

the intestines following ingestion, or through the skin following physical contact. A fish can absorb them through its gills. Once inside the body, they gradually distribute themselves equally among all fat deposits present, and tend to stay there. A fish swimming in polluted water may accumulate them until its tissues contain up to 300,000 times as much PCB as was in the water.

Fig. 8

The body's methods of getting rid of PCB's are slow and inefficient. PCB's aren't volatile enough to be breathed out through the lungs, and they exit only slowly through the action of the liver, bile and intestines. Another way the body tries to get rid of lipid-soluble foreign chemicals is by converting them into water-soluble substances by chemical processes. These processes are catalyzed by enzymes called microsomal oxidases. The water-soluble products of these reactions can be excreted in the urine. Unfortunately, this route is also inefficient. The enzymes apparently attack PCB molecules too slowly, particularly if there are more than four chlorines present. In addition, the PCB species present may not be capable of inducing the formation of the particular kinds of enzymes needed.

Fig. 9

The enzyme-inducing activities of substances such as those found in PCB mixtures can result in a number of biological effects, which are most strongly expressed in the liver. The effects on liver tissue occur in more or less of a progression. First, the liver produces quantities of those microsomal enzymes I mentioned that carry out chemical reactions to get rid of foreign substances. This is rapidly followed by proliferation of microsomes — bits of protoplasm inside the liver cells that provide sites for the reactions — and by new cell growth, to provide room for all this activity. All of these changes are mild and reversible as far as the liver is concerned. But the high load of microsomal enzymes can alter the levels of fats and steroids in the circulation. With more toxicant, some liver cells may die, and eventually fatal collapse of liver function may result. Alternatively, particularly in rodents, there may appear benign or even malignant tumors.

Fig. 10

This brings up the issue of the possible role of PCB's in causing cancer. I should point out that chemicals are known to be able to contribute to cancer development in several ways. Some, like chloromethylether, are direct acting carcinogens. They can attack and mutate DNA molecules. Some, like vinyl chloride, are so-called procarcinogens, which means they can be converted to direct-acting carcinogens in the body and then cause mutations. Some, like cigarette tar, can be either promoters, which make mutant cells turn malignant, or enzyme inducers or activators, which activate other procarcinogens. Many people are particularly concerned over direct-acting carcinogens and procarcinogens since DNA mutation is an irreversible event. Hence, they argue that no additions to the natural burdens of such species should be tolerated. Conversely, promotion and enzyme induction are regulatory events. They are parts of the body's regular system of biochemical checks and balances and do not occur unless the control limits are exceeded. At present, I believe most scientists would regard it as highly unlikely that PCB's are either direct acting or procarcinogens. However, we cannot rule out the possibility that they could play a role as promoters or activators in some circumstances.

Fig. 11

The next generic effect I'd like to mention is chloracne. This syndrome comprises a slowly developing skin condition resembling severe adolescent acne. In addition, the victim feels generally sick, and may exhibit other signs such as gastrointestinal and neurological disturbances. In chickens, which are very

sensitive to the agents that produce chloracne in man, other effects can include edema disease and birth defects. Chloracne has been known for more than 80 years. It is now known to be induced by chlorinated coal tar; chlorinated naphthalene, or Halowax; some bad batches of PCB; and many bad batches of polychlorinated phenol fungicides, 2, 4, 5-T herbicide, and Agent Orange.

Fig. 12

A good deal of scientific work has been aimed at relating the molecular structures of individual PCB and contaminant species to their biological effects. From this work, three interesting correlations have emerged.

First, various molecules having a propeller-like shape have been found to induce the P450 cytochromes, which are the less active oxidases. Many individual PCB species, especially those of the more highly chlorinated Aroclors, have this shape and show P450 activity.

Second, molecules shaped like flat rectangles with chlorines at the corners are active at inducing P448 cytochromes. Three minor PCB isomers, which constitute less than one percent of the higher Aroclors, and virtually none of the lower Aroclors, can assume this shape and do show some P448 activity. The really active P448 inducers, however, are the PCDF's, sometimes present as PCB impurities, and the dioxins, which were the actively toxic impurities in 2, 4, 5-T and Agent Orange.

Third, although it is known that P448 itself is not toxic in the body, it has been found that all species active in causing chloracne possess comparable activity in inducing P448.

Fig. 13

What all this shape-specificity means is that all three types of biological response — P448, P450, and chloracne — must be initiated by binding to shape-specific recognition sites on receptor proteins inside the cell. Such proteins have indeed been identified. It is believed that the agent-receptor complex then induces expression of normally silent gene sequences. These result in the production of several new proteins, some of which are known and some aren't. It is presumed that some of the latter interfere with normal cell growth and function.

Fig. 14

The practical implications of this theory are both encouraging and cautionary. The bad news is that even apart from dibenzofuran impurities, the PCB's themselves may not be totally devoid of some forms of regulatory activity. The good news is that if the biological effects are basically regulatory — enzyme induction, cell growth stimulation, and so on — then there should be definite threshold doses below which no effects occur.

Fig. 15

Let's move now from basic biological properties to the actual health effects on humans. What have they been?

Fig. 16

First, chloracne. Chloracne is a very easily observed condition. The American occupational health literature relating to Halowaxes, herbicides, and tetrachloroazobenzenes repeatedly describes chloracne outbreaks. However, there are only three reports of unambiguous chloracne outbreaks associated with PCB's. The first, in 1933, involved workers at Swann Chemical in Alabama, who ran into one bad batch of the stuff, that had been made from off-spec benzene. It was recognized at the time that an impurity must have been responsible; however, attempts to identify it were fruitless and the effort

GENP 010339

was forgotten. The other two episodes both involved strongly heated PCB. Neither was as thoroughly investigated at the time as was the first episode, but the occurrence of chloracne was noted as being unusual.

Fig. 17

Second, occupational liver disease. Early test at the Harvard School of Public Health showed that heavy doses of various Aroclors, whether administered orally or by inhalation, would cause liver injury in rats. However, they also established no-effect doses, upon which the long accepted threshold limit values or TLV's were based. During the 40 years between 1937 and 1977, U.S. manufacturing industry handled over one billion pounds of PCB's under these guidelines, with no reported incidents of liver disease, none of unambiguous chloracne except for the episodes I just mentioned, and no other indications of significant health problems. This was an impressive accomplishment. Unfortunately, it produced not one single bit of medical or scientific documentation to prove that it actually had been the occupational health experience.

Fig. 18

Then in 1968 came Yusho. In southwestern Japan, 1300 people came down with some of the worst and most persistent cases of chloracne syndrome ever seen. Even newborn babies had it. Simultaneously, an epidemic of chick edema disease killed 400,000 chickens in western Japan. Patient investigations by a group at Kyushu University tracked the human disease down to use of a particular brand of rice oil, and to lots produced on two days in February 1968. The chickens, it turned out, had got the inedible by-products of these lots in their feed. Both lots had been produced in one leaky heat exchanger, and in that heat exchanger was the Japanese PCB, Kanechlor 400. The average amount of heat exchanger fluid consumed by the victims was estimated at two grams.

As this news trickled out of Japan from 1969 to 1972, many people became concerned over PCB hazards, and many new toxicological studies were initiated. PCB's were fed to everything: Bacteria, algae, water-fleas, oysters, lobsters, minnows, bluegills, trout, quail, chickens, rats, mice, rabbits, guinea pigs, cows, pigs, mink, dogs, and monkeys. In all cases, it was found that PCB's, like all other known substances, could sicken and kill the creatures if the dose were made high enough. Three of the species tested, chickens, mink, and monkeys appeared relatively susceptible; however, the magnitudes of the effects produced in them varied considerably from one experiment to the next.

Fig. 19

The first clue to the source of the variation was provided by a Dutch scientist, J.G. Vos, who showed that the ability of PCB's to cause birth defects in chickens or chloracne in guinea pigs depended upon the PCDF content of the PCB used. It was the impurity, polychlorinated dibenzofuran, PCDF, not PCB, that was the major source of the problem. It gradually developed that the European and Japanese PCB's contained 5-20 ppm of PCDF's, while the American Aroclors had only 0-2 ppm, even before purification for electrical use.

The Japanese picked up this observation, and rechecked their specimens. The Yusho heat exchanger fluid was found to have been thermally decomposed, mostly to polychlorinated quarterphenyls, or PCQ's. Only 40% of the original PCB remained. In addition, it contained 2000 to 5000 ppm PCDF's, which can be formed from PCB's by thermal oxidation above 500°F.

The Yusho victims were found to have rather thoroughly eliminated their body burdens of PCB; however, the PCDF's lingered, particularly in the liver, and the people remained sick with chloracne.

Fig. 20

Meanwhile, PCB's were tested for carcinogenicity, with quite variable results. American tests, made with commercial Aroclors as received, gave uniformly negative findings for the lower, capacitor-type Aroclors such as 1221, 1016, and 1242. However, both positive and negative findings were reported for 1254 and 1260. Japanese tests with Kanechlors in mice also gave positive findings of tumors, but showed that the PCB's could reduce the incidence of tumors induced by other common carcinogens. The official FDA assessment of this situation in 1979 was that the evidence for the carcinogenicity of PCB's themselves was inconclusive.

Fig. 21

At the present time, it is our impression that the more knowledgeable public health officials are well aware that the PCB's found in our equipment, service shops, landfills and neighboring waterways are chemically different from that at Yusho, and almost certainly toxicologically different. However, they face a public that has become very alarmed over the earlier parts of the Yusho story, and the subsequent PCB testing frenzy. What they really need, and what they've repeatedly asked for, is good epidemiological data on whatever it is that ordinary electrical grade PCB's do to people.

Fig. 22

Which leads to may final question: What do our PCB's do to people? Can we supply the data that the authorities are asking for? Remarkably enough, it appears that a definitive human exposure event actually did occur. This exposure event involved a statistically-large human population, heavy exposure to electrical PCB's alone, and sufficient elapsed time for the development of some delayed health effects. This population consists of present and past GE employees at the Capacitor Department in Hudson Falls. About 1000 people worked there in jobs providing significant exposure between 1946 and 1977. The exposure, in fact, is still in progress, since the people involved are retaining the accumulated PCB's in their bodies, and hence must be continuing to experience whatever effects they produce. As a result, these people are beginning to attract medical attention, both by ourselves and others.

Fig. 23

In 1976, Dr. Irving Selikoff of Mt. Sinai Medical School was called in by the union to check on their health status. He examined 326 volunteers (one-third of whom had jobs providing medium to high PCB exposure), and eventually reported that they were mainly normal; however, he also indicated some evidences of decreased pulmonary function, which did not correlate with PCB body burden, and some evidences for increased incidence of dermatological symptoms, such as rash, burning sensation, and acne, which did correlate, somewhat.

Fig. 24

Meanwhile, in 1976 the plant physician, Dr. Joseph Feingold, initiated a study of his own on 195 heavily-exposed employees and 20 Fairfield controls. Blood samples were taken, clinical examinations made, and the absence of obvious health problems noted. However, no detailed analysis of the data was attempted until the fall of 1979, when CRD got permission from the Corporate Environmental Quality Council to undertake a research study of the health effects issue. This stimulated a repeat examination of the people involved by Dr. Feingold and an ongoing statistical analysis of each individual clinical parameter by CRD. Current objectives are to determine, first, whether there are any significant departures from normal within this group; second, whether they correlate with PCB body burdens; and third, how both body burdens and health parameters are changing with time.

Fig. 25

Shortly after Dr. Feingold's repeat examination, Dr. Selikoff's associates arrived to repeat their 1976 tests. They have stated privately that one of their concerns is to confirm the reality of their alleged findings of respiratory impairment and significant dermatology. What they actually did was to draw more blood samples for PCB analyses and subject the members of their volunteer panel to an extraordinarily extensive medical examination. Dr. Selikoff's lab people are currently quite badly backlogged with other work, however, and it may be many months before any data emerge.

Fig. 26

In parallel with these efforts, in 1977, the National Institute of Occupational Safety and Health — NIOSH — initiated a mortality data analysis on both the GE capacitor population at Hudson Falls and the Aerovox capacitor population at New Bedford, Mass., using information supplied by the companies in both cases. In the fall of 1979 the NIOSH investigator issued a draft report on his findings, and turned further investigation of the Hudson Falls population over to Dr. Phil Taylor of the NYS Health Dept. Dr. Taylor secured our cooperation for the identification of individuals who had been missed in the earlier study and initiated a more rigorous analysis of the mortality data than had been done by NIOSH, and also an appraisal of the available records on births.

Finally, in late 1979, a small effort was begun by Mt. Sinai and American Cancer Society investigators to track down other evidences of unusual health effects or birth defects in the area. This is being done primarily by telephone survey.

Fig. 27

From the data now available, we know, first, that people who had moderate to heavy work exposure to capacitor PCB's now have an average of 1.5 grams of PCB in their bodies. Individuals in the top one percent are carrying about 10 grams. By way of comparison, normal Americans carry a little less than one-tenth of a gram as a background level, and the average Yusho victims ingested eight-tenths of a gram of actual PCB.

We have no evidence yet of any significant decline in PCB levels in the people's bodies between 1976 to 1979. This is in marked contrast to the findings of Yusho, where the extra PCB load was 99% gone after a year. Obviously, whatever PCB excretion process got induced by the PCB/PCDF/PCQ mixture at Yusho is not operative at Hudson Falls; the PCB's are persisting.

Third, our own clinical findings to date, and the preliminary NIOSH mortality data, both indicate a lack of serious health effects in our population. The death rates for cancer, cardiovascular disease, neurological disease, accidents, and for all causes are all slightly below normal.

Fourth, there are indications of subclinical abnormalities in the exposed population. In addition to the presence of PCB's themselves, we see some elevations of serum triglycerides in males though not in females, and some elevations of serum enzymes, both of which suggest slight alteration in liver function. Other parameters may also be slightly abnormal; our analyses of the available data aren't yet complete. In addition, we have no inkling as to the other examples of physiological alterations or statistically-marginal health effects that may be revealed by Dr. Selikoff's colleagues at Mt. Sinai and the Cancer Society.

Fig. 28



So, what are we now doing about this, what are our plans, and what do we hope to accomplish?

First of all, we are helping the New York State Health Department obtain the personnel and exposure data needed for their studies of mortality and birth abnormalities. Conceivably, they may need financial encouragement later on. We consider it very important that this study be completed and published in the open literature.

Second, we are continuing to monitor the Mt. Sinai/Cancer Society investigations as best we can.

Third, we are attempting to resolve anomalies and establish the reliability of the PCB analyses run on the Hudson Falls population. It is important for us to have solid data on PCB body burdens if our story is to be credible.

Fourth, we are continuing the analysis of Dr. Feingold's 1976 and 1979 data to identify any clinical abnormalities and establish correlations with the levels of both higher and lower PCB's.

Fifth, we are working on an unambiguous analytical method for defining the level of PCDF impurities in PCB specimens. When we have it, we will be applying it to PCB's from many old capacitors to establish the levels of PCDF's in the PCB's that produced the occupational exposure during the 1946-77 period.

Our immediate objectives, in short, are to be able to show, first, that our PCB was actually virtually free of PCDF, second, that heavy human uptake of this PCDF-free PCB did occur, and third, that this uptake had no serious health consequences.

Fig. 29

This is not all that we are doing about PCB problems, of course. Until last fall, all of our efforts at CRD were focused on the environmental fate of PCB's; environmental clean-up procedures involving microbial degradation or other techniques, and procedures for getting PCB's out of contaminated transformer oil. We are still heavily involved in all of these areas; however, they don't bear on the health effects issues, so I will pass over them for the present.

Fig. 30

Some projected future activities that do bear directly on emerging health effects issues are listed here. One issue is that both we and Dr. Selikoff will soon know that we have a GE employee population that will carry PCB's with them for the rest of their lives. They aren't clearly sick, but they aren't entirely normal either. The first thing that we will have to do is to maintain medical surveillance of them to ensure that the alterations seen are physiological rather than pathological, and to be able to answer their inevitable questions.

Second, we should endeavor to learn how to measure P448 cytochromes in these peoples' blood cells and to correlate these measurements with other data on subclinical health effects. Animal data show that these cytochromes represent the most sensitive, specific, and quantifiable indicators of PCB bioeffects. We need to know what they can tell us about our employees' physiological status.

GENP 010343

Third, we need to determine PCDF levels in both new and used transformer fluids, in order to ensure that the transformer PCB's are as low as capacitor PCB in toxic contaminants.

Fourth, we need to ensure the development of analytical capabilities for determining the PCDF levels in environmental specimens, such as fish and sludge. If the agencies are ever to regulate environmental PCB hazards on the basis of the toxic impurity present, rather than the total PCB, they will have to have access to some analytical method that can be used on field specimens by their enforcement agents.

Where will all this lead?

Fig. 31

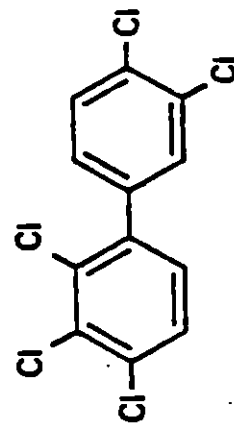
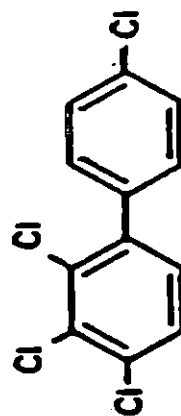
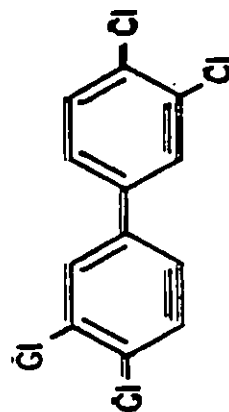
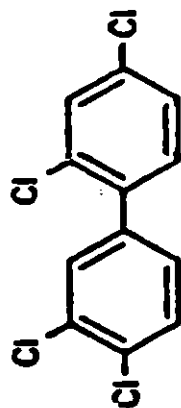
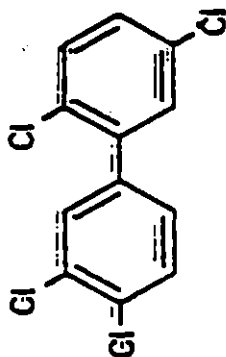
Looking into the future, we anticipate that ongoing academic medical research will gradually reveal the detailed chemical and biological mechanisms involved in toxic responses to PCB's. Concurrently, we would hope to see the sort of health status documentation effort that's now shaping up at Hudson Falls extended to other locations. As the results of such findings become available, we anticipate some easing of regulatory controls, particularly those over fishing and low level waste management. Such relaxation could be most easily instituted by changing the basic criterion for human protection from the level of PCB to that of PCDF. With such regulatory reform, deescalation of the PCB supertoxicity myth, which has so bemused the media in recent year, could begin.

HEALTH EFFECTS OF PCB's

- WHAT ARE PCB's?
- WHAT ARE THEIR BIOLOGICAL PROPERTIES?
- HOW TOXIC ARE THEY?
- WHAT DO ELECTRICAL PCB'S DO TO PEOPLE?

783156

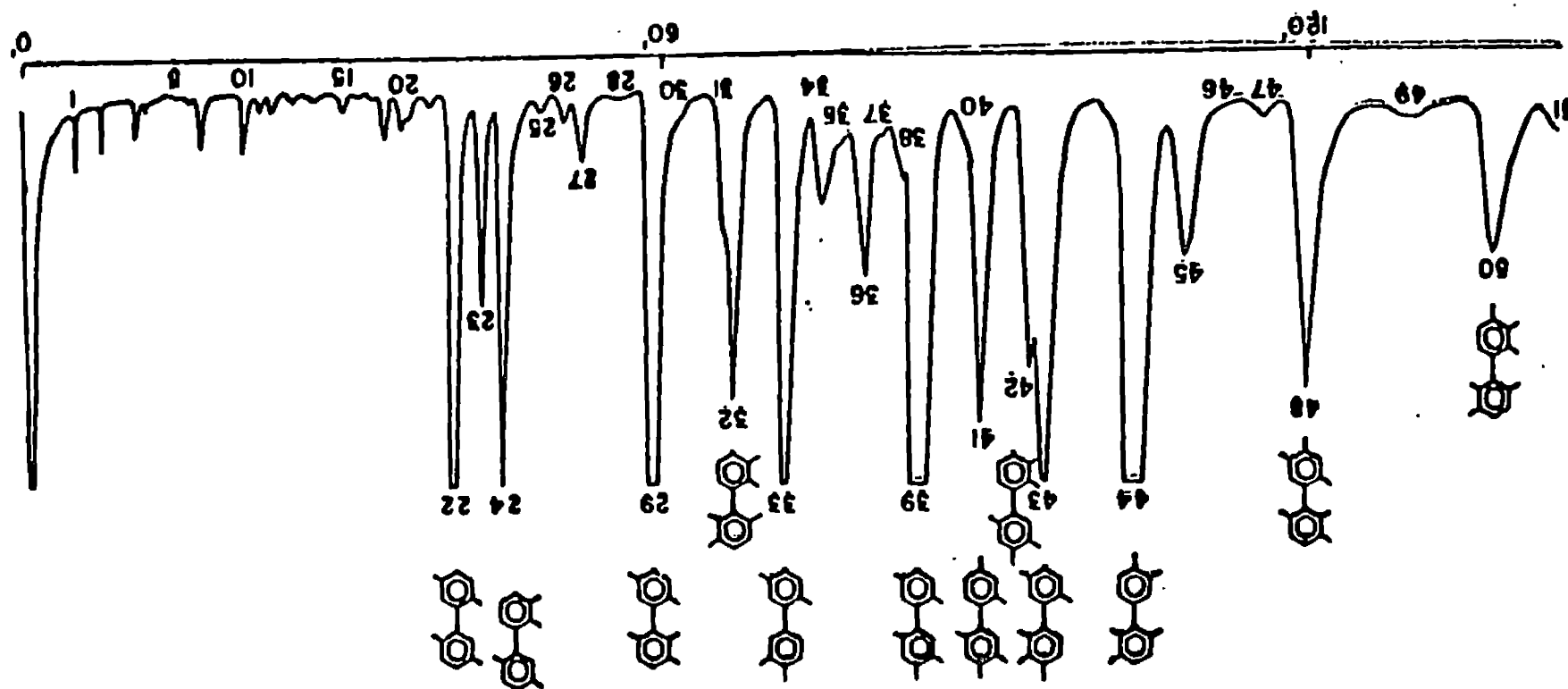
TYPICAL PCB STRUCTURES



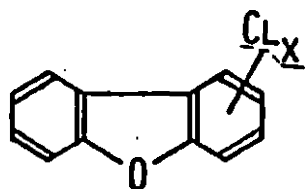
POLYCHLORINATED BIPHENYLS

<u>AVERAGE No. OF CL'S</u>	<u>"AROCOR" DESIGNATION</u>	<u>ELECTRICAL Use</u>
1.5	1221	CAPACITORS
3	1242	CAPACITORS
5	1254	TRANSFORMERS
6	1260	TRANSFORMERS

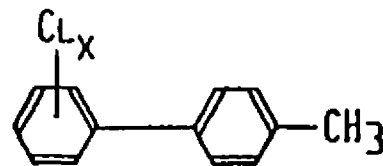
783158



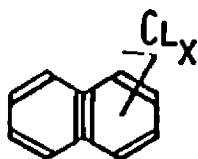
CONTAMINANTS IN PCB's



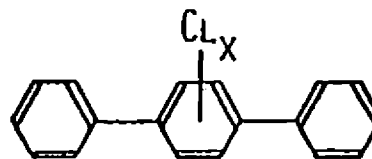
POLYCHLORINATED
DIBENZOFURAN
PCDF



POLYCHLORINATED
METHYL BIPHENYL



POLYCHLORINATED
NAPHTHALENE



POLYCHLORINATED
TERPHENYL

HEALTH EFFECTS OF PCB's

- WHAT ARE PCB's?
- WHAT ARE THEIR BIOLOGICAL PROPERTIES? 

783161

BIOLOGICAL PROPERTIES

- EASY TISSUE PENETRATION

LUNGS INTESTINES

SKIN GILLS

- BIOACCUMULATION AND RETENTION

783162

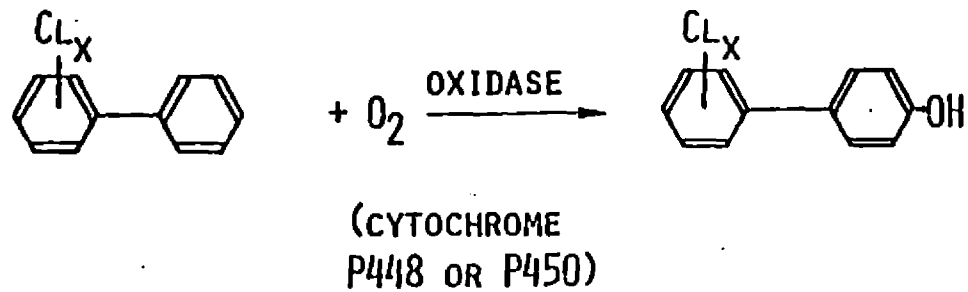
GENP 010351

EXCRETION ROUTES

- VOLATILIZATION THROUGH LUNGS

- LIVER $\longrightarrow$ BILE $\longrightarrow$ INTESTINES $\longrightarrow$ FECES

- OXIDATION $\longrightarrow$ KIDNEYS $\longrightarrow$ URINE



EFFECTS OF CHLORINATED ORGANICS ON LIVER

PHYSIOLOGICAL RESPONSES

- MICROSOMAL ENZYMES FORMED
- PROLIFERATION OF MICROSOMES
- CELL GROWTH

TOXIC EFFECTS

- CELL DEATH
- COLLAPSE OF LIVER FUNCTION
- TUMORS

783164

GENP 010353

CANCER-CAUSING AGENTS

- DIRECT-ACTING CARCINOGENS

MUTATE CELL'S DNA

- PROCARCINOGENS

METABOLIZABLE TO DIRECT-ACTING CARCINOGENS

- PROMOTERS

MAKE MUTANT CELLS MALIGNANT

- ACTIVATORS

INDUCE ENZYMES THAT ACTIVATE PROCARCINOGENS

783165

CHLORACNE SYNDROME

MAN

- SKIN: BLACKHEADS, PUSTULES, PIGMENTATION
- SOMETIMES GASTROINTESTINAL, NEUROLOGICAL DISORDERS

CHICKENS

- EDEMA DISEASE; BIRTH DEFECTS (TERATOGENESIS)

783166

RELATION OF STRUCTURE AND BIOLOGICAL EFFECTS

- PROPELLER-SHAPED MOLECULES INDUCE P450 CYTOCHROMES
 - MANY INDIVIDUAL PCB SPECIES

- FLAT RECTANGULAR MOLECULES INDUCE P448 CYTOCHROMES
 - THREE MINOR FORMS OF PCB
 - PCDF IMPURITIES
 - DIOXINS

- SPECIES ACTIVE IN CAUSING CHLORACNE ALSO INDUCE P448

783167

THEORY OF BIOLOGICAL RESPONSE

- ACTIVE AGENT BINDS TO SHAPE-SPECIFIC RECOGNITION SITE ON RECEPTOR PROTEIN
- AGENT-RECEPTOR COMPLEX INDUCES EXPRESSION OF NORMALLY SILENT GENE SEQUENCES
- NEW PROTEINS PRODUCED:
 - P448 OR P450 CYTOCHROMES
 - OTHER INTRACELLULAR ENZYMES
 - STIMULATORS/SUPPRESSORS OF CELL GROWTH?
 - STIMULATORS/SUPPRESSORS OF CELL FUNCTION?

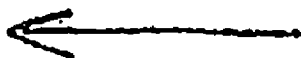
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GENP 010357

IMPLICATIONS

- BIOLOGICAL EFFECTS ARE BASICALLY REGULATORY;
SHOULD EXHIBIT DEFINITE THRESHOLDS FOR OCCURRENCE
- EVEN PURE PCB'S MAY NOT BE DEVOID OF SOME FORMS OF
REGULATORY ACTIVITY

HEALTH EFFECTS OF PCB's

- WHAT ARE PCB's?
- WHAT ARE THEIR BIOLOGICAL PROPERTIES?
- HOW TOXIC ARE THEY? 

783170

GENP 010359

REPORTED U.S. EPISODES OF PCB-RELATED CHLORACNE

<u>EPISODE</u>	<u>DATE</u>	<u>AFFECTED/ EXPOSED</u>	<u>COMMENT</u>
1	1933	23/24	BAD BATCH PCB FROM EARLY PRODUCTION
2	1950-2	7/14	VAPORS FROM LEAKY HEAT EXCHANGER
3	~1963	13/15	VAPOR FROM BAKING PCB- PLASTICIZED ENAMEL

783171

GENP 010360

OCCUPATIONAL HEALTH STANDARDS AND EXPERIENCE

- HEAVY DOSES OF AROCLORS CAUSED LIVER DISEASE IN RATS
- NO-EFFECT DOSES TLV'S ESTABLISHED
- 40 YEARS OCCUPATIONAL HEALTH EXPERIENCE
 - NO LIVER DISEASE
 - NO FURTHER CHLORACNE EPISODES
 - NO OTHER HEALTH PROBLEMS
- LACK OF SCIENTIFIC DOCUMENTATION

783172

YUSHQ EPISODE - SOUTHWEST JAPAN 1968

- 1300 CASES OF SEVERE CHLORACNE
- 400,000 DEAD CHICKENS
- TRACED TO RICE OIL CONTAMINATED WITH KANECHLOR 400
FROM HEAT EXCHANGER

783173

GENP 010362

ANALYSIS OF YUSHO INCIDENT

- FINDINGS OF J.G. Vos
 - BIRTH DEFECTS AND CHLORACNE IN ANIMALS
CORRELATE WITH PCDF CONTENT OF PCB'S
- PCDF CONTENT OF PCB'S VARIES
 - EUROPEAN AND JAPANESE: 5-20 PPM
 - AMERICAN: 0-2 PPM
- YUSHO HEAT EXCHANGER CONTAINED 2000-5000 PPM PCDF'S
- PCB'S ELIMINATED; PCDF'S LINGERED

783174

FDA FINDS CARCINOGENICITY TESTS INCONCLUSIVE
(44 FR 38330 - JUNE 29, 1979)

EVIDENCE:

<u>AROCLOR</u>	<u>SPECIES</u>	<u>RESULT</u>	<u>AUTHOR</u>	<u>DATE</u>
1221	RABBIT	NEG.	KELLER	1973
1016	RAT	NEG.	BURSE	1974
1242	RAT	NEG.	MONSANTO	1969
1242	RAT	NEG.	BURSE	1974
1254	RAT	NEG.	MONSANTO	1969
1254	RAT	POS.	KIMBROUGH	1972
1254	RAT	NEG.	NCI	1977
1260	RAT	NEG.	MONSANTO	1969
1260	RAT	POS.	KIMBROUGH	1972

783175

GENP 010364

CONCLUSIONS FROM YUSHO
AND RELATED ANIMAL TESTS

- AMERICAN PCB'S CHEMICALLY AND TOXICOLOGICALLY
DIFFERENT FROM THOSE CAUSING YUSHO EPISODE
- BUT PUBLIC HAS BECOME ALARMED

783176

GENP 010365

HEALTH EFFECTS OF PCB's

- WHAT ARE PCB's?
- WHAT ARE THEIR BIOLOGICAL PROPERTIES?
- HOW TOXIC ARE THEY?
- WHAT DO ELECTRICAL PCB's DO TO PEOPLE?



HEALTH INVESTIGATIONS AT HUDSON FALLS

DR. I. SELIKOFF - MT. SINAI MEDICAL SCHOOL - 1976

- EXAMINED 326 VOLUNTEERS
- REPORTED THEM MAINLY NORMAL
- SOME EVIDENCE OF DECREASED PULMONARY FUNCTIONS
- SOME EVIDENCE OF INCREASED DERMATOLOGICAL SYMPTOMS

783178

GENP 010367

HEALTH INVESTIGATIONS AT HUDSON FALLS

DR. J. FEINGOLD - GENERAL ELECTRIC - 1976

- 195 HEAVILY EXPOSED EMPLOYEES AND 20 FAIRFIELD CONTROLS
- BLOOD SAMPLES, CLINICAL EXAMINATIONS
- NO OBVIOUS HEALTH PROBLEMS SEEN
- DETAILED DATA ANALYSIS BEGUN FALL 1979
- REPEAT EXAMINATION 1979

783179

GENP 010368

HEALTH INVESTIGATIONS AT HUDSON FALLS

DR. I. SELIKOFF'S ASSOCIATES - MT. SINAI MEDICAL SCHOOL - 1979

- REPEAT OF 1976 TESTS
- MANY NEW TESTS PERFORMED
- RESULTS STILL UNKNOWN

783180

GENP 010369

HEALTH INVESTIGATIONS AT HUDSON FALLS

NIOSH - 1977

- MORTALITY DATA ANALYSIS OF HUDSON FALLS AND AEROVOX POPULATIONS
- DRAFT REPORT ISSUED 1979
- INVESTIGATION TURNED OVER TO NYS HEALTH DEPARTMENT 1979

783181

FINDINGS TO DATE

1. PCB LEVELS MUCH ABOVE NORMAL

EXPOSED CAPACITOR WORKERS	1.5 GRAMS
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FAIRFIELD CONTROLS	0.1 GRAMS
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2. PCB LEVELS REMAINING HIGH

3. NO SERIOUS HEALTH EFFECTS

EXAMINATION RESULTS NORMAL

MORTALITY RATES ALL BELOW NORMAL

4. POSSIBLE SUBCLINICAL ABNORMALITIES

TRIGLYCERIDES, SERUM ENZYMES

ONGOING ACTIVITIES

- ASSISTING NIOSH/NYSDH IN COMPLETING THEIR MORTALITY STUDIES
- MONITORING MT. SINAI/AMERICAN CANCER SOCIETY STUDIES
- QUALIFYING RELIABILITY OF PCB ANALYSES
- WORKING ON ANALYSIS OF DR. FEINGOLD'S CLINICAL DATA
- DEVISING RELIABLE METHOD FOR PCDF ANALYSIS
- DETERMINING PCDF LEVELS IN 1946-77 CAPACITORS

OTHER ONGOING PCB-RELATED ACTIVITIES

- ENVIRONMENTAL FATE
 - MICROBIOLOGICAL STUDIES
 - ADSORPTION
 - VOLATILIZATION

- DESTRUCTION OF PCB'S IN CONTAMINATED TRANSFORMER OIL
 - LABORATORY INVESTIGATIONS
 - FIELD TEST AT ASBD PHILADELPHIA SITE
 - ADVANCED METHODS

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FUTURE ACTIONS

- **MAINTAIN SURVEILLANCE OF EXPOSED HUDSON FALLS
POPULATION**
- **CORRELATE LEVELS OF P450 AND P448 CYTOCHROMES WITH
OTHER SUBCLINICAL DATA**
- **DETERMINE PCDF LEVELS IN TRANSFORMERS**
- **DETERMINE PCDF LEVELS IN FISH, RIVER BOTTOM SLUDGE,
DUMP SITES**

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OUTLOOK

- RESEARCH RESULTS ON DETAILED TOXICITY MECHANISMS
- EXTENSION OF HEALTH STATUS DOCUMENTATION
- EASING OF REGULATORY CONTROLS

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