - 800	260
Salvage drain	400 - 800	
Bldg. 10 Inst. Lab. 2nd floor	644	
Bldg. 10 office area	260	
Carousel #1		365 - 2000
Carousel #2		170 - 675
CEB Crimp station		1000 - 1500
Washer		1000 - 1500
Refinery		500

ATTACHMENT 3
6-23

(10)

NIOSH PERSONAL* AND AREA** AIR SAMPLE SURVEY April 1977

Area in Plant/Type of Job	Air Levels $\mu g/m^3$
Treat Area*	55 - 264
Soldering*	400 - 600
Moveman Sealing*	300 - 400
Moveman Testing*	170 - 180
Final Assembly**	100 - 150
Maintenance*	150
Recovery and Repair*	281 - 316
Testing, Reworking*	30 - 52
Rework Solder*	173 - 183
Salvage*	30 - 642
Rework Packer*	130 - 300
EMF Operator*	100 - 170
Winding**	3 - 54
Assembly, Shipping*	16 - 100
Storage**	19 - 94
Testing and Painting**	30 - 52
Can and Cover Manufacturer**	25 - 67

Deaths[1] in 2,984 Hourly Males,[2] 2,544 Hourly Females,[3] 1,078 Salaried Males[4] and 469 Salaried Females[5]

	HOURLY				SALARY			
Cause of Death	Males Obs/Exp	SMR (95% C.I.)	Females Obs/Exp	SMR (95% C.I.)	Males Obs/Exp	SMR (95% C.I.)	Females Obs/Exp	SMR (95% C.I.)
All Causes	586 / 699	83.8** (77-91)	380 / 420	90.4* (82-100)	177 / 328	53.9** (46-62)	52 / 75	69.0** (52-91)
All Cancers	128 / 158	81.1* (68-97)	150 / 136	109 (93-129)	56 / 81	69.1** (52-90)	19 / 25	75 (45-118)
Cancer of Tongue	1 / 0.9	103 (3-576)	2 / 0.4	483 (59-1745)	0 / 0.1	—	1 / 0.1	1346 (34-7498)
Other Cancer of Buccal Cavity	2 / 1.1	178 (22-642)	2 / 0.5	365 (44-1317)	0 / 0.5	—	1 / 0.1	1021 (26-5690)
Cancer of Pharynx	4 / 2.0	199 (54-509)	2 / 0.7	253 (31-915)	0 / 1.0	—	0 / 0.1	—
Cancer of Esophagus	5 / 3.8	131 (42-304)	1 / 1.1	87 (2-482)	1 / 2.0	49 (1-272)	0 / 0.2	—
Cancer of Stomach	4 / 5.9	68 (18-173)	4 / 3.0	132 (36-339)	1 / 2.7	36 (0.9-200)	0 / 0.5	—
Cancer of Intestine	8 / 14.0	57 (25-112)	20 / 12.7	157 (96-242)	7 / 7.1	98 (40-203)	1 / 2.2	44 (1-247)
Cancer of Rectum	3 / 3.4	87 (18-255)	4 / 2.2	169 (46-434)	3 / 1.6	185 (38-540)	0 / 0.4	—
Cancer of Biliary Passages & Liver	2 / 2.5	80 (10-289)	2 / 2.2	89 (11-321)	1 / 1.2	79 (2-439)	0 / 0.3	—
Cancer of Pancreas	9 / 7.8	115 (53-219)	7 / 5.9	117 (47-241)	6 / 3.9	150 (55-327)	0 / 1.1	—
Cancer of Larynx	3 / 2.0	147 (30-428)	1 / 0.4	215 (5-1198)	0 / 1.0	—	0 / 0.1	—

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	HOURLY				SALARY			
Cause of Death	Males Obs/Exp	SMR (95% C.I.)	Females Obs/Exp	SMR (95% C.I.)	Males Obs/Exp	SMR (95% C.I.)	Females Obs/Exp	SMR (95% C.I.)
Cancer of Trachea, Bronchus & Lung	42 / 54.5	77 (56-104)	32 / 25.2	127 (87-179)	12 / 29.6	40.5**(21-71)	5 / 4.7	104 (34-244)
Cancer of Breast	--	---	25 / 30	82 (53-121)	--	---	6 / 5.7	104 (38-226)
Cancer of Cervix Uteri	--	---	6 / 4.7	128 (47-277)	--	---	1 / 0.9	112 (3-622)
Cancer of Other Parts of the Uterus	--	---	5 / 3.8	131 (43-306)	--	---	0 / 0.6	---
Cancer of Ovary, Fallopian Tube, Broad Ligament	--	---	8 / 9.3	85 (37-168)	--	---	2 / 1.7	115 (14-415)
Cancer of Prostate	12 / 10.9	110 (57-192)	--	---	3 / 5.3	56 (5-136)	--	---
Cancer of Kidney	3 / 4	75 (15-219)	2 / 2.1	94 (11-341)	0 / 2.1	---	--	---
Cancer of Bladder & Other Urinary Organs	3 / 3.8	77 (16-226)	2 / 1.3	151 (18-545)	1 / 1.8	54 (1-300)	0 / 0.2	---
Cancer of Skin	5 / 3.8	130 (42-303)	3 / 2.0	144 (30-421)	4 / 1.9	210 (57-538)	0 / 0.4	---
Cancer of Brain and Nervous System	2 / 5.1	39 (5-140)	2 / 3.7	53 (6-192)	4 / 2.5	156 (42-398)	0 / 0.7	---
Lymphosarcoma	2 / 2.1	92 (11-331)	1 / 1.5	65 (2-364)	0 / 1.0	---	0 / 0.2	---
Leukemia & Aleukemia	4 / 6.3	63 (17-162)	4 / 4.3	93 (25-238)	5 / 3.1	166 (54-387)	0 / 0.8	---
Other Lymphatic & Hematopoietic Tissue	5 / 5.7	87 (28-202)	5 / 4.7	105 (34-245)	4 / 3.0	131 (36-336)	0 / 0.8	---

ATTACHMENT 3
7-23

10.9364

	HOURLY				SALARY			
Cause of Death	Males Obs/Exp	SMR (95% C.I.)	Females Obs/Exp	SMR (95% C.I.)	Males Obs/Exp	SMR (95% C.I.)	Females Obs/Exp	SMR (95% C.I.)
Diabetes	4 / 10.5	38* (10-97)	9 / 10.3	87 (40-165)	5 / 5.1	97 (32-226)	0 / 1.8	—
Ischemic Heart Disease	182 / 205	89 (76-103)	71 / 87	81 (64-103)	44 / 97.5	45**(33-61)	8 / 14.3	56 (24-110)
Cerebrovascular Disease	26 / 34.9	74 (49-109)	27 / 30	89 (59-130)	3 / 15.2	20**(4-58)	6 / 5	120 (44-260)
Diseases of Arteries, Veins, Pulmonary Circulation	19 / 17.0	112 (67-174)	10 / 11	95 (46-175)	4 / 8.0	50 (14-128)	1 / 1.8	56 (1-310)
Emphysema	7 / 7.3	96 (39-198)	3 / 3.1	97 (20-283)	1 / 3.3	30 (0.8-168)	0 / 0.5	—
Pneumoconioses & other Respiratory Diseases	18 / 19.6	92 (54-145)	10 / 11.9	84 (40-154)	3 / 10.2	29* (6-86)	0 / 2.1	—
Cirrhosis of the Liver	13 / 17.9	72 (39-124)	6 / 9.2	65 (24-142)	3 / 9.1	33* (7-96)	1 / 1.7	57 (1-318)
Transportation Accidents	29 / 34.7	84 (56-120)	14 / 9.1	153 (84-257)	3 / 12.6	24** (5-69)	3 / 1.9	156 (32-455)
Other Accidents	10 / 14.4	69 (33-127)	5 / 3.1	158 (51-369)	3 / 5.9	51 (11-148)	1 / 0.6	159 (4-886)
Suicide	14 / 21.3	66 (36-110)	3 / 2	44 (9-128)	2 / 8.6	23* (3-84)	2 / 1.4	140 (17-507)
Homicide	3 / 8.4	36 (7-104)	2 / 2	96 (12-345)	0 / 3.0	—	0 / 0.4	—

* Significant at p <0.05; ** significant at P <0.01

[1] Expected numbers based on age-sex-race and time specific United States rates coded according to the rules of the International Classification of Diseases coding in force at the time of death.

[2] 85, 991 person-years of observation

[3] 75, 674 person-years of observation

[4] 34, 755 person-years of observation

[5] 16, 358 person-years of observation

(14).

Observed and Expected[1] Deaths in 1268 Ever Highly Exposed, 584 Only Low Exposed and 2037 Ever Undefinably Exposed Hourly Male Workers

Cause of Death[4]	High[1] Obs/Exp	SMR[5] (95% C.I.)	Low[2] Obs/Exp	SMR (95% C.I.)	Undefinable[3] Obs/Exp	SMR (95% C.I.)
All Causes	244 / 299	82** (72-93)	135 / 144	94 (79-111)	389/469	82.9** (75-92)
All Cancers	52 / 68	77 (57-100.5)	25 / 32	79 (51-116)	93/107	86.7 (70-106)
Cancer of Tongue	1 / 0.4	241 (6-1345)	0 / 0.2	—	0 / 0.66	—
Other Cancer of Buccal Cavity	1 / 0.5	207 (5-1151)	0 / 0.2	—	2 / 0.77	261 (32-944)
Cancer of the Pharynx	1 / 0.9	115 (3-642)	3 / 0.4	742* (153-2168)	1 / 1.4	73 (2-404)
Cancer of Esophagus	2 / 1.7	121 (15-435)	2 / 0.7	259 (31-936)	2 / 2.6	76 (9-276)
Cancer of Stomach	2 / 2.5	80 (10-290)	0 / 1.3	—	4 / 3.9	102 (28-261)
Cancer of Intestine	1 / 6	17* (0.4-93)	2 / 2.8	70 (9-252)	6 / 9.4	63 (23-138)
Cancer of Rectum	1 / 1.4	69 (2-386)	0 / 0.7	—	3 / 2.3	131 (27-383)
Cancer o the Biliary Passages and Liver	2 / 1	187 (23-676)	0 / 0.5	—	1/ 1.7	59 (2-329)
Cancer of Liver not Specified	1 / 0.4	228 (6-1268)	0 / 0.2	—	1 / 0.7	143 (4-799)
Cancer of Pancreas	3 / 3.3	90(19-263)	3 / 1.6	188 (39-549)	5 / 5.2	95 (31-221)

10.9366

ATTACHMENT 3
8-23

Cause of Death	High[1] Obs/Exp	SMR[5] (95% C.I.)	Low[2] Obs/Exp	SMR (95% C.I.)	Undefinable[3] Obs/Exp	SMR (95% C.I.)
Cancer of the Peritoneum	0 / 0.3	—	0 / 0.1	—	0 / 0.5	—
Cancer of the Larynx	3 / 0.9	342 (71-999)	0 / 0.4	—	2 / 1.4	143 (17-517)
Cancer of the Trachea, Bronchus, & Lung	16 / 24	68 (39-110)	5 / 11	46 (15-108)	35 / 37	93 (65-130)
Other Respiratory Cancer	1 / 0.3	353 (9-1967)	0 / 0.1	—	0 / 0.45	—
Cancer of the Prostrate	3 / 4.7	65 (13-190)	2 / 2.2	88 (11-316)	10 / 7.1	139 (67-256)
Other Male Genital Cancers	0 / 0.4	—	0 / 0.1	—	0 / 0.8	—
Cancer of the Kidney	2 / 1.7	115 (14-416)	1 / 0.8	125 (3-695)	2 / 2.7	73 (9-264)
Bladder & Other Urinary Cancers	3 / 1.6	184 (38-538)	0 / 0.8	—	3 / 2.6	117 (24-341)
Cancer of the Skin	4 / 1.7	236 (64-605)	0 / 0.7	—	4 / 2.7	152 (41-388)
Cancer of the Brain and Nervous System	1 / 2.3	44 (1-247)	1 / 1	100 (3-557)	1 / 3.6	28 (0.7-156)
Cancer of Other & Unspecified Sites	1 / 4.5	22 (0.6-124)	2 / 2	97 (12-350)	4 / 7.1	57 (15-145)
Lymphosarcoma & Reticulosarcoma	0 / 0.9	—	1 / 0.5	221 (6-1230)	1 / 1.5	68 (2-378)
Hodgkin's Disease	0 / 0.7	—	0 / 0.3	—	0 / 1.1	—
Leukemia & Aleukemia	0 / 2.7	—	2 / 1.3	157 (19-567)	2 / 4.3	47 (6-168)
Other Lymphatic & Hematopoietic Tumors	3 / 2.6	119 (25-349)	1 / 1.1	90 (2-500)	3 / 3.9	76 (16-221)
Ischemic Disease	71 / 87	82 (64-103)	48 / 44	110 (81-146)	116 / 136	85 (70-102)
Chronic Disease of Endocardium	3 / 0.9	326 (67-952)	0 / 0.4	—	1 / 1.45	69 (2-386)

16

Cause of Death	High[1] Obs/Exp	SMR[5] (95% C.I.)	Low[2] Obs/Exp	SMR (95% C.I.)	Undefinable[3] Obs/Exp	SMR (95% C.I.)
Hypertension with Heart Disease	1 / 2.4	42 (1-232)	0 / 1.3	—	5/3.8	134 (43-312)
Other Diseases of the Heart	16 / 15	105 (60-171)	7 / 6.8	104 (42-215)	25/23	107 (69-157)
Hypertension without Heart Disease	1 / 0.8	125 (3-698)	0 / 0.4	—	2/1.2	163 (20-589)
Cerebrovascular Disease	12 / 14.7	82 (42-143)	6 / 7.9	76 (28-166)	14/22	62 (34-105)
Diseases of Arteries, V eins, Pulmonary Circulation	11/ 7.2	152 (76-272)	3 / 3.7	82 (17-241)	15/11	134 (75-221)
Transportation Accidents	12 / 15	79 (41-138)	11 / 6.6	167 (83-299)	16/23	67 (38-109)
Accidental Poisonings	2 / 1.4	146 (18-527)	0 / 0.6	—	3/3.2	93 (19-272)
Other Accidents	4 / 6.3	63 (17-162)	3 / 2.8	108 (22-315)	6/9.9	61 (22-132)
Medical Complications	2 / 0.4	532 (64-1921)	0 / 0.1	—	1/0.6	169 (4-941)

*Significant at p <0.05; **significant at p <0.01
[1] 37,739 Person-years of exposure
[2] 16,249 Person-years of exposure
[3] 59,226 Person-years of exposure
[4] Expected numbers based on age-sex-race and time specific United States rates coded according to the rules of the International Classification of Diseases coding in force at the time of death.
[5] SMR equals deaths observed divided by deaths expected based on United States rates and multiplied by 100. Values in parentheses are the 95 percent confidence intervals.

10.9368

ATTACHMENT 3
9-23

PCBs: Environmental Considerations

Jim Cogliano, Ph.D.
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National Center for Environmental Assessment
Washington, D.C.

10.9369

ATTACHMENT 3
10-23

PCBs: Environmental considerations

- PCBs in the environment
- PCBs in living organisms
- Health effects of concern

PCBs

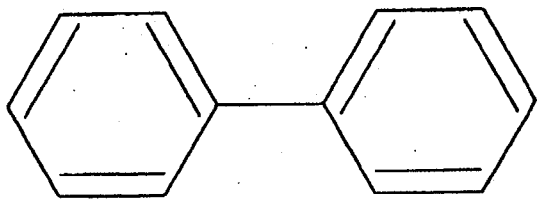
- Chlorine substitution
- Congeners
- Homologues
- Aroclors

ATTACHMENT 3
11-23

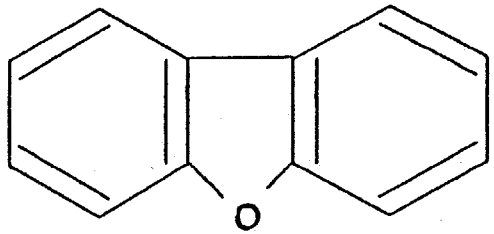
10.9371

Similarity of PCBs to dibenzofurans and dioxins

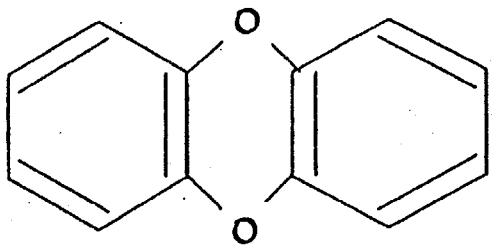
PCBs



Polychlorinated dibenzofurans



Polychlorinated dibenzo-*p*-dioxins



Typical composition of some Aroclor mixtures

	Aroclor 1016	1242	1248	1254	1260
Mono-CBs (%wt)	2	1	—	—	—
Di-CBs	19	13	1	—	—
Tri-CBs	57	45	21	1	—
Tetra-CBs	22	31	49	15	—
Penta-CBs	—	10	27	53	12
Hexa-CBs	—	—	2	26	42
Hepta-CBs	—	—	—	4	38
Octa-CBs	—	—	—	—	7
Nona-CBs	—	—	—	—	1
Deca-CB	—	—	—	—	—
Chlorine content (%)	41	42	48	54	60
Production, 1957–1977 (%)	13	52	7	16	11

"—" denotes less than 1%.
Sources: Adapted from U.S. EPA (1996), Cogliano (1998).

10.9373

ATTACHMENT 3
12-23

Environmental fate is related to chlorine substitution

1 ..... Chlorine content 10

Higher ..... Volatility ..... Low

Higher ..... Solubility in water ..... Low

Low ..... Adsorption to soil and sediment ..... High

Low ..... Persistence in the environment ..... High

PCBs partition in the environment

Air —	Higher proportion of lower-chlorinated congeners
Water —	Higher proportion of lower-chlorinated congeners
Soil —	Higher proportion of higher-chlorinated congeners
Sediment —	Higher proportion of higher-chlorinated congeners

Metabolic fate is related to chlorine substitution

Oxidative metabolism is facilitated by the absence of chlorines in adjacent positions

PCBs bioaccumulate in the environment

- Each link in the food chain passes on congeners most difficult to eliminate
- PCB composition can be significantly altered

Which exposure pathways are of greatest concern?

- Bioaccumulated mixtures
  - Fish
  - Birds that eat fish
- Contaminated soil and sediment

PCBs and cancer

Mayes (1998) tested Aroclors 1016, 1242, 1254, and 1260 in rats

- All cause significant increases in liver cancer
- Some Aroclors increased thyroid cancer in males
- Potency differs for these mixtures

These mixtures contain overlapping groups of congeners that, together, span the range of congeners most often found in environmental mixtures

Conclusions

- All PCB mixtures can pose a risk of cancer
- There is a basis for distinguishing the cancer potential of different environmental mixtures

PCBs and cancer

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Conclusions

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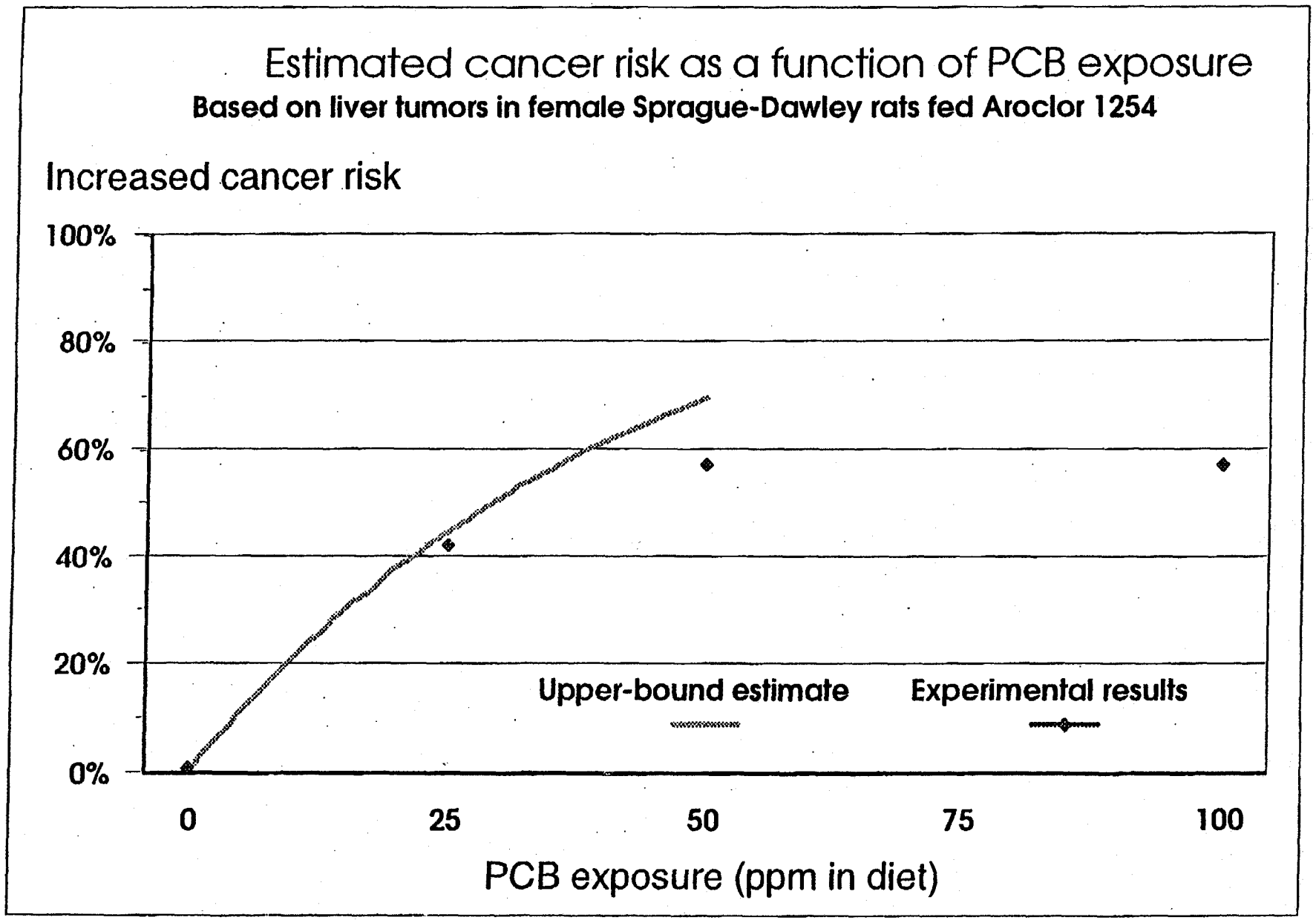
Liver tumor incidences from the 1996 rat study

Mixture	Dose	Females	Males
Aroclor 1260	Control	** 1/85 (1%)	** 7/98 (7%)
	25 ppm	10/49 (20%)	3/50 (6%)
	50 ppm	11/45 (24%)	6/49 (12%)
	100 ppm	24/50 (48%)	10/49 (20%)
Aroclor 1254	Control	** 1/85 (1%)	7/98 (7%)
	25 ppm	19/45 (42%)	4/48 (8%)
	50 ppm	28/49 (57%)	4/49 (8%)
	100 ppm	28/49 (57%)	6/47 (13%)
Aroclor 1242	Control	** 1/85 (1%)	7/98 (7%)
	50 ppm	11/49 (24%)	1/50 (2%)
	100 ppm	15/45 (33%)	4/46 (9%)
Aroclor 1016	Control	** 1/85 (1%)	7/98 (7%)
	50 ppm	1/48 (2%)	2/48 (4%)
	100 ppm	6/45 (13%)	2/50 (4%)
	200 ppm	5/50 (10%)	4/49 (8%)

**Statistically significant ($p<0.05$) by Cochran-Armitage trend test.
Hepatocellular adenomas, carcinomas, cholangiomas, or cholangiocarcinomas in rats alive when the first tumor was observed.
One control group supported all experiments.
Source: Brunner (*1996*), reported by U.S. EPA (*1996*); Mayes (*1998*).

10.9381

Attachment 2
16-23


Estimated cancer risk as a function of PCB exposure
Based on liver tumors in female Sprague-Dawley rats fed Aroclor 1254
Increased cancer risk
100%
80%
60%
40%
20%
0%
Upper-bound estimate
Experimental results
0
25
50
75
100
PCB exposure (ppm in diet)

Three tiers of environmental PCBs

HIGHEST RISK AND PERSISTENCE

- Food chain exposure
- Sediment or soil ingestion
- Dust or aerosol inhalation
- Early-life exposure (all pathways and mixtures)

LOWER RISK AND PERSISTENCE

- Ingestion of water-soluble congeners
- Inhalation of evaporated congeners
- Dermal exposure, if no absorption factor has been applied

LOWEST RISK AND PERSISTENCE

- Congener or homologue analyses verify that congeners with more than 4 chlorines comprise less than 1/2% of total PCBs

Less-than-lifetime exposure to the more persistent mixtures may pose disproportionately high risks

Mixture	Dose	Less-than-lifetime exposure	Lifetime exposure
Aroclor 1260	Control	** 1/85 (1%)	** 1/85 (1%)
	25 ppm	4/24 (17%)	10/49 (20%)
	50 ppm	3/24 (12%)	11/45 (24%)
	100 ppm	17/24 (71%)	24/50 (48%)
Aroclor 1254	Control	** 1/85 (1%)	** 1/85 (1%)
	25 ppm	5/24 (21%)	19/45 (42%)
	50 ppm	7/24 (29%)	28/49 (57%)
	100 ppm	6/24 (25%)	28/49 (57%)
Aroclor 1242	Control	** 1/85 (1%)	** 1/85 (1%)
	50 ppm	3/24 (12%)	11/49 (22%)
	100 ppm	6/24 (25%)	15/45 (33%)
Aroclor 1016	Control	1/85 (1%)	** 1/85 (1%)
	50 ppm	0/24 (0%)	1/48 (2%)
	100 ppm	0/24 (0%)	6/45 (13%)
	200 ppm	0/24 (0%)	5/50 (10%)

**Statistically significant ($p<0.05$) by Cochran-Armitage trend test.
Less-than-lifetime experiment involved rats dosed for 52 weeks and killed after 104 weeks.
Source: Brunner (*1996*), reported by U.S. EPA (*1006*).

Bioaccumulated PCBs may be more toxic and more persistent than the Aroclors

- In mink fed Great Lakes fish, reproductive toxicity and liver toxicity were greater than for other mink fed equivalent amounts of Aroclor 1254
- In monkeys fed a mixture representative of PCBs found in human milk, long-term behavioral impairments have been found
- In people eating Great Lakes fish, the rate of decline in serum PCB levels was much smaller than what has been reported for people exposed to Aroclors in the workplace

Noncancer effects of PCBs

PCBs have significant adverse health effects other than cancer, including

- Learning deficits
- Neurological effects
- Immune dysfunction
- Thyroid effects
- Hormonal effects

Recent studies raise new concerns about environmental exposure

Study of children whose mothers ate L. Michigan fish

3 days	Motor immaturity, ⇓ ability to quiet, ⇑ startle, ⇓ reflexes
7 months	⇓ short-term memory
4 years	⇓ verbal scale, ⇓ memory scale, ⇓ activity, ⇓ short-term memory, ⇓ visual discrimination
11 years	⇓ full-scale and verbal IQ, ⇓ work and reading comprehension, ⇓ memory and attention

Highest PCB group . . .

had average IQ 6 points below average

3x more likely to have low IQ

2x more likely to be 2 years behind in reading ability

Study of children whose mothers ate L. Ontario fish

Infancy	Abnormal reflexes, ⇑ startle, ⇑ tremor
12 months	⇓ habituation
36 months	⇓ general cognitive index

Study of PCBs from food (N. Carolina)

Early infancy	⇓ reflexes, ⇓ activity
6–12 months	⇓ psychomotor development
24 months	⇓ psychomotor development
3, 4, 5 years	No effect on motor or memory scales

ATTACHMENT 3
20 - 23

Study of PCBs from food (Netherlands)

10, 21 days	⇓ reflexes, hypotonicity
3 months	⇓ psychomotor score, immunological changes
7 months	⇓ psychomotor score
18 months	⇓ psychomotor development, immunological changes
42 months	⇓ general cognitive scale, ⇓ high-level play, ⇑ non-play time, ⇑ reaction time, ⇑ withdrawn/depressed behavior, ⇑ prevalence of chicken pox, ⇓ antibodies to measles

These effects were seen at 3 ppb in blood serum

Studies of PCBs in monkeys

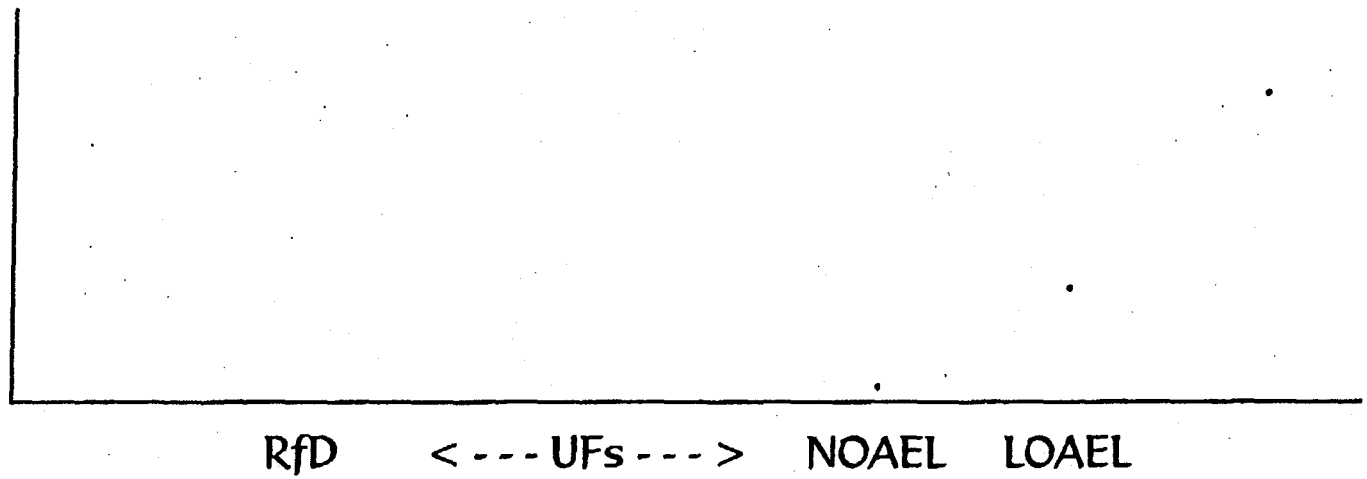
Independent studies in animals show that PCBs alone can cause effects analogous to those seen in the human studies, including

- ⇓ learning
- ⇓ memory
- ⇓ ability to adapt
- ⇓ ability to organize behavior
- ⇓ attention

These studies increase our confidence that the effects seen in the human studies can be attributed to PCBs

ATTACHMENT 3
2-1-23

Noncancer reference dose



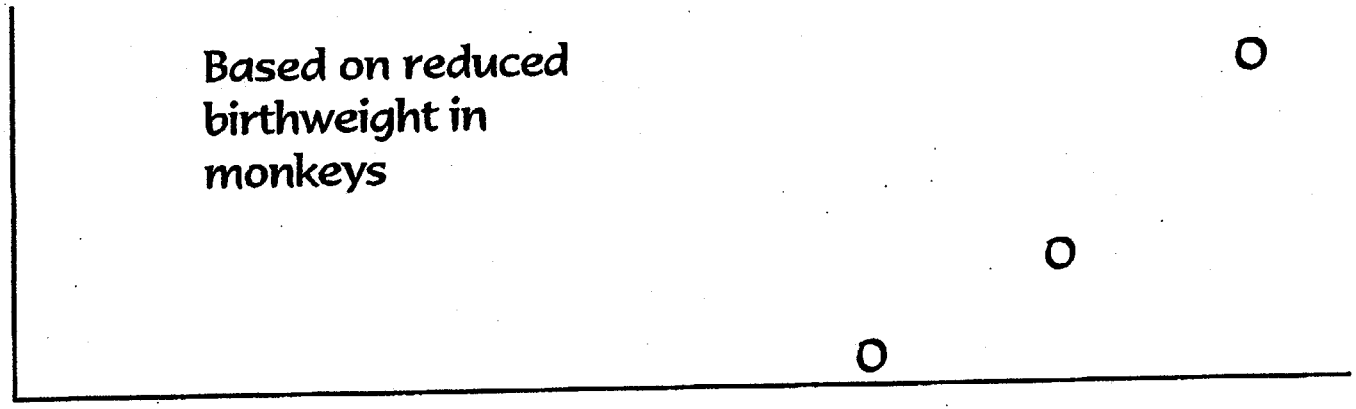
Human variability
Animal-to-human uncertainty
LOAEL-to-NOAEL uncertainty
Subchronic-to-chronic uncertainty
Database limitations
Modifying factor

ATTACHMENT 3
22-23

for Aroclor 1254

Based on decreased antibody (IgG and IgM) response to sheep erythrocytes in monkeys	0	0	—	5 ug/kg-d	0.02 (300)
				10	
				3	
				3	
				3	
				—	
				—	

for Aroclor 1016



0.07 (100) 7 ug/kg-d 28 ug/kg-d

3
3
1
3
3

10.9394

Summary

- PCB mixtures are altered in the environment — in some cases increasing the mixture's persistence and toxicity
- Principal exposures of concern are bioaccumulated PCBs and PCBs attached to soils or sediments
- Evidence is strong that environmental PCBs pose a risk of cancer
- Evidence is mounting that noncancer effects, especially learning deficits and neurological effects, have occurred from environmental PCB exposure

WHAT CAN YOU DO?

- Pay attention to fish advisories

10.9395

ATTACHMENT 3
23-23

Summary

- PCB mixtures are altered in the environment — in some cases increasing the mixture's persistence and toxicity
- Principal exposures of concern are bioaccumulated PCBs and PCBs attached to soils or sediments
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WHAT CAN YOU DO?

- Pay attention to fish advisories

ATTACHMENT 4

Hudson River PCBs Site Reassessment RI/FS
June 10, 1999 (Updated March 8, 2000)

Milestone	Completed	To Public
PHASE 1 Report	✔	Aug 1991
PHASE 2 Field Sampling Program - 1992 to 1994	✔	N/A
Database Report (DBR)	✔	Nov 1995
Preliminary Model Calibration Report (PMCR)	✔	Oct 1996
Data Evaluation & Interpretation Report (DEIR)	✔	Feb 1997
Low Resolution Sediment Coring Report (LRC)	✔	Jul 1998
Human Health Risk Assessment Scope of Work	✔	Jul 1998
CD-ROM Database Reissue	✔	Jul 1998
Peer Review 1 - Modeling Approach - Begins	✔	Jul 1998
Peer Review 1 Meeting	✔	Sept 1998
Ecological Risk Assessment Scope of Work	✔	Sept 1998
DBR, PMCR, DEIR Responsiveness Summary	✔	Dec 1998
Peer Review 2 - DEIR & LRC - Begins	✔	Jan 1999
LRC Responsiveness Summary	✔	Feb 1999
Peer Review 2 Meeting	✔	Mar 1999
Human Health Risk Assmt SOW Responsiveness Summary	✔	Apr 1999
Ecological Risk Assmt SOW Responsiveness Summary	✔	Apr 1999
Baseline Modeling Report (BMR)	✔	May 1999
Human Health Risk Assessment (HHRA) Upper Hudson	✔	Aug 1999
Ecological Risk Assessment (ERA)	✔	Aug 1999
Addendum HHRA - Mid-Hudson	✔	Dec 1999
Addendum ERA - Future Risks for Lower Hudson	✔	Dec 1999
Peer Review 3 - BMR - Begins	✔	Jan 2000
Revised BMR	✔	Jan 2000
BMR Responsiveness Summary	✔	Feb 2000
Response to Peer Review 1 Comments	✔	Feb 2000
Peer Review 3 Meeting		Mar 2000
HHRA and ERA Responsiveness Summaries		Mar 2000
Peer Review 4 - HHRA & ERA - Begins		Mar 2000
Peer Review 4 Meeting		May 2000
PHASE 3 Feasibility Study Scope of Work (FS SOW)	✔	Sept 1998
FS SOW Responsiveness Summary	✔	Jun 1999
FS Report		Dec 2000
PROPOSED PLAN		Dec 2000
RECORD OF DECISION (including Responsiveness Summary)		Jun 2001