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HUMAN HEALTH EFFECTS

OF

ELECTRICAL GRADE PCB's

J.F. Brown, Jr., Ph.D.
Manager - Life Sciences Branch
Corporate Research and Development Center
Schenectady, NY

J.T. Coe
Staff Executive
Environmental Quality & Safety
Corporate Health & Safety Operation
Fairfield, CT

H.D. Pocock, Jr., M.D.
Associate Company Medical Director
Corporate Health & Safety Operation
Fairfield, CT

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Executive Summary

Definition of any actual human health hazard posed by the polychlorinated biphenyls (PCB's) continues to be an important issue, since their potential for human exposure cannot be eliminated completely.

PCB's were widely used in the United States for nearly 50 years chiefly as dielectric fluids and plasticizers. They are now controlled by law because they are a persistent environmental contaminant, and because they are popularly believed to be very toxic. Although PCB use in new products has been banned, large amounts are still present in electrical equipment that will require servicing and disposal; PCB's are also present in numerous environments that may, or may not, merit containment or restorative actions; and small, but measureable levels are present in some fish available for consumption.

The available animal data show that PCB's, like many other substances, can produce toxic effects in animals under the conditions of toxicity testing. However, the doses required to produce such effects are usually large with respect to body weight. Toxicity in humans is not so readily demonstrated.

In the Japanese Yusho disease episode, which followed eating rice oil contaminated by a PCB fluid leakage, the key toxic agent is now thought to have been a PCDF contaminant, present at exceptionally high levels in the thermally decomposed heat exchange fluid. The presence of PCDF impurities also appears indicated in some incidents when PCB's were found to cause human chloracne.

The conditions required to convert PCB's to PCDF's may be encountered in high temperature heat exchangers or bake ovens, but not ordinarily in the manufacture or use of electrical equipment (the major past use of PCB's), nor that of plasticizers, carbon paper, or printing inks (the main secondary uses). Available data indicates toxicologically insignificant (0-2 ppm) levels of PCDF's in U.S.-made electrical grade PCB's as manufactured, though additional research is needed to confirm the level in certain environments.

The most extensive data on PCB human health effects comes from an increasing number of occupational health studies of capacitor workers. Thousands of such individuals received heavy exposure to PCB's of Aroclor types 1254,1242, and 1016 over many years. While additional research may be needed to rigorously exclude all possibilities of subtle or infrequent health damage in such individuals, all common indicators, such as clinical examination findings, health complaints, or death rates due to cardiovascular disease, cancer, or nervous system disease indicate lack of serious health impairment. A recent study by NIOSH, which documents the long-term experience of a large group of such workers, found no excess of total mortality nor of overall cancer mortality.

In view of the paucity of evidence for health damage in people who had heavy PCB exposure in the past (daily skin contact and air inhalation levels from 100-1000 ug/m^3), there seems to be little basis for concern over the effects of occasional exposure to environments containing electrical grade PCB's at the levels which may typically be encountered today (e.g. 0.1-10 ug/m^3 in air or occasional skin contact).

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I Introduction

At present, Company managers, employees, physicians, and public officials concerned with appropriate handling of situations involving human exposure to PCB's face a contradictory array of information on their possible health hazards.

On the one hand, the U.S. press and environmental literature (e.g., ref. 1), increasingly portray PCB's as very toxic materials, and situations posing human exposure at any level as hazardous and alarming. This sentiment is also reflected in national bans on new PCB use and regulations for PCB control, transport, storage, and disposal in the U.S.

On the other hand, European countries have apparently made a different assessment, because new PCB usage in closed electrical equipment, with attendant occupational exposures during manufacture and service, continues as before. Moreover, an increasing number of medical studies of human populations which were heavily-exposed to PCB's continue to reveal absence of significant human health problems (2,3,4,5,6,7,8,9,10). Public statements by knowledgeable health authorities and scientists are sometimes so hedged as to make the authors' positions unclear.

In order to provide perspective for General Electric managers and physicians, to help identify situations of human exposure to PCB's which actually might present cause for concern, and to outline some future needed research, the writers have undertaken this paper. It draws on extensive GE experience in plants manufacturing PCB-filled capacitors, as well as on review and analysis of the scientific literature.

The PCB literature is voluminous, comprising several thousand original articles. The present paper will not attempt to survey all aspects of this information, but will focus on those portions most relevant to human health effects evaluation. Literature citations have been included in the bibliography, as have condensations of much of the health-related material in the form of conference reports (11,12,13,14,15) and several review articles (Kimbrough (16), Fishbein (17), NIOSH Criteria Document (18), and the IARC Monographs (19,37).

Many quantitative observations are included in this paper because these are critically important to any health effects perspective. The fact that PCB's are detectable does not necessarily mean that they are toxicologically significant.

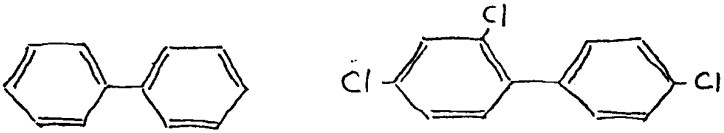
The paper's content is organized as follows:

Abbreviations which may be unfamiliar to some readers include:

ppm	parts per million
ppb	parts per billion
kg.	kilogram; 1000 grams or 2.2 pounds
gm.	gram (s) or 1/28 oz.
mg.	milligram; one thousandths of a gram
ug/m^3	micrograms (millionths of a gram) per cubic meter
LD_{50}	an acute toxicity measurement meaning lethal dose for 50% of the test species

II PCB Composition and Terminology

In chemical terminology "phenyl" denotes a ring structure of six carbon atoms attached to something else; "biphenyl" results when two such rings are attached to each other; and a "polychlorinated biphenyl" (PCB) is any molecule having chlorine atoms attached to the carbon atoms of a biphenyl nucleus. Biphenyl and a representative trichlorobiphenyl (PCB) are illustrated below:



Biphenyl 2,4,4'-Trichlorobiphenyl (PCB)

Usually "PCB" is used as a generic term, not as the name of any one individual type of molecule.

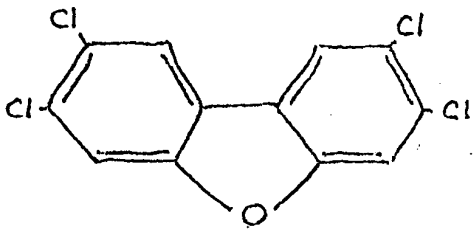
There are 209 theoretically-possible PCB molecules, differing in the numbers (homologs) and positions (isomers) of the attached chlorines; of these 209 species, about half have been actually identified among the complex PCB mixtures that constituted the commercial PCB products. These products ranged from light oily fluids (di-, tri-, and tetra-chlorobiphenyls) to heavy, honey-like oils (penta-chlorobiphenyls) to greases and waxes (more highly chlorinated).

The manufacturers of the PCB's sold them under brand names, e.g., "Aroclor" (Monsanto, USA); "Phenoclor" (Prodelec, S.A. France); "Clophen" (Farbenfabriken Bayer AG, Germany); or "Kanechlor" (Kanegafuchi Chemical Industrial Co., Ltd., Japan). They also assigned product numbers that usually reflected either the average degree of chlorination, or, what is equivalent, the weight-percent chlorine in the mixture. Thus, the American Aroclor 1242, its French equivalent, Phenoclor DP3, and its Japanese equivalent, Kanechlor 300, all contained 42% chlorine, or 3 chlorine atoms per biphenyl on the average. Likewise, Aroclor 1254 and its German equivalent, Clophen A50, contained 54% chlorine, or 5 chlorine atoms per biphenyl, on the average.

The major electrical users of PCB's subjected these commercial products to further purification to insure electrical performance, blended them with stabilizers and diluents, and then put them into use under their own brand names and product numbers. General Electric's generic name was "Pyranol"; Westinghouse's was "Inerteen". "Askarel" is an industry term for a PCB or other fire-resistant dielectric fluid.

In short, the material that we now refer to as a "PCB" could have originally had a variety of brand and product names and it was a complex mixture of chemicals. It always contained several dozen individual PCB isomers and homologs clustered around some average degree of chlorination, not infrequently some trichlorobenzenes as diluents, often about 0.5% of an aliphatic epoxide as a stablizer, and parts-per-million levels of trace impurities, such as polychlorinated methylbiphenyls, polychlorinated terphenyls, polychlorinated naphthalenes, and sometimes polychlorinated dibenzofurans (PCDF's).

Returning to chemical terminology, "furan" denotes a structural arrangement comprising a ring of four carbon atoms and one oxygen; "benzo" an arrangement of four carbon atoms fused onto an adjacent pair in some other structure so as to form a six-membered ring; and hence "dibenzofuran" a tricyclic arrangement of two benzene rings and one furan ring fused together. This arrangement can result if an oxygen atom is added to a biphenyl system. If chlorine atoms are attached to this nucleus, then a "polychlorinated dibenzofuran", or PCDF, results.

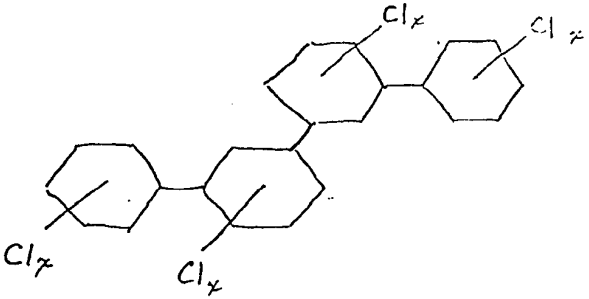


2,3,7,8 tetrachlorodibenzofuran (PCDF)

Trace levels of PCDF (or other species giving PCDF color tests) have been detected in laboratory samples of U.S. PCB's, including specimens of individually synthesized isomers as well as those of commercial mixtures (20,21).

We know of two ways that PCDF's get into commercial PCB's; first, contamination may occur during manufacture, possibly as a result of oxygen in the benzene used to make the original biphenyl. Such contamination may have been present in French and Japanese PCB's, but apparently not significantly in American-made PCB after one episode in 1933. Second, the PCDFs may be formed as a result of high temperature oxidation (e.g., a week above 500°F, or a few seconds at 900°F). Such conditions are not expected in the normal manufacture or use of capacitors and transformers, but may occur in high temperature heat exchangers.

Another related molecular species are the polychlorinated quaterphenyls, (PCQ's) which contain four phenyl units and a variable number of chlorine atoms:



Polychlorinated quaterphenyl (PCQ)

III History of PCB Use

PCB materials have had a long history of use in the U.S. and throughout the world. Monsanto (and the Swann chemical predecessor plant) was the sole U.S. producer, manufacturing PCB's from 1929 to 1977, initially at Anniston, Alabama, and later at Sauget, Illinois.

PCB's are excellent non-flammable, high-boiling, thermally and chemically stable dielectrics and solvents, and these properties led to a wide range of industrial applications. In addition to serving as dielectric fluids in capacitors and transformers, PCB's were used as plasticizers, hydraulic fluids, heat exchange fluids, dust-settling agents, die-casting lubricants, and in paints. They were widely used in printing ink formulas and in carbonless reproduction paper, and they entered paperboard production. Transport of PCB's from these products into the environment, combined with their slow biodegradability and high bioaccumulation factors, resulted in their persistence in sediments under rivers and lakes, accumulations in fish, and entry, in trace quantities, into human food. As a result, PCB's are found (in trace quantities) in the blood and fatty tissue of almost all human populations.

Concern over such accumulations in the environment led Monsanto in 1970 to begin restricting PCB sales voluntarily to manufacturers of sealed electrical equipment (27). A phaseout of PCB use in the U.S. was mandated by Congress in the Toxic Substances Control Act of 1976, and specific regulations to achieve this purpose were completed by EPA in 1979. Although similar PCB phaseouts were instituted in Japan, Canada, and Sweden, other industrial nations (e.g., U.K., Germany, France, Italy, Spain and others) continue to permit new PCB use in closed electrical applications.

Estimates for the cumulative total of U.S. industrial uses (1930-1975) and their service status are noted below (22).

Table I

Millions of Pounds

Use	Industrial PCB Purchases	PCB's Currently in Service
Capacitors	630	450
Transformers	335	300
Plasticizer uses	115	n.a.(but small)
Hydraulics and Lubricants	80	↓
Carbonless copy paper	45	
Misc. industrial	28	
Heat transfer	20	↓
Total	1253	750

The same study made rough estimates of degradation and of other environmental distribution as follows:

Table II

Disposition	Millions of Pounds
Environmentally biodegraded	30
Incinerated	25
Landfills and dumps	290
In soil, water, air and sediment	150
Currently in electrical service	750
Other	8
Total	1253

General Electric manufactured capacitors containing PCB's at the Hudson Falls and Ft. Edward, New York plants from 1946 until 1977. Pyranol-filled transformers, which comprised a portion of the plants' transformer production, were manufactured at Pittsfield, MA from the 1930's until 1977, at Rome, GA from 1954 until 1977, and at Oakland, CA from 1938 to 1968. GE apparatus service shops have repaired Askarel-filled transformers for many years.

IV Health Effects of PCB and PCDF Mixtures

A. Observations on Test Animals

1. General Bioeffects PCB's resemble other fat-soluble chlorinated organic chemicals in biological uptake and internal transport. An animal can readily absorb PCB's through the lungs upon inhalation, through the intestines following ingestion, or through the skin after physical contact. Fish can absorb them through their gills and the bioconcentration factors can be large (10^3-10^5). Once inside the body they gradually distribute themselves equally among all fatty deposits present. Elimination processes are generally slow, especially for the more highly chlorinated homologs.

When increasing doses of PCB's are administered to animals the first observable effect is the induction of the microsomal enzymes variously known as "drug metabolizing" or "detoxification" enzymes, or as "mixed function" (i.e., chemically non-specific) oxidases. Such effects may also be induced by many other substances, and are generally regarded as physiological rather than pathological processes.

At high enough doses PCB's can produce many types of toxic effects and eventually death. The reported toxic effects include edema disease and teratogenesis in chickens; liver hypertrophy, fibrosis, and neoplasia in rodents; gastric and dermatological lesions in monkeys; and reproductive disfunction in rats and monkeys. An extensive literature on this subject is referenced in the bibliography (12-18). There can be substantial differences between different PCB products in chronic toxicity, with the higher homologs generally being the more toxic. There can also be large differences observed in toxicity between different specimens of the same type of PCB.

2. Tolerance Levels Early concerns over possible human health effects of chlorinated aromatic hydrocarbons (including PCB's) led to a series of long-term inhalation tests in animals at the Harvard School of Public Health in the 1930's (26) and confirmatory tests at the University of Cincinnati's Kettering Laboratory in the 1950's (27). Both test series showed cumulative, but reversible, effects on rat livers at high doses, and also identified threshold levels for such effects. From these levels were established the threshold limit values (TLV's) of 1000 ug/m^3 for PCB's with 42% chlorine or less, and 500 ug/m^3 for the more highly chlorinated PCB mixtures. These TLV's, used by industry during the remaining period of PCB use, were accepted as standards by the American Conference of Governmental Industrial Hygienists (ACGIH) in 1956 and by OSHA in 1971.

Repeated short-term feeding tests have shown the acute toxicities of PCB's in animals to be low, with LD_{50} values ranging from 1 to 10 gms. per kg. of body weight.

3. Impurity Effects The association of human chloracne (see below) with an unidentified PCB impurity was reported in 1936 (23) but was apparently forgotten. Then, in 1970, a Dutch scientist, J.G. Vos, observed striking differences in chick embryotoxicity among three commercial PCB's that all contained 60% chlorine: Aroclor 1260 (which had virtually no effect), Clophen A60, and Phenoclor DP6. He then demonstrated that the variable toxic agent in the system was the impurity, polychlorinated dibenzofuran (PCDF) (30), and that this impurity was also responsible for chloracne in PCB dermal tests with rabbits (31). Chemical analyses subsequently showed that European and Japanese PCB's contained 5-20 ppm PCDF as manufactured, while the US-made Aroclors had 0-2 ppm.

It is not possible to also ascribe the toxic response variabilities observed in other animal tests to variations in PCDF because, unfortunately, few of the investigators reported impurity levels in the PCB specimens they tested.

4. Structure-Activity Relationships Many recent animal studies have aimed at relating the molecular structures of individual PCB, PCDF, or similar molecules to their biological activities. These studies have shown that chloracnegenic activity is specifically associated with molecules shaped like flat rectangles with chlorines at the corner (21). All such chloracnegenic species are also capable of inducing one specific type of oxidase enzyme, designated P448 because of its spectral band position.

Three rare PCB isomers, which constitute less than one percent of the higher Aroclors, and which are virtually absent from the lower Aroclors, have approximately the right shape and exhibit detectable P448-inducing activity. The really strong P448 inducers, however, are the PCDF's, which are typically 1000 times as active as these three PCB isomers .

5. Carcinogenicity Test Results The American PCB's (Aroclors) have been tested for carcinogenicity in animals in many ways, with results listed in Table III below. Similar tests with the Japanese PCB's (Kanechlors 300, 400, and 500), which presumably contained 5-20 ppm PCDF's, have also produced positive, negative, and anticarcinogenic findings.

The reports of some positive findings in lifetime rodent tests at maximum tolerated dosage levels (e.g., 100 ppm in the diet), while prompting caution, represent a common observation. Examinations of HEW's "Survey of Compounds Which Have Been Tested for Carcinogenic Activity," PHS Document 149, Vols. 4,5,6, and 7, shows that a substantial majority of all substances tested since 1960 (3233 out of the 4233 PHS-149 substance listings) have been reported to produce excess tumors in animals.

TABLE III
U.S. PCB CARCINOGENICITY TESTS

Aroclor No.	Route	Species Result	Author	Date
1221	rabbit-oral	No cancer	Koller, Zinkl	1973
1016	rat-oral	No cancer	Burse, Kimbrough et al	1974
1242	rat-oral	No cancer	Monsanto Contractors *	1971
1242	rabbit-oral	No cancer	Koller, Zinkl	1973
1242	rat-oral	No cancer	Burse, Kimbrough et al	1974
1254	rat-oral	No cancer	Monsanto Contractors *	1971
1254	rat-oral	Liver cancer	Kimbrough et al	1972
1254	mouse-oral	Liver cancer	Kimbrough and Lindner	1975
1254	rat-oral	No cancer	NCI Carcin. Program	1978
1254	mouse-skin	Not tumor-initiating	DiGiovanni et al	1977
1254	mouse-skin	Not tumor-promoting	Berry et al	1978
1254	mouse-skin	Anticarcinogenic	Berry et al	1978
1260	rat-oral	No cancer	Monsanto Contractors *	1971
1260	rat-oral	Liver cancer	Kimbrough et al	1975

* Testing by Industrial Biotest; 1975 data review by D. Gordon (Industrial Biotest), W. Richter (U. of Chicago) and P. Pour (Eppley Institute for Research in Cancer). (27)

After the Vos identification of PCDF impurities as the active toxic agent in two PCB animal test systems, Japanese scientists reanalyzed some of the rice oil specimens which had caused Yusho illness (32). By 1975 it was known that these rice oil samples contained about 1000 ppm PCB's, 5 ppm of PCDF's and 1000 ppm of another chlorinated material, later identified as polychlorinated quaterphenyls (33). The PCDF's and PCQ's presumably were formed from PCB's by thermal oxidation and condensation in the heat exchanger at high temperature. This analysis would imply that the leaking heat exchange fluid itself contained PCDF's at the extraordinary level of 2500 ppm.

As the Yusho victims were estimated to have ingested an average of 2.0 gms. of the heat exchanger fluid, this amount would have included about 1.0 gm. each of PCB and PCQ, along with 5 mg. of PCDF. As will be noted later, 1.0 gm. of PCB is less than is detected in heavily-exposed capacitor workers. Unlike capacitor workers, the Yusho victims rapidly eliminated their PCB burdens and showed normal background levels of PCB's in about a year. However, the patients remained sick, and such autopsy liver samples as became available showed that significant levels of PCDF's were retained in that organ.

Since doses of electrical-grade PCB's, absorbed by inhalation and skin contact, larger than the Yusho ingestion have not induced the Yusho illness, these later Japanese investigations link the disease to the PCDF content of the rice oil, a hypothesis which is consistent with PCDF activity in animal tests.

3. Effects of Eating Fish Containing PCB's The Michigan Department of Public Health conducted an FDA-sponsored study (38) of 182 adults, 105 of whom consumed over 26 pounds of Great Lakes fish (year) per year. A significant correlation between blood PCB levels and quantity of fish eaten was observed. The mean blood PCB value for the exposed group was 73 ppb, while that of the comparison group was 20 ppb. Blood PCB levels ranged from 7 ppb for a person who ate no fish to 366 ppb for an individual who ate 132 pounds per year.

Evaluation of health histories and current medical problems of the study subjects failed to identify a significant difference between the exposed and comparison groups. Symptoms characteristic of reported PCB toxicity were not found nor did those with the highest PCB levels have a consistent pattern of complaints or conditions. Although the data demonstrate an association between fish consumption and PCB levels in humans, no toxic manifestations of this exposure have been identified to date. The question of PCB accumulation over time and the impact of long-term exposure are being evaluated in a continuation of this study. (39)

4. Occupationally-exposed Capacitor Workers The most extensive and severe long-term exposure of humans to PCB's has probably occurred in capacitor plants around the world (there were 17 capacitor plants using PCB's in the U.S.). Many employees in these plants had daily PCB skin contact for several years and the inhalation levels have been measured in the 100 to 1000 ug/m3 range. General Electric's major capacitor fluid useage was

originally Aroclor 1254, then changed to Aroclor 1242 around 1952, and subsequently transitioned to Aroclor 1016 (and some 1221) around 1971. This useage pattern was probably typical of the industry. PCB blood tests of heavily-exposed GE capacitor workers have indicated a log normal distribution whose geometric mean was around 330 ppb. Ten percent of the individuals' analyses were above 1000 ppb. (41)

A blood PCB value of 330 ppb roughly corresponds to a 130 ppm level in fatty tissue of the same individual, or a body content of about 1.5-2.0 grams. By comparison, background PCB blood levels for industrially unexposed persons in the U.S. range from 5-25 ppb (13).

In capacitor plants the most frequent health effect observed has been transient skin rashes affecting a small percentage of exposed employees. For example, analysis of GE medical records of the Ft. Edward and Hudson Falls, N.Y. plants for the 1960-1975 period showed a cumulative total of 49 allergic dermatitis cases, but none of chloracne, attributed to PCB's among an exposed employee group which totalled about 1300 individuals (28). The condition responded to simple topical treatment and employee reassignment to other work areas. Only 16 other GE clinical cases of possible PCB health effects were noted in this 15-year period. Of significance is the fact that the medical records of this worker population showed no obvious incidence of systemic disease attributable to PCB exposure.

Observations of high PCB blood levels, some dermal conditions and isolated cases of chloracne are reported in the industrial hygiene literature of Sweden, Australia and Japan (2,3,9,10), but other causally-related adverse health effects have not been shown in heavily-exposed capacitor workers.

Public concern about Yusho and inferences drawn from animal tests have stimulated many other clinical investigations of human health effects in addition to those cited above. In one study, 32 capacitor workers were examined in a program sponsored by the South Carolina State Department of Health and Environmental Control. Newspaper reports (January 1978) quote Dr. D.H. Robinson of the DHEC as saying that the study clearly shows that at present there is no evidence of physical harm from working with PCB's (4).

A study of occupationally-exposed Bloomington, Indiana capacitor workers and other residents, reported in 1978, did not note liver disease, but found that levels of a liver enzyme increased with increases in serum PCB levels and that plasma triglyceride levels were higher than normal in capacitor workers (5). A letter to the Bloomington study participants from the U.S. Center for Disease Control noted that human cancer risk would remain unknown until the extensive study (reported below) then being conducted by NIOSH had been completed.

In 1976 Dr. Irving Selikoff, of Mt. Sinai School of Medicine was asked to check on the health status of capacitor workers in the GE plants at Hudson Falls and Ft. Edward, N.Y. Three hundred

326

twenty-six volunteers who generally had a long history of some occupational exposure to PCB's were given extensive medical examinations. In one paper the investigators reported average PCB blood levels of 124 ppb (lower homologs) plus 48 ppb (higher homologs) and noted a correlation between individual values and the estimated job exposure (6). Some dermatological findings were reported to correlate with blood PCB levels of the higher homologs. The authors noted a paucity of other abnormal findings on physical examination, including a very low prevalence of abnormal liver findings.

THE UNIVERSAL CONCLUSION

In another 1979 paper the Mt. Sinai group reported that 14% of the workers examined had abnormal vital (lung) capacity, compared with 5.6% cited in this paper for Morris' normal population (7). The significance of this observation is uncertain and requires further study.

Medical surveillance by General Electric of 193 heavily-exposed capacitor workers has consisted of multiple examinations over the last four years. As previously noted, persistent high PCB blood levels have been found; but there has been a general absence of serious health problems. There are elevations of blood lipids in some males. Pulmonary function tests of vital capacity in non-smokers have been in the normal range. Statistical analysis of this examination data is continuing (41).

A rough correlation which is emerging from this study of GE capacitor workers is that people who are regularly exposed to PCB's may eventually accumulate about 1 ppb in their blood for each 1 ug/m^3 present in the air. Thus, long-term environmental exposure to air containing 2 ug/m^3 PCB might be expected to add about 2 ppb to an average person's 5-25 ppb PCB background level, a small amount compared with the normal background, or with the levels observed in heavily-exposed capacitor workers (200-1000 ppb).

5. NIOSH Epidemiology Study With respect to human carcinogenic effects, three reports of possible cancer association with PCB-exposure (27,34,35) have been regarded as inconclusive, as they involved small groups of people, few deaths, uncertain levels of exposure to PCB's and to other chemicals, generally short latency intervals, and lack of a consistent cancer site incidence.

After reviewing both the animal tests and the above human reports, the FDA assessment in 1979 (36) was that the question of carcinogenicity of PCB's was unresolved though a matter worthy of further serious inquiry. The International Agency for Research on Cancer has evaluated the degree of evidence for PCB's causing cancer in humans as "inadequate" (37). Neither of these 1979 evaluations had yet received a report of NIOSH's long-term mortality study of a PCB-exposed capacitor worker population, which showed no excess in overall cancer mortality.

A report of this NIOSH epidemiology study, by far the most comprehensive ever conducted concerning human PCB exposure, has recently become available (8), and it provides the best data available on mortality experience. The retrospective cohort standardized mortality study includes 2349 PCB-exposed capacitor workers in two plants. One of these plants was the GE Hudson Falls and Ft. Edward, N.Y. complex. The cohort was defined as all workers who accumulated at least three months employment in areas of the plant where there was potential for exposure to PCB's. Exposure in one plant began as early as 1938, in the other by 1946, and a total of 38,890 person-years had been accumulated by the January 1, 1976 study cutoff.

The NIOSH study reports that the incidence of all cancer mortality for these plant populations has been slightly lower than that of the general U.S. population, 39 deaths due to malignant neoplasms vs. 40.62 expected based on national norms adjusted for age and sex. For other causes of death the findings were: from cardiovascular disease, 60 vs. 61.86 expected; from nervous system diseases, 11 vs. 12.3 expected; from accidents, 13 vs. 17.64 expected; and from all other causes, 40 vs. 41.62 expected. Deaths from all causes were 163 vs. 174.04 expected.

No statistically significant excesses of specific cancer types were observed, though the authors called attention to four rectal and three liver cancer cases. This should be followed up by extension of the study, although these present mortality numbers are too small to be meaningful. There was no latency relationship demonstrated with total cancer mortality, nor was there an increase in mortality with increasing lengths of occupational exposure. Liver cirrhosis deaths were higher than normal in one plant (with some alcohol association possibility noted) and lower in the other, the total cohort experience being about equal to the national norms.

C. Summary

To summarize, there have been many PCB toxicological studies, of both animal and human subjects, dating back to the 1930's. These led to the establishment of PCB exposure guidelines (later, standards) that were apparently quite successful in preventing serious injury to heavily-exposed American capacitor workers. A human hazard that was not recognized early was the possibility that PCB's used in very high temperature environments, such as heat exchangers or baking operations, could be partially oxidized to PCDF's, with consequent increase in toxicity. Even in such cases, the health hazard is finite. The Japanese Yusho disease investigations reported that the threshold dose may have been about 0.5 gms. for ingestion of the thermally-decomposed heat exchange fluid that was involved. (PCB-PCQ-PCDF mixture containing about 2500 ppm PCDF).

As regards the ordinary, unpyrolysed, U.S.-made electrical grade PCB's which may be involved in some industrial or environmental exposure situations, the hazard potential can be evaluated from the occupational health experience of capacitor workers, who had many years of heavy exposure (regular skin contact plus 100-1000 ug/m^3 in air). The paucity of evidence for health damage in these individuals, combined with the finding of normal long-term mortality experience, indicates that there is little basis for concern over the effects of occasional environmental exposure to such PCB's at the levels typically encountered (0.1-10 ug/m^3 and/or occasional skin contact).

There are some unanswered questions regarding possible low level or subtle effects in very heavily-exposed individuals, and there are on-going programs to attempt to resolve them. These are discussed in this paper's concluding section. But the extensive human health experience cited in this paper and the low future exposure potential do not indicate that typical environmental levels of electrical grade PCB's present a significant human health hazard.

V U.S. PCB Regulations

Early fire protection regulations covering confined-space electrical equipment essentially mandated the use of PCB-filled transformers. However, the subsequent concern about environmental accumulation and about possible human health effects led in 1976 to a national prohibition (TSCA, Section 6e) of new production and continued manufacturing use of PCB's. This prohibition is embodied in EPA phaseout rules which define" PCB" capacitors and transformers as those containing "PCB liquids", defined in turn as any liquid in which PCB concentration is greater than 0.05% (500 ppm). "PCB-contaminated" transformer oils (and the corresponding transformer) are defined as those containing 0.005%* to 0.05% of PCB material. The regulations contain the following major requirements: (40)

- PCB capacitors and transformers and PCB-contaminated transformers may be used for the remainder of their useful life.
- Most PCB equipment remaining in use is subject to marking requirements.
- Small PCB capacitors may be disposed of without restriction in municipal landfills (except by equipment manufacturers).
- Large PCB capacitors which are taken out of service must be drained and may be shipped (temporarily) to an approved chemical landfill. After high temperature incinerators are approved, these must be used for the future disposal of large PCB capacitors.
- Any PCB waste fluids ($\geq$ 0.05%) must be disposed of by high temperature incineration in EPA-approved incinerators. (No commercial incinerators have yet received approval at this writing).
- PCB-contaminated waste fluids (0.005%-0.05%) must be disposed of in high efficiency boilers, in approved chemical waste landfills, or by high temperature incinerators.
- Waste oil containing any detectable level of PCB's cannot be used as a dust control agent.
- PCB liquids and equipment awaiting final disposal are subject to marking and storage requirements.

* A recent court case requires EPA to reexamine this definition.

The Food and Drug Administration noted the presence of PCB's in food and established regulations in 1973 for certain foods in interstate commerce. Present tolerance limits for various food items were adjusted downward effective August 28, 1979 to the following levels: (29)

Milk and dairy products	1.5 ppm (fat basis)
Poultry	3 ppm "
Eggs	0.3 ppm "
Fish and Shellfish	2 ppm *(currently 5 ppm)

* The proposed 2 ppm tolerance limit for fish and shellfish has been stayed, pending resolution of objections raised concerning the final rule.

VI On-going Programs

Notwithstanding the experience of 50 years PCB useage and the extensive research which has been completed, some questions and uncertainties remain. General Electric and others are continuing research on some of these issues.

The actual PCDF content of PCB's in existing capacitors and transformers as well as in various environmental samples needs to be determined. Based on the reported PCDF content of Aroclors (0-2 ppm) one may postulate that PCDF's will be detected only in such minute quantities that they should not be of concern. This question needs to be resolved by analysis and evaluation, and GE is continuing an analytical research program to make some of this data available.

A number of medical research programs are continuing which will further clarify the PCB health effects understanding. New York State's Department of Health (Dr. Phillip Taylor) is analyzing further the epidemiology of the GE capacitor worker population, and is extending the research to include reproductive experience. General Electric is continuing medical surveillance of previously exposed capacitor workers to further characterize their health status. And some additional observations may become available from the Mt. Sinai School of Medicine work. The Michigan State Department of Health is extending and broadening their study of individuals who have ingested PCB's by eating fish.

These on-going studies may indicate if medical surveillance is appropriate for individuals exposed to PCB's in the past. While a confirmed* blood or fat tissue PCB analysis significantly higher than the local average can indicate exposure, on the basis of present knowledge there is apparently no further examination procedure which is meaningful. Nor is there any therapy or medical advice that can properly be given such an individual other than to reassure him or her that, on the basis of capacitor workers' experience, there is no reason to believe that significant adverse health effects will occur.

Another question concerns industrial exposures of other than capacitor workers. Transformer plants, for example, typically used Aroclor 1254 and 1260, usually mixed with chlorobenzenes, for a portion of the plant's production, whereas Aroclor 1260 was not used in capacitors. And epidemiological studies of transformer workers exposed to PCB's are not available. The capacitor worker experience may be relevant, however, as Aroclor 1254 and 1260 contain a substantially overlapping, though not identical, population of PCB molecules; and capacitor worker blood analyses show retention of molecules associated with 1260.

Lastly, many research programs are being directed at economically effective containment and conversion processes for PCB's in order to respond to Federal and State requirements. General Electric research is active in some of these programs.

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* An unconfirmed individual PCB measurement should be regarded cautiously: a recent round-robin study by the N.Y. State Health Dept. showed at least one case where 2 out of 10 commercial analyses of the same biological sample reported PCB values that were 20-fold different from the average.

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ACOUSTIC TRANSIENT SUPPRESSOR FOR ROTARY PNEUMATIC TOOLS

A significant reduction in noise from one of the major noise sources in the metal-working industry has been achieved through the realization of the basic mechanism of the source. H. A. Scarton of the Rensselaer Polytechnic Institute (R. P. I.) Laboratory for Noise Control Research (Troy, NY 12181) discussed the transient run-down noise of a horizontal grinder powered by a sliding-vane pneumatic motor and how it was reduced.

Speaking at NOISE-CON 81 held at North Carolina State University at Raleigh, June 8-10, Mr. Scarton explained that the transient run-down noise is caused when the air supply to the rapidly-rotating pneumatic motor is suddenly interrupted causing a braking effect on the shaft. The energy source thus ceases to be the air supply and becomes the rotational kinetic ($1/2\ I\omega^2$) of the rapidly rotating shaft. Once one realizes that no useful work is being performed during this time when the air is being sucked back into the tool as the tool decelerates, then it becomes clear that a simple check valve should prevent all such fluid motion from occurring and lower the sound level.

To evaluate results, the steady-state free-running and transient rundown signals were obtained using a Bruel and Kjaer Type 2209 Sound Level Meter at a distance of 24 inches from the source for a Dotco horizontal grinder when equipped with original factory-supplied mufflers, and the same grinder when equipped with the R. P. I. Transient Suppressor. The meter attenuator settings were set at 110 dB for both measurements. A 15 dB drop in transient noise level to 92.5 dB was recorded for the R. P. I. Transient Suppressor as compared with the Dotco original version. The dramatic reductions of noise levels in both free-running and transient-rundown states for the R. P. I. Transient Suppressor version over the Dotco version which has no such device require only a modest increase in the diameter of the muffler housing sufficient to accommodate the worker's gloved hand.

NEW PASSIVE DOSIMETER FOR MONITORING PERSONNEL EXPOSURE TO PNA VAPORS

A new device for the collection and detection of multi-ring PNA compounds in the vapor phase has been developed at the Oak Ridge National Laboratory (Oak Ridge, TN 37830). The device is a passive dosimeter able to determine time-weighted average concentration using the principle of molecular diffusion. The PNA pollutants are adsorbed onto a filter paper treated with a heavy-atom salt. After exposure the filter paper is inserted into a spectrometer and the levels of PNA determined directly without requiring extraction. The detection method uses the room temperature phosphorescence technique. The limits of detection for several PNA compounds such as phenanthrene, pyrene, and quinoline are in the part-per-billion range for concentration in air. A description of the device was given by T. Vo-Dinh at the American Chemical Society meeting in New York City, Aug. 23-28.

NEW METHOD FOR DETERMINATION OF FORMALDEHYDE IN AIR

A new sampling and analytical method for determination of formaldehyde in air was described by Eugene R. Kennedy of the National Institute for Occupational Safety and Health (4676 Columbia Parkway, Cincinnati, OH 45226) at the American Chemical Society's national meeting in New York City, Aug. 23-28.

Formaldehyde in air reacted with N-benzylethanolamine(BEA)-coated Chromosorb 102 sorbent to produce a derivative of formaldehyde, 3-benzyloxazolidine. Sorbent-tube samples were desorbed with isooctane and this derivative determined by capillary gas chromatography using the splitless injection technique. Analysis was accomplished using a 25-meter Carbowax 20M fused silica capillary column. Separation of this oxazolidine of formaldehyde from other low-molecular-weight aldehyde oxazolidine derivatives was possible. The range of the method was from 0.55-2.88 mg/m^3. By utilizing the split injection technique, the range was extended to at least 4.71 mg/m^3. Samples were collected at a rate of 50 cm^3/min for four hours. This volume translated to 6.6-56.5 µg of formaldehyde per sample for the range of the method. Overall relative standard deviation for the sampling and analytical method was 9.1 percent.

POTENTIAL HEALTH HAZARDS OF VIDEO DISPLAY TERMINALS INVESTIGATED

Based on low levels of radiation emitted by video display terminals examined in an investigation of three workplace sites, the National Institute for Occupational Safety and Health has determined that routine radiation surveys of VDTs are not warranted. The investigation was undertaken at the request of three unions in California where numerous complaints had been received from employees using VDTs in information processing applications. The operators reported a wide range of symptoms including headaches, general malaise, eye strain and other visual and musculoskeletal problems.

Three companies in the San Francisco Bay area participated in the survey which was conducted in four phases: (1) radiation measurements; (2) industrial hygiene sampling; (3) a survey of health complaints and psychological mood state, and (4) ergonomics and human factors measurements. Measurements of ionizing and nonionizing radiations were made on a random sample of 25 percent of the VDTs. Samples of workroom air were obtained and analyzed to determine exposure to selected airborne chemical contaminants. Health complaints and psychological mood state in VDT operators and a comparison group of non-operators were evaluated using a multifaceted questionnaire. The ergonomics and human factors evaluation was conducted by examining several workplace and VDT characteristics.

The radiation surveys demonstrated that exposure to x-ray, radio-frequency, ultraviolet, and visible radiation was well below current occupational exposure standards, and, in many cases, below the detection capability of the survey instruments. The air samples showed that there were no hazardous chemical exposures. Specifically, workplace ambient levels of carbon monoxide, formaldehyde, hydrocarbons, acetic acid and ozone were all well below current occupational exposure limits. The questionnaire survey indicated that a higher percentage of VDT operators reported more visual complaints at two of the sites, more muscular complaints at one site, and more emotional complaints at all three sites. The ergonomic evaluation of the VDT workstations indicated that measured illumination levels were generally acceptable; however, glare was a problem at a number of workstations. Some problems were noted with the physical dimensions of the workstations, including excessive keyboard height and VDT screen height.

Details of the investigation are contained in the newly released NIOSH report number 81-129. Single copies may be obtained by sending a self-addressed label to NIOSH, Publications Dissemination, 4676 Columbia Parkway, Cincinnati, OH 45226.

A SAMPLING DEVICE FOR SKIN-CONTAMINATION EVALUATION

A method has been developed by which a representative sample of contaminants on the skin of workers in coal-liquifaction plants may be collected and analyzed at extremely low levels regardless of the hazardous substance in question. The following technique was described at the American Industrial Hygiene Association meeting in Portland, Ore., in May by R. R. Keenan and S. B. Cole of Gulf Science and Technology (P.O.Box 1166, Pittsburgh, PA 15230).

In this procedure, approximately 10 ml cyclohexane is sprayed onto a limited area of the skin (5 cm^2) to be evaluated. Samples are collected by affixing a 12-ml vial to the sampling cup, holding the cup against the surface of the skin to be sampled and spraying cyclohexane through the cup onto the skin. The washings automatically drain into the sample vial. Sampling is terminated when the level of the cyclohexane in the vial reaches the sampling cup drip tube (approximately 10 seconds). The vial is removed, sealed with a Teflon-lined screw cap and returned to the laboratory for analysis. The cup is cleaned between samples by rinsing with a small amount of cyclohexane.

Prior to analysis, the samples are equilibrated to room temperature and the volume of each sample is determined by comparison with a calibrated vial. Colored samples are diluted before analysis. The samples are then transferred to 10-mm ultraviolet fluorescence cells and analyzed on an Aminco Model SPF-500 Ratio Spectrophotofluorometer by excitation at 293 nm. The presence or absence of fluorescence quenching is checked by viewing the sample cell in its holder with the excitation shutter open and the emission shutter closed. If excessive fluorescence is observed, the sample is diluted. The fluorescence emission spectra are recorded from 300 to 550 nm at a scan rate of 600 nm/min. Quantitation is accomplished by comparing the fluorescence intensity of the sample to that of the heavy distillate calibration curve. If the maximum fluorescence of the sample does not coincide with 355 nm, but falls within the 330- to 375-nm range, the fluorescence intensity at the wavelength of maximum fluorescence is determined, recorded and compared to the calibration curve previously generated.

Approximately 75 samples per day can be analyzed with this procedure. The limit of detection is 0.2 µg (0.04 µg/cm^2). Preliminary studies have exhibited recoveries of greater than 95 percent. While this sampling technique has been used solely for the collection of coal-derived materials for analysis by ultraviolet fluorescence, its application can be extended to include other contaminants as long as suitable analytical procedures reasonably free of interferences are available.

USE OF VIBRATION DAMPING IN NOISE CONTROL

Vibration damping is a practical and effective noise control measure for all types of products, especially where resonances exist, according to Robert N. Baker of Blachford Engineers (1441 E. Maple Rd., Troy, MI 48084). Speaking at NOISE-CON 81 held June 8-10 at North Carolina State University at Raleigh, Mr. Baker cited the following case histories:

In one instance, a large vibratory parts feed device (which are common sources of noise in many plants) was used to provide washers to a thread roller. The parts left the feed device, entered an indexing wheel mechanism and were assembled on the screw blank.

The thread roller then formed the thread, capturing the washer on the screw. Noise of the feed device was a result of forced vibration of the large sheet metal surfaces as they were repeatedly struck by the washers. The inside surfaces of the feed devices were covered with an epoxy-based resilient lining which reduced the peak accelerations as the washers fell against the surface, and which also provided vibration damping to the steel. Noise was reduced 9 to 10 dB(A).

A small electrical slip ring assembly produced an objectionable high frequency sound variously described as "squeaking," or "hissing." The slip ring assembly was basically comprised of two dish-like plastic components known as the stator and the rotor, respectively. In the rotor were mounted five cantilever spring-type brushes. These made electrical contact with five stator-mounted slip rings to allow relative motion between rotor and stator. Small irregularities in the slip ring surfaces caused variations in frictional forces on the contact tips or "buttons" at the end of the cantilever spring brushes. Any of a number of the vibrational modes of the spring could be excited as a result. The lowest frequency of these was at about 1000 Hz. A metallic foil-faced vibration damping tape with a pressure sensitive adhesive was applied to the brushes and the face of the rotor (which was excited to vibration by the vibration of the brushes). This treatment reduced noise from the assembly by 5 dB(A).

A significant portion of the noise heard by the operator of a large U.S. Army forklift truck was due to the forced vibration of a number of sheet metal surfaces. Resonances were not a particular problem in this case. After feasible vibration isolation treatments had been employed, those surfaces which still contributed significantly to operator ear position noise level were treated with an extensional dampling treatment. Thickness was typically about 3 mm for the damping material. Panel thickness was typically about 1.6 mm. The average reduction in the acceleration levels in the panels was 13 dB and occurred at those frequencies where the noise levels at the operator's ear were highest.

HEALTH STATUS OF WORKERS WITH LONG TERM EXPOSURE TO PCBs

To evaluate the health effects from occupational exposure to polychlorinated biphenyls, a clinical survey was conducted of 326 workers employed in a capacitor and transformer manufacturing facility where PCBs had been used since the 1940s. At the American Chemical Society's national meeting in New York City, Aug. 23-28, Alf Fischbein of the Mount Sinai Medical Center (New York, NY 10029) gave the following summary of the study and findings:

Aroclor 1016 and, to a minor degree Aroclor 1221, had replaced the higher chlorinated PCBs two years prior to this study. Forty percent of the workers, half of whom were females, had been employed for 20 years or longer. Mean plasma concentrations of higher (Aroclor 1254) and lower (Aroclor 1242) congeners of PCBs were 124 ppb and 48 ppb respectively. Despite high plasma concentrations of PCBs, which correlated well with intensity of exposure, there was a paucity of clinical and laboratory abnormalities. However, there was a high prevalence of abnormal dermatological findings, including chloracne (6 percent) and conjunctival and palpebral abnormalities (16 percent). Association was found between dermatological abnormalities and plasma levels of higher congeners of PCBs. A similar relationship was demonstrated between higher congeners of PCBs and serum triglyceride levels. No evidence of porphyria or peripheral nerve dysfunction was found in a subgroup of this population. These findings give credence to the hypothesis that clinical effects ascribed to PCBs, may, in part, reflect a higher disease-inducing potential of contaminants, such as dibenzofurans.

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PROCEEDINGS OF THE CONFERENCE
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SARDINAS: I'd like to welcome you today. My name is Anthony Sardinas. I'm the Division Director for Preventable Diseases at the Connecticut State Department of Health Services. Today, the Department is co-sponsoring this meeting on polychlorinated byphenyls and the health effects of these substances. We're particularly interested in the public health implications of PCBs, polychlorinated byphenyls. We're co-sponsoring this meeting with Toby Moffett's office today. It was coordinated at the request of the Connecticut Citizens' Watchdog Committee. Our goal is to inform the public of the current state of knowledge in respect to the health hazards imposed by PCB's and the general public. We're particularly interested, and this subject, of course, is timely, with regard to the PCBs that have been found in fish in our rivers. We hope that this conference will help the public to make an informed decision as to whether or not they want to risk eating contaminated fish or they want to risk drinking water with PCBs and that sort of thing. It's also hoped that this meeting will provide a better understanding of Connecticut's PCB program. Now, we have with us today two panels actually which are somewhat intermingled. We have a national panel of experts and we also have a local panel of experts. Let me first introduce to you our expert panel of speakers. We have with us Dr. Albert C. Kolbye. Dr. Kolbye is the Associate Director of Science, Bureau of Foods, for the Food and Drug Administration. We also

SARDINAS: (Continued) have Dr. Elizabeth Weisburger. Dr. Weisburger is the Chief of the Laboratory of Carcinogen Metabolism, the Division of Cancer Cause and Prevention, with the National Cancer Institute in Washington. Also with us today is Dr. James Douglas. Dr. Douglas is with the Yale School of Epidemiology of Public Health, he's also a member of the Pierce Foundation in New Haven, and he's on the Toxic Substances Advisory Board for the Environmental Protection Agency. We have Dr. John F. Brown, Jr. Dr. Brown is the Manager of Life Sciences Branch for General Electric in New York. I'll introduce the local panel to you after we've had an opportunity to hear from the expert panel, and, and give the local panel an option to ask some questions. So, let's begin in alphabetical order with the National panel first, and I'll call on Dr. Brown, again, to begin, to try to limit your comments as much as possible to about 10 minutes. Dr. Brown.

BROWN: (READS THE FOLLOWING)

THE PERILS OF PCB: MYTH AND REALITY

Remarks presented at public panel discussion:
"Public Health Implications of PCB's"
Hartford, Connecticut, Sept. 12, 1979

by John F. Brown, Jr., PhD
Mgr. - Life Sciences Branch
General Electric Research and Development Center
PO Box 8, Schenectady, N.Y.

At the outset, let me say I am not a Company officer, and my remarks do not represent Corporate policy statements. I am the head of a Life Sciences R&D Branch, and my opinions are those of chemical, medical, and biological research scientists who have been concerned with PCB's.

I have been asked to focus my present remarks upon PCB health hazards. However, I should point out that our studies have also addressed several other PCB issues that I would be glad to discuss if questioned. These include chemical composition, analytical methods, disposal technology, stability and fate in the natural environment, microbial degradation and biological effects.

With respect to present scientific knowledge about the health effects of PCB's, I'd like to make five main points.

First, there is a large body of scientific literature out there, over 500 original papers on PCB bioeffects. This means that many questions regarding persistence, toxicity, and carcinogenicity can be answered on the basis of observations already reported, if you're just willing to dig in, search, and correlate.

Second, the basic patterns of effects induced in warm-blooded animals by the toxic types of PCB's are identical to those produced by polychlorinated dibenzodioxins and polychlorinated dibenzofurans. Such patterns vary somewhat with the animal species. In chickens, the characteristic result is edema disease; in mink, it is reproductive failure. In rat and mice, there is progressive damage to the liver, culminating in cancer. That result, however, has never been seen in guinea pigs,

monkeys, or man. Instead, the characteristic pattern in humans features acne-like skin eruptions, called "chloracne," along with changes in skin pigmentation, peripheral numbness, digestive upsets, headaches, and fatigue.

Third, if one compares the reported observations, whether on chickens, rats, guinea pigs, mink, monkeys, or man, one finds an enormous variability in the ability of different PCB specimens to produce toxic effects. In many cases, the original investigators themselves recognized this variability, recognized that they were dealing with an impurity-dependent toxicity, and clearly reported it as such. Within the past year, these variabilities in effect have led both the International Agency for Research on Cancer, and our own FDA to declare the evidence for the carcinogenicity of PCB's to be inconclusive.

But there's nothing new about this. The very first American report on PCB-related chloracne, back in 1936, recognized that toxicity in PCB's as an unusual phenomenon that was associated with unidentified impurities in a single bad batch of early PCB.

Fourth, since 1970 the toxic impurity has been repeatedly identified as a polychlorinated dibenzofuran, or PCDF. PCDF's have apparently been virtually absent from industrial PCB's as made in this country for at least the last 25 years, but they have been present in European and Japanese production at levels of 5-30 parts per million, and in a number of the laboratory specimens used in biological testing. More significantly, these highly toxic PCDF's can be formed by scorching PCB's at temperatures above 500°F. The high temperature heat exchanger fluid that caused the Yusho illness episode in Japan is now known to have been a PCB that had been more than half decomposed to polychlorinated quaterphenyls and PCDF's.

Fifth, there is abundant evidence that human exposure to ordinary, unscorched PCB is without obvious health effects. PCB's were in widespread industrial use in all developed countries for nearly fifty years. Despite this, there have been only a handful of definite reports of

chloracne syndrome - all associated with baking, soldering, or heat-transfer applications.

The lack of toxic effects in the vast majority of exposed workers cannot be related to lack of PCB uptake, exposure time, or medical observation. Repeated studies of exposed electrical workers in both the US and Japan have shown them to be carrying in their bodies anywhere between 10 and 1000 times as much PCB as was found in the Japanese suffering from Yusho illness or roughly 100 times as much as the FDA figures you would get by eating fish controlled by their new 2 ppm tolerance limit. Many of these people had accumulated up to 30 years exposure before PCB use was stopped in 1977. Despite this, their health seems statistically indistinguishable from that of their unexposed cohorts.

In short, whether on grounds of toxicity or carcinogenicity, the scientific record provides very little basis for concern over low level exposure to ordinary, electrical grade PCB such as that presumably present in the Housatonic.

We all know, however, that this is not the popular perception of the case. One reads again and again the dread phrases, "acutely toxic," "linked to cancer," and "deadly." One sees pictures of policemen dressed like plutonium workers in order to clean up a contaminated grease spot, or farmers of the upper Hudson readying both shotguns and injunctions to stop PCB dredging, or New Jersey citizens mounting opposition to the use of an EPA-approved PCB incinerator. And all this over a widely used chemical that has produced no clear case of human illness in this country in the last 25 years.

This gulf between popular perception and scientific reality seems to have begun with good intentions. About 10 years ago it became apparent that PCB's were flowing into the environment faster than they were being degraded. This led to the prudent decisions, first, to sharply restrict PCB use and then to ban it completely. In order to

help promote the drive for a ban, the idea was advanced that perhaps any toxic effect exhibited by any type of PCB mixture in any animal should be considered a potential hazard of all PCB's in all creatures. This made an interesting as well as useful story, and it grew larger and larger with each retelling.

I believe that the PCB supertoxicity myth has now outlived its social value. The economic use and disposal of PCB is no longer an open issue. All PCB's were legally banned in the US in 1976. Their manufacture, import, and all major uses stopped in 1977. Elaborate procedures to limit the flow of scrap PCB into the biosphere were instituted by EPA in 1979. However, none of this can alter the fact that a hundred thousand tons have already passed into the biosphere. It is present, at varying levels, in the oceans, rainfall, soil, rivers, and fish, and there is no presently known human action that can radically change these environmental levels in the short term. There will be detectable levels of PCB's in fish from the Housatonic for decades, lower levels to be sure, but nevertheless detectable.

The real challenge we face today is learning to live with these realities. We must decide, for example, whether we'd rather give up fishing or readjust our perceptions of the perils of PCB's.

In my opinion, the FDA ruling of June 29, 1979, which proposed setting the Federal tolerance limit for PCB's in fish at 2 parts per million, makes sense only as a temporary measure. Basically, it is designed to provide protection to someone eating fish from waters contaminated with PCB's that are in turn contaminated with 1% of PCDF's, which was the extreme situation that caused the Yusho illness in Japan. The probability of this actually being the case is pretty low. However, the agency is forced to regulate on the basis of PCB's present rather than PCDF's feared because of the problem of chemical analysis. At present, chemists can reliably analyse fish for PCB's at parts-per-million levels, but not for PCDF's in the parts-per-billion range.

Fortunately, analytical methodologies are evolving very rapidly, and we think that valid high sensitivity techniques for PCDF's in fish should be available in a year or two. In due course, I would then hope to see the present Federal tolerance limit of 2 parts per million PCB replaced by a 20 parts per billion PCDF standard. This regulatory reform would probably permit the resumption of commercial fishing in several closed areas.

Meanwhile, I would hope that responsible State agencies could make diligent efforts to examine their PCB pollution problems on a region-by-region basis, and adopt regional fishing regulations that reflect the toxic hazard posed by the kind of PCB actually present locally, not just the worst possible kind as presumed by the Federal standard.

Obviously, any attempt to promulgate regional tolerance standards less rigorous than the Federal one will invite challenge. To develop and defend such standards will require hard work, citizen support, serious scholarly effort directed at the original scientific literature rather than agency summaries, and intellectual courage. However, it will not represent the first time that public health authorities have had to make hard choices between scientific reality and popular mythology. I would urge you to address this challenge.

SARDINAS: Thank you, Dr. Brown. Our next speaker is Dr. James Douglas, again from the Yale School of Medicine, Department of Epidemiology and Public Health, and the Pierce Foundation. Dr. Douglas.

DOUGLAS: Thank you, Mr. Chairman. First let me say that I am here as a toxicologist, and not with any special knowledge of the PCBs themselves. I have done no work in the area. And as far as we can ascertain there is little or no work being done on PCBs at Yale or in the Pierce Foundation. I have therefore spent some time reading the literature, and my knowledge is based on the material which is published. I think that there are a number of basic questions which come to my mind as a result of reading the literature. And I'm just going to briefly state what they are and not make any other formal presentation, but try to help in any questions that might come along. First of all, the fact that we can detect materials does not necessarily mean that they are toxic. Analytical methods become more and more sensitive all the time, and we can detect smaller and smaller quantities of materials. That does not necessarily mean that they exert toxic effects. And one of the things that we have to think about is the fact that PCBs are detectable does not mean that they are necessarily toxic. The methods of detection and analysis are particularly important. Toxicity is related to a particular substance. The PCBs are a group of substances which as, has been

DOUGLAS: (Continued) just mentioned, are, in some instances, contaminated with very small amounts of benzofurans. Certainly the toxicity of benzofurans seems to suggest that they are a good deal more potent than the PCBs. One wonders, therefore, whether many of the effects described to PCBs may not be due to contamination of that particular material. Toxicologists face a very difficult problem in that many materials are handled by animals in quite different ways. This is quite clear in the area of the PCBs where really there are fairly few animals which are truly representative of man, and I think that its fair to say that probably the primates give the closest types of responses to PCBs as compared to man, and there is, for various reasons relatively little work being done with the species. Having surveyed the evidence that is available, one of the concerns which I have with regard to the PCBs is the fact that the higher, more toxic species are very poorly metabolized, and are accumulated rather easily in various species. I think that that is an important consideration. It reminds me very much of, of DDT, for example, although DDT is a substance which could very easily be metabolized eventually. On the basis of the available information, I think that I would have to conclude that there's no conclusive evidence that the PCBs are carcinogenic. I think that considerable amount of data is equivocal. But I think that some of the more recent data would suggest that there are some effects in man, for example, Dr. Fishbein

DOUGLAS: (Continued) of Mount Sinai Hospital in New York, has reported some effects on pulmonary function in workers exposed to PCBs. But again, some of these studies are difficult to evaluate, since they do not have proper control groups. Thank you.

SARINAS: Thank you. Dr. Albert Kolbye, again, from the Food and Drug Administration. Dr. Kolbye.

KOLBYE: Thank you, Mr. Chairman. Ladies and gentlemen. I'll be speaking on behalf of the Food and Drug Administration, and explaining some of the viewpoints both legally and scientifically. My background is in medicine, epidemiology, toxicology and law. The Food and Drug Administration primarily has authority over interstate commerce with respect to food. And so the action levels that are set by Food and Drug refer to food that is in interstate commerce. At this meeting, if we are discussing sport fishing or intrastate commerce, I am here in an advisory capacity to share what knowledge and insight we in FDA have. To be a little bit more specific about the law, and not to get too legalistic, Section 406 of the Food and Drug Act is the one that applies to a PCB and fish situation. It's an Act that covers environmental contaminants in foods. And the law essentially says that FDA has the authority to set standards or action levels in fish, for example, to the extent necessary to protect public health. Let's

KOLBYE: (Continued) talk for a moment about human exposure and keep in mind that an action level is only one parameter. That's the level, so to speak, of permitted residues in a food commodity. A very important parameter that influences your exposure is how much food you eat at that level, and another important parameter is the frequency with which you are exposed to food contaminated at that level. Said in slightly different terms, if one has a systematic exposure, if one is eating day to day highly contaminated fish, one is getting a total exposure that is far different, and far stronger, if you will, than somebody who only occasionally consumes a particular food commodity that contains some residues of PCBs. So when we look at the overall health situation, the primary human experience has been referred to by other speakers, and that dealt with the disease suffered by the Japanese people, called Yusho. Back in the early 1970's, when FDA first got involved in regulating PCBs, we reviewed the evidence and available data from the Japanese populations and attributed all of the illness experienced by the Japanese to PCBs alone. Subsequently, we have determined that about half the organic chlorinated material in the contaminated rice oil was a quaterphenyl and that the level of polychlorinated dibenzofurans that were present in that rice oil approximated 500 parts per million. It has been mentioned that here in the United States the, the PCBs produced have been much cleaner with respect to

KOLBYE: (Continued) the presence of dibenzofurans. Here in the United States, to the best of my knowledge, the highest level of dibenzofurans that has been determined to exist approximate maybe about 10 parts per million in the PCBs. And so the American products have been much cleaner than the products manufactured in Europe or in Japan. One does have a problem though, as mentioned by an earlier speaker, that when PCBs have been exposed to heat formation of more of the dibenzofurans occurs. Now, the feeding studies in animals that have been conducted to attempt to assist the carcinogenic and chronic toxicity of PCBs also involve exposing the animal, since they're exposed to commercial preparations of PCBs, to whatever contaminants are present in that PCB product. And this then includes the likelihood that test animals were exposed to approximately 10 part per million level dibenzofurans in the amount of PCBs they received in their diet. So it's unclear to some scientists and to myself personally, what the exact role of PCBs is in terms of influencing the expression of cancer. And I'll be interested in Dr. Weisburger's comments in a few minutes. But basically the thing that one has to remember when considering PCBs, we're considering a spectrum of chemicals where the degree of chlorination and the positions chlorinated on the carbon rings are important determinants of the toxicity and the metabolic activity of these compounds. For dibenzofurans, there is about a thousandfold range of toxicity depending on the degree

KOLBYE: (Continued) of chlorination and the position of chlorination. And the same holds true for the dioxins. A 2378 tetrochlorodibenzodioxin is very, very toxic. But other tetroforms of the dioxins, if they're not chlorinated in those particular positions, have different toxicity. If you have higher chlorinated ones depending on position, they can be far less toxic. Thank you.

SARDINAS: Thank you, Dr. Kolbye. Dr. Elizabeth Weisburger is our next speaker, and again, she is from the National Cancer Institute. Dr. Weisburger.

WEISBURGER: (READS THE FOLLOWING

Delivered before Connecticut State Department of Health

on Seminar

"What is the Present Understanding of Health Hazards of PCB's"

Elizabeth K. Weisburger, Ph.D.
National Cancer Institute
National Institutes of Health
Bethesda, Maryland

Summary

A bioassay of Aroclor® 1254 for possible carcinogenicity was conducted by administering the test chemical in feed to Fischer 344 rats.

Groups of 24 rats of each sex were administered Aroclor® 1254 at one of three doses, either 25, 50 or 100 ppm, for 104-105 weeks. Matched controls consisted of groups of 24 untreated rats of each sex. All surviving rats were killed at 104-105 weeks.

Mean body weights of males and females receiving mid and high doses and females receiving low doses of the chemial were consistently below those of the corresponding controls, beginning at about week 10 of the study. The decrease in survival among males, but not among females, showed a significant dose-related trend. Adequate numbers of animals of both sexes survived for meaningful statistical analyses of the incidences of tumors.

The combined incidences of lymphomas and leukemias showed a significant dose-related trend in males (controls 3/24, low-dose 2/24, mid-dose 5/24, high-dose 9/24, P = 0.008). However, the direct comparisons of treated groups with those of the matched controls were not statistically significant, and the tumors cannot clearly be related to administration of Aroclor® 1254.

Hepatocellular adenomas and carcinomas were found in the dosed groups, but not in the controls (males: mid-dose 1/24, high-dose 3/24; females: mid-dose 1/24, high-dose 2/24). Additionally, a high incidence of non-neoplastic hyperplastic nodules was noted in the dosed animals (males: controls 0/24, low-dose 5/24, mid-dose 3/24, high-dose 12/24; females: controls 0/23, low-dose 6/24, mid-dose 9/22, high-dose 17/24). Although the incidences of tumors were not significant, the occurrence of the hyperplastic nodules appeared to be related to administration of the chemical.

In the stomach, jejunum, or cecum, adenocarcinomas were observed in two dosed males and in two dosed females as well as a carcinoma in one dosed male. None of these lesions was found in control animals in this study. Historical incidences of these tumors at this laboratory (6/600 males [1%], 2/600 females [0.3%]) suggest that the lesions -- although not statistically significant -- may be related to the administration of Aroclor® 1254.

It is concluded that under the conditions of this bioassay, Aroclor® 1254 was not carcinogenic in Fischer 344 rats; however, a high incidence of hepatocellular proliferative lesions in both male and female rats was related to administration of the chemical. In addition, the carcinomas of the gastrointestinal tract may be associated with administration of Aroclor® 1254 in both males and females.

Aroclor™ 1254 (CAS 27323-18-8; NCI C02664) is the registered trademark of the Monsanto Chemical Company for their polychlorinated biphenyls (PCBs). PCBs were developed in 1929 primarily for use as heat transfer fluids and dielectrics (insulators). Aroclor™ 1254, a biphenyl containing approximately 54% chlorine, is a nonflammable heat transfer agent which functions in the range of 290-360°C (Hubbard, 1964; Poffenberger and Hubbard, 1965).

This bioassay of Aroclor™ 1254 was conducted as part of a larger study designed to assess the combined effects of a group of known or suspected carcinogens. Only the results of the study of the administration of Aroclor™ 1254 are reported here.

Aroclor™ 1254 was obtained in a single batch (Lot No. KBOI-504) from the Monsanto Chemical Company, St. Louis, Missouri. The identity and relative purity of the test chemical were confirmed at Stanford Research Institute. Elemental analyses (C, H, Cl) indicated 54.6% chlorine. Gas-liquid chromatography and mass spectroscopy showed that the Aroclor™ 1254 contained at least 18 isomers of polychlorinated biphenyls ranging from 3 to 7 chlorine atoms per molecule. Identity was confirmed by nuclear magnetic resonance, infrared, and ultraviolet spectra, which were in agreement with the structure. No attempt was made to identify or quantitate impurities.

The chemical was stored at room temperature in amber glass jars.

Male and female Fischer 344 rats, obtained through contracts of the Division of Cancer Treatment, National Cancer Institute, were used in these bioassays. The rats were obtained from Simonsen Laboratory.

Gilroy, California. On arrival at the laboratory, all animals were quarantined for 2 weeks as an acclimation period. Following this period, all males gaining less than 25 grams, all females gaining less than 15 grams, and all unhealthy animals were culled. The remaining animals were assigned to cages, one per cage, until each cage contained three animals. Cages were then numbered and assigned to control and treated groups using a computer-generated randomization table. Rats were ear-clipped for individual identification.

All animals were housed in temperature- and humidity-controlled rooms. The temperature was maintained at 22°C with a range of 21-24°C, and the relative humidity was maintained at approximately 45%. The room air was changed 10 times per hour and was maintained under positive pressure to the access halls. Fluorescent lighting provided illumination 12 hours per day. Food and water were available ad libitum. Drinking water was softened, filtered, sterilized with ultraviolet light, and supplied by means of an automatic watering system.

Subchronic feeding studies were conducted with male and female Fischer 344 rats to estimate the maximum tolerated dose of Aroclor® 1254, on the basis of which low, mid, and high concentrations (hereinafter referred to as "low doses," "mid-doses," and "high doses") were determined for administration in the chronic studies. In the subchronic studies, Aroclor 1254 was added to feed at concentrations of 2%, 50, 100, 200, or 400 ppm. Treated and control groups each consisted of 15 male and 15 female rats. The chemical was provided in feed to the treated groups for 3 weeks.

The animals receiving 400 ppm were inactive, had occasional diarrhea and tremors, and failed to gain weight. At this dose 4/15 males and 1/15 females died. Enlarged livers were observed on gross examination, and histologically atypical hyperplasia was observed. At 200 ppm, body weights for both males and females were approximately 70% of those of the controls, and mild hepatocellular pleomorphism was seen histologically in the livers. Rats treated with 25 ppm Aroclor 1254 had enlarged livers, but no evidence of histologic abnormalities. Weight gain in all animals treated at doses lower than 200 ppm was comparable to that in controls, and there was no mortality below 400 ppm. The low, mid and high doses for the chronic studies were set at 25, 50, and 100 ppm.

All animals were observed daily for signs of toxicity and palpated for masses at each weighing. Animals were weighed individually every other week for 12 weeks, and once every fourth week for the remainder of the study. Animals that were moribund at the time of clinical examination were killed and necropsied.

The pathologic evaluation consisted of gross examination of major organs and tissues from killed animals and from animals found dead. The following tissues were routinely examined microscopically from both treated and control animals: lungs and bronchi, spleen, liver, testes, pituitary, kidney, and brain. In addition, sections of stomach, urinary bladder, thyroid, uterus, and ovary were examined in a majority of the controls. These tissues were taken from treated rats only if a lesion was found at necropsy. Occasionally, additional tissues were examined

microscopically. The different tissues were preserved in 10% buffered formalin, embedded in paraffin, sectioned, and stained with hematoxylin and eosin. Special staining techniques were utilized when indicated for more definitive diagnosis.

Beginning at about week 10 for the high-dose groups and about week 20 for the mid-dose groups, mean body weights of both male and female rats fed Aroclor® 1254 at the doses used in this bioassay were lower than those of the controls. Mean body weights of low-dose males appeared comparable to those of controls throughout the study, while mean body weights of low-dose females were lower during the second year of the study. At week 30, an intercurrent respiratory infection in the colony caused weight loss, but no deaths; animals recovered within 30 days without treatment for the infection.

Clinical signs associated with administration of Aroclor® 1254 included alopecia, amber-colored urine, facial edema, exophthalmos, and cyanosis. These signs were apparent among the high-dose groups beginning at week 72 and among the mid-dose groups at week 104 of the study.

A variety of neoplastic processes were observed in both the control and treated rats, and, with the exception of the liver, the incidences of these neoplasms were comparable in the control and treated groups. Interstitial-cell tumors of the testes were present in the majority of control and treated males. The next most frequently observed neoplasm was leukemia of either the granulocytic or lymphocytic type, and it involved multiple organs. The incidence of this neoplastic process was

comparable in the control and treated groups. The following neoplasms were also present in some control and treated rats but without compound association: squamous-cell carcinomas of the skin, alveolar/bronchiolar adenomas of the lung, and uterine endometrial stromal polyps.

In male rats, the results of the Cochran-Armitage test for positive dose-related trend in the incidences of leukemia and of combined leukemia and lymphoma are significant ($P = 0.022$ and $P = 0.009$, respectively). The corresponding results of the Fisher exact test, however, are not significant in any treated group when compared with the controls. There is no other incidence of tumors at any specific site in either sex which is statistically significant. A significant Cochran-Armitage trend in the negative direction is observed in the incidence of interstitial-cell tumor of the testis, where the incidence in the controls exceeds those in the mid- and high-dose groups.

At the doses used in this bioassay, Aroclor™ 1254 was toxic to both male and female Fischer 344 rats, as shown by the dose-related depression of mean body weights and the clinical signs which occurred during the second year. Mean body weights of mid- and high-dose males and of all treated females were consistently lower than those of the corresponding controls after the initial growth phase. An intercurrent respiratory infection at week 30 resulted in temporary weight loss, but no deaths, in all groups including the controls; the animals later recovered without treatment. Clinical signs including alopecia, amber-colored urine, facial edema, exophthalmos, and cyanosis occurred in the high-dose groups beginning at week 72 and in the mid-dose groups at

week 104. Survival among males, but not among females, showed a significant dose-related trend. Adequate numbers of animals of both sexes survived for meaningful statistical analyses of the incidences of tumors.

The combined incidences of lymphoma and leukemia in males were significant (controls 3/24, low-dose 2/24, mid-dose 5/24, high-dose 9/24, P = 0.009) using the Cochran-Armitage test for positive dose-related trend, but not in females (controls 4/24, low-dose 6/24, mid-dose 6/24, high-dose 6/24). Since the results of the Fisher exact test for increased incidence were not significant for any of these groups, the occurrence of these lesions cannot clearly be related to the administration of Aroclor® 1254.

Hepatocellular changes including hyperplastic nodules, adenomas, and carcinomas were found in treated animals, but none of these lesions were found in controls animals in this study. Hepatocellular carcinomas were observed in one mid-dose and two high-dose males, and hepatocellular adenomas were observed in one high-dose male, one mid-dose female, and two high-dose females. Nodular hyperplasia was diagnosed with a dose-related frequency in the low-, mid- and high-dose male and female rats. Although the incidences of the tumors were not significant, the occurrence of these proliferative lesions appeared to be related to treatment.

In the stomach, jejunum, or cecum, adenocarcinomas were observed in two treated males and in two treated females as well as a carcinoma in one treated male. None of these lesions was found in control

animals in this study, suggesting that the lesions -- although not statistically significant -- may be related to the administration of Aroclor™ 1254.

SARDINAS: Thank you, Dr. Weisburger. We'll now move on to the state panelists. The first panelist is Dr. Kenneth Dixon. Dr. Dixon is the Scientific Advisor from the American Chemical Society to Representative Toby Moffett's office. He is a member of the PCB Citizen Watchdog Committee. And he's from Southbury, Connecticut. Dr. Dixon.

DIXON: Thank you. I'd like to say first of all that any remarks I make should not be attributed to the American Chemical Society, nor are they the opinions of the Watchdog Committee. Question one is, reports indicate that different PCBs have different toxicities. Also, PCDF, which is present in PCBs is reported to be more toxic than PCBs. That's incidently is what Dr. Brown said. My question is, would it be more desirable to establish the nature of PCBs and the PCDF content of the PCBs in the fish which we collect before issuing any health warnings?

SARDINAS: Dr. Kolbye, would you like to start off with that?

KOLBYE: One brief comment. Most residue patterns in fish approximate Aroclor 1254 to 1260. That means that you're dealing with the more highly chlorinated isomers. And those are the ones that have the longer biological half-lives, the ones that are

KOLBYE: (Continued) not necessarily metabolized to its greater degree. The recent reports of the chlorinated dibenzofurans in fish reported by Dr. Stalling and his associates from Columbia, Missouri and the other cooperating centers, concern whole fish. Now, one of the things that we have perceived, from some studies in humans and in laboratory animals is that the dibenzofurans have a different solubility pattern than do the PCBs, or at least there are differences. The dibenzofurans appear to want to concentrate in liver. And since Dr. Stalling did his studies on the whole fish, one of the questions that I discussed with him yesterday is whether or not he would run some fish livers to see what the concentration was of the dibenzofurans. And then compare that with the filet portion of the fish which is what humans ingest. I think certainly if I had responsibility for giving specific advice to a specific locality, it would be helpful to know in general whether the dibenzofurans are concentrating in fish liver and whether or not they are present to a significant degree in the edible filet. Also, I think that in any public health provision one makes, one has to talk about educating people primarily not to consume contaminated fish or you're going to have to close your streams if that is the ultimate judgment that you make. There are always going to be people who fish whether the stream is closed or not; and one has to be mindful of educating the people as to what the situation is, and to speak primarily to those sport

KOLBYE: (Continued) fishing people who will go back to the same portion of the river.

SARDINAS: Anybody else on the panel like to comment on that? Dr. Dixon?

DIXON: May I answer that? I've asked Randy Most of the Connecticut Department of Health Services and the people associated with her, to perform the same analysis on the fish which she collects. If she doesn't do that, will she have the whole story? I'm asking that the State Health Department have the same kind of analysis made for PCB and PCDF that Dr. Stalling has. And before that analysis is made, should our State Health Department issue any directives or comments on the entire situation?

SARDINAS: Okay, that's been noted and we will at the State Health Department discuss that suggestion further with our laboratory people. Did you have a further question, Dr. Dixon?

DIXON: And now the second question is, some experts have calculated that PCB toxicity and carcinogenicity would represent a health hazard if one were to consume one half a pound of fish containing 5 parts per million everyday for 8 to 21 months. This is a shortened statement of what Dr. Lewis has said in a publication.

DIXON: (Continued) Now I'm saying, isn't this an unlikely exposure? Wouldn't the hazard be greatly reduced if the intake were cut in half for those eating that much fish?

SARDINAS: I think perhaps for the sake of the national panelists who probably haven't seen that publication, I should just briefly mention that these data were based on some extrapolations from data that was existing on PCBs where we tried to some extent to give a perspective on the relative risk of eating a quantity, having an extended exposure based on levels that were found in fish flesh and in fish skin. We basically produced a linear model that would extrapolate foward something about the levels of risk. And I guess what you're basically asking though, Dr. Dixon, is two things. First of all whether or not half a pound a day ingestion of fish is a reasonable expectation for the population over a long period of time and secondly, whether or not if half that quantity were consumed this would sign ficantly decrease the risk of our projected morbidity. I don't, know if any of the panelists would like to speak to that issue. Perhaps Dr. Lewis, would you like to make a brief statement.

LEWIS: Two things, first of all. the use of those particular estimates was totally arbitrary. We were trying to get in the range of at least the kind of quantities somebody might eat in

LEWIS: (Continued) a day. We were extrapolating it to the entire state population and counting it over a long period. Now, we were also making assumptions, possibly not warranted, about extrapolating this to people that eat less. But at least it gave us a standard of comparison. It is interesting, however, that this summer, as we have been going around getting blood samples and interviewing people that eat fish from the Housatonic River, that we found of the people that eat fish about a quarter of them are eating in the range of 3 to 6 pounds per month. So we're not all that far off.

DIXON: May I? My third question is very simple, and indicates my attitude towards this problem. Three, why not say that the intake of fish may be, "Hazardous to the Health." We recognize that the level of consumption would then be left to the consumer. That little notice along the Housatonic River should read "Hazardous to the Health" to eat these fish.

SARDINAS: We have duly noted those comments. Okay, our next panelist is Mr. Edward Kluck; he's the President of the Housatonic Fly Fishermen's Association, and a member of the PCB Citizen Watchdog Committee. Mr. Kluck?

KLUCK: Thank you. I suspect my role up here is partly to

KLUCK: (Continued) represent not only our own group of trout fishermen, but also the sport fishermen who use the entire watershed. And I had a two part question primarily relating to the proposed, and now postponed for a period of time, 2 part per million threshold. I'm wondering, and it's been answered to a degree, wondering what the rationale was for achieving this 2 part per million as opposed to the existing 5 part per million?

SARDINAS: Dr. Kolbye?

KOLBYE: On a broad scale, we've had approximately a decade, nationwide, of exposure to PCBs and probably longer than that. Look at the types of incidents that have occurred, such as recycling paper that has then become contaminated with PCBs and PCBs that found its way into animal feed. Some of it can cause migration of residues from paper board to human food. We've controlled that situation. We've controlled the situation of using heat exchangers, PCB heat exchange fluid. PCBs were used as silo sealants in many parts of the country for a while, and residues got into milk, into the silage and into dairy cattle. That situation is under reasonable control. So we come full circle. The only problem exposure, so to speak, is fish from certain geographic areas where there is a propensity for PCB residues. There's one other aspect that's been involved. We've been monitoring, and

KOLBYE: (Continued) so has EPA, the residues of PCBs in human breast milk. There has been some research with which I'm not completely comfortable that would seem to indicate the expected, that fetus and weening animals that are being nursed, have an exquisite sensitivity or may have a more exquisite sensitivity to PCBs than anything else therein. And so, what one ends up doing is drawing a bright line in a spectrum of grays and making a value judgment as to whether there is enough protection for the coming decade from the 5 part per million level versus bringing it down. The decision was to try to bring it down and 2 as the bright line was drawn. Now it's impossible to give you lucid explanations why it isn't 1½, why it isn't 3, it's ultimately a value judgment.

KLUCK: My question was not relative to the number chosen but to the tests and results which have related to that number at this point. The other half of my question deals with the assumption that at one point in the ball game 2 ppm will be the minimum. In the past of the various fresh water species that have been tested in the Housatonic watershed, pretty much every species has shown itself at or above the 2 ppm. Striped bass that have been previously tested have just been marginally below 2 ppm. I want to know what effect this 2 ppm will have on health activities, fisheries management, further investigatory activities,

KLUCK: (Continued) public education, etc., along the entire Housatonic watershed and the Sound.

SARDINAS: As Dr. Kolbye mentioned earlier, the Food and Drug Administration is primarily concerned with interstate transport of these materials and I guess what you're really asking is for an intrastate consideration of these materials.

LEWIS: I think so far we've been fairly consistent with the old standard of 5 parts per million and I don't anticipate any change just because the standard changes to 2 parts per million. Dr. Kolbye pointed out correctly that the FDA is looking primarily at interstate. They're also looking primarily at commercial operations. In the Housatonic River we're talking about sports fishing and private collection and consumption of fish. FDA certainly isn't going to regulate this. We don't have any regulatory powers. It's not really a question of imposing any standard. I think the promulgation of a standard is irrelevant to our policy and what we tell the public, with the possible exception that it does at least give a rough ballpark figure upon which to hang our hat. What we're anticipating doing with the 2 parts per million standard is simply considering the areas of the river that we post. We also are considering warning about salt water fish consumption such as striped bass, which may fall into this 2 parts per million

LEWIS: (Continued) standard. This is in no way a prohibition. The only thing we're doing and it's not effected by the standard, is trying to inform the public to the best of our knowledge, what risk they're taking. We're not in any way restricting them, or forcing any prohibition on fishing. Our main goal is to put whatever risk may be involved in context with other risks we're exposed to. Now, there's one other area that you sort of touched on and it's really within DEP's responsibility rather than ours, and that is the question of whether it's going to be state policy to stock the Housatonic River with trout. That's quite a different question. I don't see how our health policy has any effect on that whatever since presumably the stocking of trout has nothing to do with treating the river as a food source. It has to do with sports fishing. And again, I think the question of sports fishing versus eating fish is not necessarily related, since sports fishermen presumably are fishing for the pleasure of catching fish, not necessarily for the pleasure of eating fish. I think they're not necessarily related.

SARDINAS: Our next panelist is Dr. Armond Oppenheimer. Dr. Oppenheimer is the Conservation Chairman of the Housatonic Audubon Society, is a Professor at Columbia University, and a Fellow of the American Association for the Advancement of Science. Dr. Oppenheimer is also a member of our PCB Citizens Watchdog

SARDINAS: (Continued) Committee. And he's from Lakeville, Connecticut. Dr. Oppenheimer.

OPPENHEIMER: In view of the fact that the PCBs are found all over this planet in the polar regions and every lake and every stream in our country and in, certainly in the northern hemisphere, and since the question of the carcinogenic, mutagenic, reproductive damage and other toxic effects of PCBs with their contaminant, dibenzofural, are paramount to a national health policy program. And since the additive affects of PCBs together with other environmenta toxins are of paramount importance in consideration of the PCB problem, and finally, since PCBs enter the environment through groundwater, through evaporative processes, into the environment, into the air, they're deposited on the soil, and perhaps they contaminate our vegetable, fruit products, I'd like to address a question to Dr. Weisburger. Namely, since Aroclor 1254 is only one of a number of PCB compounds, and since the additive effects of PCBs and many other environmental toxins are not taken into account in this research, how conclusive is this experiment, and can we take any comfort in the negative findings?

WEISBURGER: I would say that we cannot take any comfort in the negative findings because we know from many animal experiments that there's a great difference in the strain response to known

WEISBURGER: (Continued) carcinogens. There can be variation with some very potent carcinogens from 0 to 100% tumor incidence. So I would agree that we cannot take comfort from this negative study. We don't know, in fact there is one other study in the Sherman strain rat with Aroclor 1260 where it was reported that there were tumors were obtained.

OPPENHEIMER: The only other aspect of it was the question of the accumulation of PCBs over a lifetime, and their interaction with other environmental toxins, as a very important consideration in assessing the pathological threat to human health.

WEISBURGER: Unfortunately, there has not been enough laboratory experiments to make any statements. It takes a great deal of time and effort to do a single study. And to do this properly one must have to test a PCB with many other environmental chemicals and that just has not been done.

SARDINAS: Dr. Kolbye, I think you wanted to make a statement.

KOLBYE: Well, perhaps just a brief comment. One of the main effects of the PCBs depending upon degree and position of chlorination, seems to be related to inducing or inhibiting various enzymes like the mixed function oxidase enzymes. It's of interest

KOLBYE: (Continued) though, that there are many naturally occurring substances in our diet that have similar properties. When we review the PCB toxicity and evaluate the various data in terms of arriving at some decision with respect to a recommended action level, we try to take into account not only the direct evidence, but anything that we know or suspect would be interacting. But there is a limit to how far one goes in that. And I suppose I should also say that while it is fashionable and political to say that there is no such thing as a safe exposure to any carcinogen, I think it is fair to say that the greater the exposure to a carcinogen, the greater the risk, and the lower the exposure, the lower the risk. And from the viewpoint of societal decision making, one has to draw a line someplace.

SARDINAS: Thank you, Dr. Kolbye.

SARDINAS: Dr. Brown.

BROWN: Yes. There has been a little work reported in the literature on the supplemental effects of PCBs with some environmental carcinogens. And the results are rather peculiar. In the case of, is worked on by the Japanese, in the case of polycyclic aromatic hydrocarbons it has been found that simultaneous administration of PCB decreased the incidence of tumors produced by the other

BROWN: (Continued) carcinogen. There is one of the chlorinated pesticides in which this effect was not observed. I don't remember which one I think it was heptachlos.

SARDINAS: So you're suggesting that in the literature, at least, there is a protective effect of PCBs?

BROWN: Yes, this would be as expected from Dr. Kolbye's remarks. That its primary effect is as an enzyme enducer. And in high doses it's going to simply change the metabolism of everything in your body.

SARDINAS: Sure. Thank you, Dr. Brown.

OPPENHEIMER: Would Dr. Brown suggest that we take a small capsule of PCBs with our vitamins? In as much as PCB contamination poses a national health problem, and since scientific and professional groups in many areas are engaged in research and technology aimed at finding solutions to the many problems related to PCBs, would it not be a great saving in time and money, and avoid duplication if the EPA set up a central office to collect, digest, evaluate, and promptly distribute information to the proper departments of the several states. As I say, this is not a debatable question, it is offered more as a suggestion.

SARDINAS: Thank you, Dr. Oppenheimer. Did you want to comment?

BROWN: Yes, that's a very interesting question and suggestion. Historically in studying any question of environmental pollution the three R's of environmental science are rates, routes and reservoirs. Better trace the flow of the pollutant. EPA as a whole has been very uninterested in these questions. The people who have studied PCB transport around the environment, and have done the most impressive jobs have been other state and regional groups, particularly in Michigan, Wisconsin, southern California, Chesapeake Bay area. Ian Nesbit of the Massachusetts Audubon Society has been very much concerned with the global flow. This is because in most cases when you get down to the problem of what do you do with a specific pond, river or lake, it is a local problem.

SARDINAS: Thank you, Dr. Brown. We have with us substituting for Mr. Melvin Schneidermeyer, Tom Turick, of the Department of Environmental Protection here at the State of Connecticut. Tom?

TURICK: Thank you, Mr. Moderator. My questions are of a general nature and they fall into three specific areas, and that is the health effects of PCBs through, number one, occupational exposure, number two, the consumption of contaminated fish; and

TURICK: (Continued) number three, possible health effects of PCBs in drinking water. My questions are addressed at no specific individual but rather at the panel in general. Dr. Brown, if I heard him correctly, today stated that the, the risks from occupational exposure to PCB could be as much as 100 times greater than the risk from consuming fish that have low levels of PCB in their tissues. I'm wondering what the panel's thoughts are on occupational risks as related to fish consumption risks. And would it be true then that, if there is to be a break through as far as cancer risks from PCB, would it not come about through research into occupational exposures versus recreational fishermen research?

BROWN: We think so. And there are some good model systems available in several populations of capacitor workers, both in the United States and Japan. These people were very heavily exposed for periods up to 30 to 40 years before the PCB use was terminated in 1977. And there are a number of studies of such populations going on. Some results have been reported already, and some are continuing.

SARDINAS: Dr. Weisburger?

WEISBURGER: It's usually thought that a period of 20 to 30 years of human exposure is equivalent to the type of experiment

WEISBURGER: (Continued) we do in rats, and therefore, [redacted] such a [redacted] chemical probably does not have an effect on humans.

SARDINAS: Did you want to comment on that Dr. Douglas?

DOUGLAS: Yes, just to say that Dr. Fisenbeth and the group in New York have done some fairly nice work in looking at people occupationally exposed for periods of longer than 15 years. [redacted] as I recall in one of their papers they state that there are remarkably few findings in these individuals that that's rather significant.

SARDINAS: Tom?

TURICK: My next question, I'm wondering do we know enough about low level exposure through consumption of fish to move towards, as far as our fish warnings on the Housatonic, a warning system, that for instance, Canada has employed. Canada's warning system is a very specific one, which recommends against eating certain size fish, and type of fish in certain areas of the river. Their system contains such parameters as the portion of fish per week that could be consumed. And also, I believe, it brings in the

TURICK: (Continued) population as a parameter. It warns specifically to certain segments of the population, such as pregnant women, and children. I'm wondering do we know enough about low level exposure through fish consumption that Connecticut could be moving towards this kind of warning?

SARDINAS: Dr. Kolbye?

KOLBYE: Well, I think one could set some priorities on the message that one conveys to the populace. Other states have faced this problem and have recommendations that in some instances appear right on the fishing license. I would recommend that this approach be considered by the State of Connecticut. It's really a matter for your own review and recommendations as to which warnings you think are most appropriate, or which cautions or recommendations. Just as an example, I think some of the authorities in the United States, state authorities have said not more than one meal of fish per week and from certain sources, not to be consumed by women in the child bearing years.

SARDINAS: Tom?

TURICK: My third question, at this time the state has no reason to suspect that we have any type of problem with PCBs in

TURICK: (Continued) drinking water in the Housatonic River basin. However, in New York, if my information is correct, communities do utilize the water from the Hudson River. They have been finding I believe up to 3 parts per billion in the drinking water supply. They will spend millions of dollars for treatment plants to try to reduce that level to 1.5 parts per billion. I'm wondering what is known about the health effects of drinking water contaminated with PCB in the range of 1 to say, 5 parts per billion?

KOLBYE: I don't think there are any data at that low level. And one only can make projections.

SARDINAS: Thank you. is Dr. John N. Lewis who's our next panelist, is the Chief of the Bureau of Health Promotion and Disease Prevention for the Connecticut State Department of Health Services. He is also our state epidemiologist. And I'll ask Dr. Lewis for his questions at this time.

LEWIS: Thank you, Tony. I basically have more comments. than questions, and they're mainly directed at Dr. Brown's talk. First of all it's been the State Department of Health's policy as regards PCB exposure in Connecticut to, in agreement with Dr. Brown's comments about the relatively low toxicity and the

LEWIS: (Continued) relatively low risk to the general population, to deemphasize the toxicity of PCBs. In fact, in Connecticut, I think in terms of overall risks to humans, in comparing it to many other things it is a very low order of magnitude. In fact, we are only concerned in considering toxicity of PCBs, primarily with people who fish in the Housatonic River and eat the fish. And that's a fairly small part of the population. But as regards that group we have considered it important to put whatever risk they may have in context. Some comments related to extrapolating the animal data to human data, and in fact, Dr. Brown said there was no evidence of cancer in monkeys or man, related to PCBs. I think that statement is somewhat deceptive. First of all, humans are not experimental animals. And the kind of data that we have collected on rats is simply not available on humans. Second, monkeys which would be a good species to use theoretically, are not available for the type of experiments that were described by Dr. Weisburger, because they're very expensive and you can't do experiments on large numbers in the way you can with rats. So we have to rely on the rat experiments. However, in addition to that, I believe there is some data in man, albeit not laboratory data, but rather epidemiologic data, that does suggest cancer and liver damage in humans. And this includes, first of all, extensive data of elevation of liver enzymes in people exposed both in this country and in Japan to PCBs. And second, some

LEWIS: (Continued) evidence, fairly weak, of an excess of cancer among some [redacted] with PCB[redacted]. Now this is inconclusive evidence, and it's certainly true that we don't have any final conclusions on this. On the other hand, I'm not aware of any very solid human epidemological data in the literature that suggests that it's not cancer causing in humans, so it's really, I think, just left in the unanswered category. And that's exactly the reason that we feel it's important to warn people about it. We have published, or at least given the press and the PCB Watchdog Committee, our estimates of just what order of risk we're talking about. And we did use figures like people in Connecticut, everyone in Connecticut eating a half a pound a day for a year of trout from the Housatonic, obviously not a situation that's likely to occur. But at least it gave us a standard of comparison, and one of the comparisons we came up with is that people eating that much fish in Connecticut would produce in our estimate, granted a crude estimate, [redacted] deaths in Connecticut. Now, that's a very small part of the deaths in Connecticut, drowning. We would estimate further that if people eat less fish the risks would be less than that of drowning. We're going to be making more estimates like this in the future to put the various substances that are not known to cause cancer in context. This, for example,

LEWIS: (Continued) is [redacted] It's a very low risk compared to the risk of working with asbestos, or working with radiation. Or a variety of other things, or the risk of not wearing your seatbelt, or the risk of eating certain fats that have a high fat content. So that, to put it in context, it is a very small risk. It is a real risk, however, and we feel that the, the standards that are being used by FDA give us a good cutoff point to inform the public that they are taking a risk when they eat fish from the Housatonic River. Some comments were made about fishing, and if you listened at what everybody says about PCBs, you would want to give up fishing. I don't believe that follows, because most fishermen I think get their rewards not out of eating the fish, but rather out of the sport of catching fish. We certainly have no warnings at all about catching fish and throwing them back. Some things have been said about standards. It's my understanding as regards standards, that standards are based primarily on toxicity other than cancer. I think we haven't heard any disagreement with the concept that with a cancer causing substance, even a very small exposure might confer a small risk. And it's for this reason that we are warning about all fish that have PCB in them, without real regard for the standard, because we think there is some risk at any level of PCB consumption. Until we know for a fact that PCBs don't cause cancer, I think we have to make that

LEWIS: (Continued) kind of warning. My comments are mainly directed as criticisms of Dr. Brown's speech, and perhaps you'd like to comment.

BROWN: Yes. On this first point, I don't think we are in as much a disagreement as it might sound. I said that in rats or mice the primary target of attack by the toxic forms of PCBs and the dibenzofurans and dioxins, appears to be the liver. And the characteristic syndrome produced by all of these agents is a progression, as Dr. Weisburger has indicated, starting out with enzyme induction, hypertrophy, adenofibrosis, and eventually hepatocellular carcinoma. This entire progression, culminating in liver cancer does not occur in guinea pigs, monkeys or man. As regards other types of cancer, the report that Dr. Lewis cited is of two refinery workers who were exposed to a large number of agents at Atlantic-Richfield, and eventually developed melanomas. The identification of the agent I think is still ambiguous, and certainly melanomas have not been associated with PCB exposures in any other case. With respect to the industrial workers who have had heavy PCB exposures for many years, the existing studies I might say, would probably be best described as superficial. The local medical people have looked at those groups, have eyeballed the data, and have said, yes, there are no gross effects, but the high quality matched cohort pair of study required to find out whether there

BROWN: (Continued) is a small incidence has not yet been done. However, I would still like to emphasize one point that you have made. These studies have all involved electrical workers who worked with highly purified PCBs. The PCB in the Hudson is not all high purity electrical grade. It has apparently come from a variety of sources. I certainly would have no idea what the toxicological quality of that particular PCB is. I think any health authority would have to act in a conservative manner until that determination had been made.

SARDINAS: Dr. Weisburger?

WEISBURGER: I'd just like to ask Dr. Lewis about these melanomas in the refinery workers, if he could tell me, did these people work in a southern location?

LEWIS: I'm afraid I don't know any more about that study. A warning came out of the National Institute of Occupational Safety and Health about three years ago. I never saw any follow up material on it.

WEISBURGER: Because melanoma is also associated with excessive exposure to sunlight. And if they worked in a southern location, and were outside all day, this would also be a factor to be considered.

LEWIS: Yes, I'm not even sure we're talking about the same study because my memory of this rough study was that it was Mobil rather than Atlantic-Richfield...

BROWN: Excuse me, you're right, I'll withdraw my comment.

LEWIS: My further memory of it was that it involved more than just melanoma, namely leukemia. But again, it was a very crude study and I'm just trying to point out that the really elegant studies that could demonstrate this epidemiologically just haven't been done. Nobody's going to hang their hat solidly on a slight excess cancer in some refinery workers who obviously are exposed to other substances, but, as you say, you have to act conservatively when you don't have enough information. And I think our responsibility is to err on the side of warning the public as opposed to not saying anything about it.

SARDINAS: Dr. Douglas, you had a comment?

DOUGLAS: Yes, I'd like to clarify one thing, with regard to the liver damage and the number of comments about the induction of liver enzymes. I just want to make it clear that it is a rather general phenomenon, and not something specifically associated

DOUGLAS: (Continued) with PCBs. Many agents which we use medicinally induce liver enzymes, so that in itself, whether it's bad or good, nobody really knows. It doesn't seem to bother people or animals in any way and is quite a reversible process. The concern about the induction of liver enzymes is that because these enzymes are more active, they may then convert other materials which we ingest, for example, nitrates, into carcinogenic agents in a more vigorous way than if we had not been exposed to PCBs and increased liver enzymes. There is evidence in the literature to suggest that exposure of humans to PCBs does produce liver enzymes. I'm not sure that there is any evidence in the literature to suggest that there is additional evidence of liver damage as is seen in the rat model.

SARDINAS: Thank you, Dr. Douglas. Dr. Kolbye, you had a comment earlier.

KOLBYE: I believe that it might be helpful to phrase estimates of risk as maximum theoretical risks from linear extrapolation. In other words, it's a very conservative approach, and probably, I should think, over estimating risk.

SARDINAS: Okay, thank you. John?

LEWIS: Just one other very brief question. The point has been made several times about the contamination of PCBs with dibenzofurans, and how that may in fact be the more important toxic principle. We've been assuming that consumption of pure PCBs would not produce exposure to dibenzofurans. On the other hand, there's the possibility that the PCBs, pure PCBs may be metabolized as dibenzofurans. I wonder if Dr. Weisburger could comment on whether humans may actually metabolize to the other substances, or whether other species such as fish, might do that?

WEISBURGER: I do not know of any studies where this has been proven.

SARDINAS: Dr. Kolbye?

KOLBYE: The information available to me, if I'm recalling it correctly, is that while with certain isomers you may get an apoxide formation of a PCB which may have electrophilic properties, and may be associated with a possible carcinogenicity. That the human metabolism of a PCB to a dibenzofuran would be rather unlikely, although theoretically possible, it would be unlikely.

SARDINAS: Well, at this point then, let me open up for questions from the audience.

MALISH: My name is Steven Malish. I'm section chief of the Toxic Hazardous Section of the State Department of Health Services. The question I have is directed to Dr. Weisburger. I've heard epidemiological evidence and toxicological evidence, but I'm still not quite sure what the effect of PCBs are on man. From animal studies it would appear that an impure grade or a grade of PCB of unknown purity, was used. Moreover, the toxicological test laboratories have recently come under fire because of shody testing procedures. Therefore, do we have any conclusive proof that pure PCBs or at least PCBs that impurities have been characterized do cause cancer in animals? Obviously we do not have any epidemiology data to this effect right now.

SARDINAS: Dr. Weisburger?

WEISBURGER: We have not done any tests on a pure PCB, containing only PCB and none of the impurities, or a PCB which has been definitively characterized. I would like to comment, however, on the remark you made about the quality of the work and the toxicology and the pathology and so forth. All the tests which were done were performed under NCI contracts. If a report is published it's a sign that the pathology has been reviewed by both NCI and outside pathologists. The statistics such as for the animal handling

WEISBURGER: (Continued) and all that, have been reviewed and we have fairly good assurance that the work is of good quality. In other words, once a report appears it's very definitive.

MALISH: I certainly hope this is true, because we don't have definitive toxicological information, and people are basing their risk estimates on faulty data. (INAUDIBLE)

SARDINAS: Thank you, Dr. Malish.

HORNBECKER: My name is Joyce Hornbecker, I'm chairman of the Conference of Lake Authorities, vice chairman of the Lake Zoar Authority. I'm from Southbury, Connecticut. I would like to make a comment. I'm actually very aware of the people who are eating the fish. First of all the majority of the people in my area don't throw the fish back. I'm talking about ethnic groups that live in Southbury and in the area. People eat the fish for two reasons; it's a free meal, and for the ethnic groups in particular, fish is a very large portion of the diet. I'm very concerned about these people who are eating high concentrations of fish. When a black man from Waterbury, for instance, catches a fish, he doesn't know whether the concentration is 2 parts or 20 parts per million. The testing that the Lake Zoar Authority did initially indicated that some of our fish were 20, 22 parts

HORNBECKER: (Continued) per million. So we're talking about a variable. Also, the people who come to Lake Zoar and Lake Lillinonah to fish are people from Waterbury, from New Haven. A lot of them are black people. The only reason I'm making a point of this is that they eat the bottom feeders, such as bullheads. These people have the higher concentrations of PCB. I would like to see emphasis put on ways to cook fish that might reduce the toxic effects. Is it true that it tends to settle in the skin? If they remove the skin would they be less apt to become contaminated or not contaminated as highly? Is PCB in the head and in the liver? Should we say don't make fish chowders using the head and so forth? If they remove the skin, if they remove the head, if they remove the intestines and make a filet, is there less contamination? I would like to request that this kind of information be made available to these people. Thank you.

SARDINAS: Dr. Kolbye?

KOLBYE: I'd just like to mention that if, if one filets a fish, takes off the skin and broils it in such a way so that the drippings move away from the flesh of the fish, you can reduce your PCB intake considerably.

SARDINAS: Thank you, Dr. Kolbye. Next.

BANDOLIN: My name is Larry Bandolin, I'm a fisheries biologist at the State of Connecticut, Department of Environmental Protection. I have a statement and a question. Tom Turick mentioned that Canada has been quite specific as to their warnings about eating fish caught in Canada. New York State has taken the opposite tact. In their fishing syllabus which is a listing of the laws of the New York State which every fisherman gets when he gets a license, they have stated, and I am paraphrasing as I do not have that brochure with me, that you should not eat more than one-half pound of fish from any waters of the State of New York. My question is, an extensive effort was undertaken this year to collect fish from the Housatonic River in Connecticut, both from the Massachusetts border right down to the lower sections of the river. These fish have not been analyzed yet for PCBs. Would it be wise to analyze these fish for both PCBs and dibenzofurens?

SARDINAS: Dr. Kolbye?

KOLBYE: Just simply to mention that dibenzofuran analysis is tricky. And the analytical sensitivity is not what it should be. And it's a tough method as I'm sure you may well be aware. Earlier I mentioned that I would be interested in somebody looking at fish liver, because I suspected it may be concentrated there.

KOLBYE: (Continued) I think it would be worth doing.

BANDOLIN: The fish have been cleaned and are now fillets, some with skin on, some with skin off, depending on how the fishermen would be eating that particular fish. They would not have their livers any longer.

KOLBYE: Well, it might be of interest to take a couple fflets where you know they came from a high PCB fish, and try it on one or two and see what you find.

BANDOLIN: Thank you.

SARDINAS: Are there any other questions from the audience?

COLVIN: My name is Frank Colvin, I represent the Bristol Fish and Game Club. We have approximately 325 members. I'm a member of the PCB Watchdog Committee, and also a member and a commissioner of the Bristol Water Department. My question is a question that has been asked me many times from some of our members who fish the Housatonic exclusively: How long does it take a stocked fish, say, in April, before the season starts, to consume PCBs from the water? Can a person catch a fish within a week and feel free that he's not eating a fish with PCB content?

KLUCK: New York State a year or two ago ran a test and showed within two or three weeks they were approaching their maximums, if I'm not mistaken.

BROWN: Yes, I'd emphasize that fish pick up PCBs mainly through the gills, partly through feeding, in studies that have been done in the past, they reach an equilibrium level within about 30 days. However, the total PCB content will vary with the fat content of the fish. And as the fish gets older and the fat content gets higher, the total PCB will be higher.

COLVIN: Thank you very much.

SARDINAS: Are there any other questions from the audience? back and forth.

FOSTER: My name is Joanne Foster, and I'm a lay person with the Southington Citizens Action Group. Recently we have discovered that we had had contaminated public water supply. This is my first exposure with the PCB incidence. I'm a little confused as to the purpose of the meeting here today. Is it to clarify if there is a potential danger with the consumption of PCBs? Or, is there not enough evidence to warrant that kind of a warning?

FOSTER: (Continued) I'm getting the impression that it's almost a choose your poison attitude. But if that's the case. I would like to be informed and to have the right to choose my poison, whether it be cigarettes, my water, or what it is. Is that the basic issue here today?

SARDINAS: The purpose of today's meeting was to discuss the public health issues, and the health effects of PCBs and to help the State of Connecticut make determinations regarding, informing the public and any continued action that we may take in the future. We're looking really to get as much informed, scientific information from national experts regarding PCBs and their health effects as possible.

FOSTER: So, the purpose eventually will be to make an informed decision?

SARDINAS: Absolutely, well, of course, we've made a decision already, and we have been trying to educate the public with regard to the information and data that we have currently. The purpose of today's meeting is to get some national opinions from experts regarding PCBs as a potential health problem so that we can make a better informed choice as time goes on with regard to informing the public.

FOSTER: Okay, thank you.

SARDINAS: John?

LEWIS: I'd like to just add to that a comment a small note about the study already going on in Connecticut, because I don't think it was mentioned specifically today. In addition to keeping an eye on what's happening nationally and in the world, from the point of view of opinions and data on PCB, we're doing a very specific study in Connecticut comparing people who are eating fish from the Housatonic River with the control groups who are not eating fish from the Housatonic River. We are evaluating what levels of PCB they may have in their bodies, and whether there's any evidence of specific physiological effects, or even disease effects from this exposure. So, although that's not apt to show anything very dramatic, because of the relatively low exposures in Connecticut, at least it may have some specific value to people in Connecticut.

DIXON: I hope that most of you have seen this bulletin, "PCBs and the Housatonic River," prepared by the Department of Environmental Protection, with some assistance from the Watchdog Committee. There are two statements I think should be read with

DIXON: (continued) caution -- that PCBs are known to cause cancer in laboratory animals. As I hear Dr. Weisburger, I don't think she said that. The next statement, should I eat fish from the Housatonic? No. The State Department of Health Services recommends that fish from the Housatonic not be eaten. I hope they aren't saying that.

SARDINAS: This is Randy Most. Randy is an epidemiologist with the Connecticut State Health Services, and has been very much involved with the study that Dr. Lewis mentioned earlier to look at some of the health effects of folks eating fish from the Housatonic.

MOST: I just would like to point out although Dr. Weisburger's study that she was discussing did not seem to show that cancer was formed in the animals, there have been a large number of studies done by other groups, both federal, private, in Japan, using various types of PCBs on rats, guinea pigs, mice, and a number of other studies that have been done that have definitely showed that this chemical produces cancer in animals. Now, I'm not stating which studies are good or bad, or making any value judgments, but the comment that Dr. Dixon just made about that statement is based on other studies that have been done and not

MOST: (Continued) Dr. Weisburger.

SARDINAS: Okay. State your name, please, who you represent.

SALCIUS: John Salcius I'm a sanitarian from the Manchester Health Department. My question is is there a reversible process in the body once you eat the i.e., PCBs, does the body rid of it? Or is it a culminative thing where you eat a little bit and it just keeps accumulating?

SARDINAS: Dr. Kolbye?

KOLBYE: It has a long biological half-life. And the biological half-life increases with the increasing degree of chlorination. You're talking about years. There is some depletion. There is some metabolism. What will happen is that some of the isomers will dechlorinate, and you'll get lower chlorinated isomers emerging and the lower the chlorination, the more quick the excretion from the mammalian body. But I wouldn't want to leave you with the impression that it's quick. You're talking about a long biological half-life generally speaking.

SALCIUS: Alright, say you ate a great deal of it, you had five, six pounds of fish, and you got PCB in your body. Would

SALCIUS: (Continued) the effect be the same thing as eating the small amount over a long period of time? I guess it would.

KOLBYE: Well, there are several implications to the statement that you just made. In terms of the residue level that you experience and the distribution in your body, they're likely to be different, a pulse exposure as compared to a low level exposure. They're not mathematically equivalent in terms of the absorption and the distribution and the compartments of the body which will have the residues; toxicity-wise the implications would be different. I hope that answers your question.

BROWN: I'd like to comment on that, because in the older literature, where people were exposed to contaminated PCBs there is data on what happened to them. For these people who got relatively low level exposures, going on chronically, generally after some months the chloracne syndrome came on. They developed facial acne and they felt awful, just felt sick and tired and headachey. When the exposure was removed, the symptoms generally disappeared over a period of months. I would anticipate that if you were able conceivably to eat that much PCB you would see the same symptoms.

SARDINAS: Dr. Kolbye?

KOLBYE: I'd just like to emphasize for the record, I believe Dr. Brown is referring to the Yusho patients in Japan.

BROWN: No, I am referring primarily to the American cases reported of industrial workers exposed in the 1930's. 40's.

KOLBYE: I see. I thought you were using the term in terms of ingested PCBs, that's why, I'm unaware of any human sickness in the United States that is related to the ingestion of PCBs.

SARDINAS: Dr. Douglas, you've done rather extensive research and have reviewed the literature, would you like to comment on that?

DOUGLAS: I'll just say that PCBs stay around for a very long time, and I think if you look at the people who had the Yusho exposures there are a number of years after their exposure where the symptoms and conditions still remain. So there is a concern that once these manifestations show themselves that they may well be around for a long time. But I am also unaware of any information in regard to U.S. exposures in that regard.

SARDINAS: Thank you.

SALCIUS: My final question. Is it through eating that you, you become exposed to PCB or can you contract it through breathing and contact?

SARDINAS: Dr. Kolbye?

KOLBYE: You can be exposed to it in a variety of ways theoretically. But the main human exposure in the United States comes from ingestion of food, primarily fish.

SALCIUS: Okay.

BLACK: I'm J. Stanley Black, I'm from New Haven. I'm an environmental sociologist with Yale University. I'm specifically referring to a number of comments both by the National panel and the State panel related to the quality of the various studies that have been done, both laboratory and epidemiological studies. There seems to be a fair agreement that truly elegant studies have in general not been done. There have been few or no primate studies and sophisticated epidemiological studies have not in general been done. Apparently some of the laboratory studies have not been of the kind of quality and size to be really definitive. I'm not certain whether anyone made the point as to whether or not there have been studies of metabolism of PCB in humans. But

BLACK: (Continued) in general, I'd like to ask whether, we can expect a continuation of the basing of public policy on research which apparently there is some agreement, is lacking in definitive results.

SARDINAS: Want to start, Dr. Kolbye?

KOLBYE: Yes, I don't think I would accept in my own mind a characterization that the available scientific evidence is tainted or grossly inadequate. If one has in mind one particular commercial laboratory, there were some studies conducted in days earlier, connected with PCBs where the reliability of that particular laboratory has been questioned. That's one issue. But in terms of the other data available from a variety of laboratories, I would say that the data that are available are reliable and are untainted. Then the question evolves, what additional tests should be done. One can evaluate whether or not to conduct more lifetime feeding studies in animals. If such are conducted, one should have a starting knowledge of what contaminants are also present, like what level dibenzofurans. From the epidemiological viewpoint, there is a study in part financed by FDA of sport fishing people in the state of Michigan. The study is attempting to get some idea of what their exposure is and to monitor to a degree that population over a longer period of time. Now the Michigan situation got further

KOLBYE: (Continued) clouded because of the polybrominatedbyfenyl incident. Anybody who would like further information on the Michigan study could contact the Michigan state health department.

WEISBURGER: I would like to make a comment too. The bioassay program of the National Cancer Institute is set up largely to test materials that people are exposed to. And that doesn't mean then, since people are not exposed to pure PCBs except in special cases, that's why we tested a commerical available aroclor with the resultant impurities. But the results show there's something there and it does give an indication that perhaps further stucies should be done.

SARDINAS: Dr. Douglas and then Dr. Lewis.

BLACK: Excuse me, I'd like to respond to that. I was not by any means complaining that a commercial preparation had been used as the test subject. It just seems to me that that's the only logical thing to do, since that's what humans will be exposed to.

SARDINAS: Dr. Douglas?

DOUGLAS: Unfortunately that's not often the logical thing

DOUGLAS: (Continued) to do as a scientist. As a scientist, we try to control as many of the variables as possible. And that necessarily means that our results do not always conform to the environmental types of exposure. I think that is the reason we express reservations about the scientific results, not because of the conduct of the experiments themselves, but because we are not sure how to interpret those experiments in the real world. That is a difficulty which we, as toxicologists, always face. We never are able to do the right experiment. It's for this reason that the standards which we set and we arrive at, are largely intuitive. They are not handed out in black and white as a result of experiments. They are the result of weighing a variety of situations. I do want to say also that one of the areas where one can get considerable information about toxicology in man is occupational exposures. Dr. Fishbein and his group, Dr. Selikoff and others in New York have done a number of excellent epidemiological studies of people who were exposed in Michigan, for example, in some capacitor factories. I reiterate again their comment was that the findings were remarkably few. And these are people who are exposed for long periods of time to quite high levels of PCBs. I think that those results are very significant.

BLACK: Just one question with regard to those sorts of studies. Dr. Brown suggested earlier that these studies relate to exposure

BLACK: (Continued) to the very pure PCBs as might have been the exposures in occupational cases. What I was given to understand from his earlier comment was that the, the actual environmental contaminants in say, the Hudson River, for example, were much, were likely to be much less pure PCB contaminant.

SARDINAS: I'm going to let Dr. Lewis respond first.

LEWIS: Yes, your original question related to public policy, and implied that public policy decisions should await more definitive answers. In this sense I would think that here at the State Health Department at least, we don't get into public policy at all because we really function more as a conduit of information then we do of setting policy. At least in this kind of area. I think in almost all of the areas where we're talking about carcinogens or suspected carcinogens in the environment, and where people are exposed to them, we're dealing with incomplete information. Our role is not to forbid people or place harsh restrictions on what happers when they're exposured to these substances, but rather to inform them, and to certainly err on the side of informing them about the risks as opposed to trying to make things look safer then they may prove to be.

BROWN: I would like to comment on the quality of the scientific

BROWN: (Continued) data available. As I said, this is an area in which there is an enormous amount of information available. There are really hundreds of papers out there. Obviously they run the complete spectrum of quality from outstanding to pretty dubious. It's also true that the questions that the investigators had in mind when they did the study are not necessarily the same as a public health authority might wish that he had in mind when he reads it. This is why I think it's quite important to go back to the original literature to find out what it said, and to compare observations from different sources. The importance of purity effects, of course, becomes evident when you make quantitative comparisons of the results of different investigators. In the case of PCB effects on monkeys, for example, successive reports out of the same group showed levels of toxicity varied by a factor of 100. This, of course, leads you to be a little bit concerned as to what they are looking for, but this is the type of thing that I think any review of the literature has to focus on.

MALISH: Steven Malish, Department of Health Services. What is the half-life of PCB in man? And if in fact it has a long half-life, wouldn't this mean that any additional exposure would build up the body burden of this substance in the body?

KOLBYE: Well, I don't know that there's a good precise answer

KOLBYE: (Continued) time-wise to your question, because one would have to in order to be accurate, specify what degree of chlorination you're talking about. Depending upon the rate of entry and the rate of excretion, you will hit a level where you should be in a degree of equilibrium. But there's no direct good answer to your question except to say that the more you're exposed to the higher the likelihood that you will be accumulating some of the residues in your body fat. The less that you're exposed to, the less likely. Whatever you are exposed to, and you do accumulate in your body fat, you will likely have there for a period of years.

MALISH: Thank you.

BROWN: Could I add to that comment? This is a question that has been on several people's minds as they review the human data. In the case of capacitor workers, it looks as though the half-life, of the lower, less highly chlorinated PCBs is probably of the order of two to four months. The data's very fuzzy still, but the half life is certainly longer than it is in rats. The higher PCBs, as Dr. Kolbye indicated, are quite persistent. This is not true for the people who have so-called Yusho disease, where they get PCB plus PCDF. Those people clean out their PCBs very rapidly, and the studies on Yusho victims show low levels of PCBs,

BROWN: (Continued) presumably due to the induction of mixed function aromatic hydrorylates enzymes.

SARDINAS: Dr. Kolybe?

KOLBYE: I'd like to ask if researchers have also taken a look at the persistence of the residues of quaterphenyls, over time, because I bet you those things really hang in there, in the Japanese people, and are complicating the entire picture to a degree.

BROWN: As far as I know, the only data on quaterphenyls come out of your laboratory.

SARDINAS: Next question, please.

GOLAS: My name is Kathleen Golas, I'm a National Science Foundation, Public Service Science Resident, working on hazardous waste management for the League of Women Voters of Connecticut. My question concerns the waste management of PCBs. One thing that I did learn this morning, definitely, is that we may be more concerned with the contaminant PCDFs, and not so much PCBs. I would expect that waste substances would be contaminated more highly than the substance that comes out of the factory, since

GOLAS: (Continued) I think I heard this morning that it's heat induced. I believe that the use of the PCBs is as an insulator, and therefore, subject to heat. I wonder if studies on disposal technologies, concur with the EPA in their estimation that PCB waste should undergo high incineration as a disposal mechanism.

BROWN: The procedure recommended by EPA for PCB disposal is high temperature incineration. This is at much higher temperatures than we're talking about, say, 2200 Fahrenheit, which is intended to guarantee absolute removal. This is currently recommended for PCB mixtures containing more than 50 parts per million of PCBs. EPA is currently recommending disposal in secure landfills for wastes containing lower levels. The EPA in its disposal practices is tending to treat all PCBs as if they were heavily contaminated with dibenzofurans and very toxic to deal with.

FLEMING: John Fleming, Watchdog Committee. I have a question to particularly Dr. Douglas, Dr. Kolbye and Dr. Weisburger. You're all acquainted with the literature that's available on the subject of PCBs relative to public health. I'm sure that you have some acquaintance with our work in the Health Department on blood sampling and fish sampling at PCB levels found in the Housatonic River. Each of you three have expressed a desire that maybe there's something we ought to know beyond what we do know. Dr. Douglas even pointed

FLEMING: (Continued) Cut one of the fallicies, really, of the scientific approach of trying to control things too carefully. Maybe somehow or other we ought to learn to work with more variables. Anyhow, my question is this, what areas of research do you think might be fruitful areas to explore toward the end of having a better understanding of the effects of PCB on public health and nature at levels found in the Housatonic River?

WEISBURGER: Well, we could start with the simple experiment and feed animals low levels of PCBs. You could feed animals PCBs at 5 parts per million, see what effects there are compared with controls. That would be at least the foundation of, or at least part of the answer to the question.

SARDINAS: Dr. Douglas?

DOUGLAS: In one sense the approach you request is somewhat alien to me. You are asking me to go on a fishing expedition and again, as a scientist, one often sees a problem and then starts to ask questions about it, from a health point of view, for example. If I had to choose any particular area in which work, should be done, which would provide more information about the toxicity of PCBs to man, I think my interests would still be in the occupational environment, where there are high levels

DOUGLAS: (Continued) of exposure for long periods of time. If there's going to be any evidence of reactions to PCBs, I believe that it's in that type of situation that we're going to see it. In terms of examining it's effects on the environment, that's a very complicated problem, because one, as the EPA has to face in its toxic substances control act, we don't have to only worry about health effects, we have to worry about its effects on birds and plants and so on. There is some information on that in regard to PCBs. Its effects in food chains may be a rather interesting area to examine.

SARDINAS: Dr. Kolbye.

KOLBYE: From the health viewpoint there is a fair amount of ongoing research in a variety of centers. And while the data may not have been published, I think there's new knowledge coming down the road which will be helpful to us. All the evidence that I've seen, if we're talking about PCBs per se, is pretty consistent with a conclusion that we ought to try to minimize human exposure. However, it is not a highly toxic compound, you know, as compounds go. That doesn't mean it's a compound that I'd like, or compound that I'd recommend to you. With respect to the dibenzofurans, we're up several orders of magnitude now, of increased toxicity if we're talking about certain isomers. And those are ones that

KOLBYE: (Continued) I think some additional research is needed on, with respect to animal studies. For example, I'm aware of a ongoing or at least a recent study, that deals with monkeys being exposed to dibenzofurans. Interestingly enough although I've not reviewed the data carefully yet, there is a suggestion that some of the effects of the dibenzofurans may be reversible as compared to the dibenzodioxin effects which were of a much more permanent nature. I'd like to close with a statement that one Hopefully will not get the impression that because an action leve' is at 5 or 2 parts per million that a human is getting exposed to the same dosage that a rat would get exposed to if that rat were eating 2 parts per million in its total diet. The rat would be getting a far higher, more intensive exposure. The human exposure anticipated either at 5 or at 2 parts per million in fish is a far less of an exposure.

FLEMING: Thank you very much. That's been very helpful to me.

SARDINAS: Well, seeing no further questions, I'd like to conclude this by, first of all thanking representative Moffett's office for participating with the State Health Department in conducting this information seminar. Secondly, by thanking the panel and particularly the National experts, Dr. Kolybe, Dr. Douglas, Dr. Weisburger,

SARDINAS: (Continued) and Dr. Brown, for giving of their time, and a portion of their rather busy schedule. Finally, I thank also the audience who appeared today with concerns; I hope we all have been able to gleen some new information about PCBs. I thank all of you and this meeting is concluded.