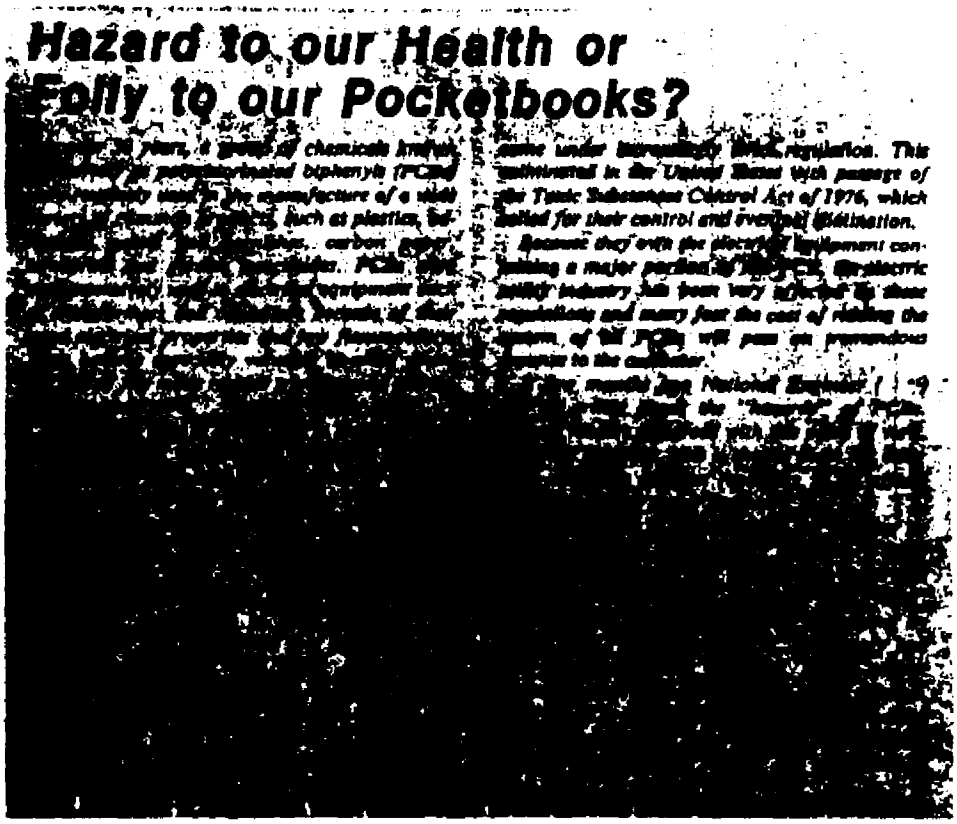

PCB White Paper

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PURPOSE

What we have done in this report is to take the arguments, and the facts behind those arguments, of our opponents (those who think PCBs are dangerous) from their own literature and we have presented both their arguments and conclusions to show that their conclusions are not justified by the facts available to them. By this method of reasoning we hope to show that the conclusion of the environmentalists that PCBs must be banned, or even more tightly regulated in transformers and capacitators than they are now, is based on *facts that do not support their conclusions.*

BACKGROUND INFORMATION

Production of Polychlorinated Biphenyls began in the U.S.A. in 1929 by Swann Chemical Company. In 1935, Monsanto bought out Swann Chemical and became the sole producer of PCBs for transformers and other uses. The reasons for this monopoly lay in the fact that the standards for purity as set down by General Electric Company — the first ones to apply PCBs to electrical equipment — were so high that no one else wanted to enter into the business. As a result of these standards, the PCBs produced in America under Monsanto Company's trade name of Aroclor® were the purest of any PCBs produced in the world.

PCBs were manufactured primarily because of their excellent dielectric and heat transfer qualities, not to mention their non-flammability. Not only did their non-flammability capabilities make them suited for use in capacitors, but also made them ideal for use in areas where public safety was concerned, such as, in transformers inside public buildings.

This set of uses, coupled with the unusual stability of the compound, have led to the calling of PCBs as the "wonder chemical of the century." They greatly aided America's industrial expansion since the early 1930's. It has been said that Russia's industrial successes could not have oc-

Material Category	Purpose	Conditions	Life Expectancy	Limitations
Asphalt Emulsion with Fiberglass or Polyester Membrane	Water repellancy and reinforcement of aged asphalt BUR	Fracturing of top felt plys; underlying felts still sound	3 to 10 years depending on product quality, installation techniques and a reflective coating	Severely affected by trapped roof moisture; requires smooth/prepared surface; should not be used if deck movement is causing roof splits
Reflective White and Aluminum Coatings	Protect roof against U.V. exposure; reduce interior temperatures; extend serviceable coating/roof performance	New BUR or new coating/reinforcement systems requiring additional protection; energy conservation	1 to 5 years depending upon product quality and coverage rate	Strictly preventive design should not be utilized to solve roof deficiencies
Proprietary Coatings	Usually as preventive maintenance protection; some products developed as weathering seal and/or remedial capability	Wide variety of conditions depending upon product formulation	Varies widely; manufacturer's guarantee suggested (including labor to replace coating if defective)	Careful and thorough scrutiny of manufacturer's reputation, integrity, and proficiency advised prior to purchase
Cold Process BUR (Felts and Factory Formulated Adhesive)	Overlay existing roof that has aged beyond other corrective techniques	Sound deck and roof substrate; not more than one roof system on deck; existing roof cannot be correctively maintained	5 to 10 years depending on application techniques and specification quality	Severely affected by trapped moisture; should have insulation underlayment; preferably mechanically fastened to substrate; deck weight load capacity must be considered
Urethane Foam Systems	Lightweight resurfacing of existing aged roofing systems; provide or enhance insulation capability and benefits	Adequately sloped building requiring additional roof insulation capability; lightweight roof weatherproofing system is necessary	2 to 10 years depending upon insulation density, coating quality, and method of installation (manufacturer's guarantee advised including labor to replace system if defecitve)	Requires expert installation expertise; severely affected by moisture conditions during application; system is totally dependent on surface coating product quality
Single-Ply Systems	Overlay existing roof that has aged beyond other corrective techniques; provide lightweight system without roof removal	Sound deck and roof substrate; minimal roof projections; existing roof mat cannot be correctively maintained	1 to 10 years (manufacturer's guarantee advised including labor to replace system if defective)	New roof technology; first generation problems anticipated; usually requires expert installation knowledge; careful scrutiny of manufacturer's reputation, integrity, and proficiency advised

by Sam Spinosa THE BRYANT ORGANIZATION INC

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curred had it not been for PCBs. What then, went wrong?

In 1966, a Swedish scientist was trying to determine the level of DDT in fish. He found some unidentified chemical in his studies. This unknown chemical turned out to be PCBs. That PCBs had been found in the environment should have come as no surprise since they are so stable and they were used in such a multitude of products (e.g. paints, adhesives, caulking compounds, plasticizers, inks, lubricants, carbonless copy paper, sealants, coatings, dust control agents, heat transfer systems, hydraulic systems, etc.).

THE PRIMARY EVIDENCE

The big blow to PCBs, however, came two years later in Japan. It was reported that PCBs, as a contaminant, got into some rice oil, which was then sold to the surrounding population. This rice oil was used for cooking purposes for a period of approximately 100 days. Up to 1300 people were adversely affected from the ingestion of this cooking oil containing PCB. Those affected ate 720 ml. to 4.4 liters of oil containing 0.5g of the contaminant, with the average consumption being approximately 2g.[1] These effects were erroneously reported as being caused by the PCB.

The symptoms of this "oil disease" ("Yusho") have been described as follows: dermal disorders, including swelling of the eyelids, acneform eruptions and pigmentation of the nails, gums and lips and an increased discharge from the eyes was observed. The people also complained of things like headaches, indefinite stomach ache, numbness or pain in the extremities, coughing, and bronchitis-like symptoms. Gains in the height and weight of boys "decreased significantly" after ingesting this contaminated oil.[2] Babies born to women with Yusho had abnormal skin pigmentation and some anatomical abnormalities. However, "these were not permanent, and postnatal body and mental development appeared normal in these and other Yusho children."[3] (NOTE: All of these symptoms were alleged to be caused by PCBs. The allegations have proven to be false. But, on the basis of "Yusho," the Congress of the U.S.A. passed legislation to Regulate and Ban PCBs — TOSCA).

These, then were the illnesses associated with Yusho — illnesses that would make the world want to rid itself of the "rice oil contaminant" that caused all these problems. (This is especially true, since the results of the contaminants on humans, were graphically portrayed in a picture book). That contaminant that was first thought to cause the Yusho disease was first diagnosed to be Kanechlor 400, a Japanese PCB in the amount of up to 3000 ppm. Thus began the movement that eventually saw PCBs banned in Japan, Sweden, Canada and the United States.

THE DEFENSE

As is often the case with initial findings and the accompanying announcements, not all the facts in this case were in at first. Kanechlor 400 was present but it was not the most toxic (if it even is toxic at all) chemical involved. Polychlorinated Dibenzofurans (PCDFs) a partial oxidation by-product of PCBs, were found to be in concentrations of up to 500 times that normally found in Japanese PCB.[1] Not only this, but the tetrachloro- and pentachloro- fractions, which have through animal toxicity studies, been determined to contain the more toxic isomers, were found in high concentrations.[4] PCDFs affect the thymus, skin (acne), liver and hematopoietic system to a much greater degree than PCBs, and are 1000 times more potent as enzyme inducers.[5]

In addition to this, PCDFs were chronically retained in the livers of Yusho victims. Three Yusho patients eventually died (two in 1969, one in 1972); these deaths were not attributed to the ingestion of the contaminated PCBs. In the tissues obtained at autopsy, PCDFs were found in high concentrations. No PCDFs were found in the tissues of two controls, "although PCB concentrations of about 1-1.5 ppm were found in the adipose tissue and liver fat (of the controls)."[6] The PCB/PCDF ratio in the rice oil was about 200, while the ratio of PCB/PCDF found in the adipose tissues at autopsy were 2, 5 and 12, a considerable difference.[4]

By the end of 1977, 31 classified deaths of Yusho patients had been recorded. Eleven deaths were from neoplasm, which is 4.5 deaths more than would normally be expected.[6] However, this 4.5 could easily be zero or not attributed to Yusho, since the data were not age-adjusted.[7] In fact, a booklet prepared by the Hazard Evaluation System of the State of California, one of the strictest states with respect to environmental issues, cites a 1979 study (Urabe, et al.) "the evidence is not conclusive that PCBs caused these deaths."[1]

PCB LAW IS BASED ON "YUSHO"

Now is the time to see what is presently being said about the relevance of Yusho to the study of the toxicity of PCBs and their possible human effects.

We will begin this exercise with a quote from John F. Brown, Jr., PhD, Manager of the Life Science Branch of G.E.'s Corporate Research and Development as he explains the irrelevance of linking PCBs and Yusho together. ". . . it was the PCDF's that were chronically retained in the livers of the chronically-ill victims. In these victims, the average dose of the PCB/PCQ (Polychlorinated Quatraphenyls)/PCDF mixture ingested was 2.0g., which is less than the PCB body burden being carried by many seemingly normal (U.S.) capacitor workers today. Thus, it would appear that the toxic agent responsible for Yusho disease was PCDF, not PCB."[8]

Dr. Renata Kimbrough, M.D. a Toxicologist at the Center for Disease Control in Atlanta, Georgia, who has been one of the government's star witnesses against PCBs was involved in the following dialogue in a North Carolina court case in 1979:[9]

Question: But, Dr. Kimbrough, did you participate in the preparations of a report by a sub-

committee of the Department of Health, Education and Welfare, July, 1976?

Dr. Kimbrough: Yes.

Question: And let me ask you if I'll read from that study and ask you if this is a fair statement. "The complexities and uncertainties associated with the most recent reports of the Yusho Incident in Japan make it extremely difficult to quantify possible human health effects resulting from exposure to PCBs alone." In other words, Doctor, isn't it true that there is a strong suspicion now that the other chemicals that got into that rice oil other than PCBs are the things that caused the problems?

Dr. Kimbrough: They certainly caused part of the problem. We don't know whether they caused all of the problem, but they are very toxic.

Question: And by the same token we don't know whether PCBs caused any of the problems because of the presence of those other chemicals. I'm talking about scientific.

Dr. Kimbrough: In the Yusho incident, no."

A 1980 report issued by the State of California Research Division contains many such statements. One of them says that "it is therefore considered highly probable that some of the cause of Yusho toxicity is due to the actions of PCDF."[4] (This same report finally concludes this: "a review of the available literature on PCB, PCDDs (Polychlorinated Dibenzo Dioxins), and PCDFs indicates that exposure to these substances, even in microgram quantities, can lead to extremely serious human health effects."[5] What is the source of this convoluted reasoning? Their bibliography of 51 sources is constituted of five articles on PCB, all five sources deal specifically with the Yusho incident, and 46 articles deal with PCDDs and PCDFs. They admit that PCBs have no proven record of human health problems, but because they "look alike," PCDFs and PCDDs, they group all three together and call them bad!!! (NOTE: It is this type of reasoning that has led to the unreasoning and unreasonable pressure of the radical environmentalist that will eventually cost our country $4,000,000,000,000.00 — or $50,000.00 for every man, woman and child in the country today!!!)

A 1981 report prepared by the Hazard Evaluation System of the State of California states that "It is not clear how much the health effects observed in Yusho victims can be extrapolated to occupational exposures for the following reasons:

1. The average amount of PCB (Kanechlor 400) ingested was estimated to be 2 grams and the minimum, 0.3 grams (Kuratsune et al., 1971). This is a higher dose than has been reported in most occupational exposures. In addition, the PCBs were ingested as opposed to inhaled or skin-absorbed as in the case with occupational exposures.

2. The contaminated oil contained "used" (oxidized) Kanechlor 400, the exact chemical composition of which is unknown.

3. Frying of foods with the rice oil could have produced further new compounds which may have altered the toxicity of the PCBs or the toxicity of possible contaminants.

4. Yusho oil was shown to contain high concentrations (2500 ppm) of dibenzofurans.

5. Reported concentration of PCBs in the oil may not have been accurate enough to permit a rigorous quantitative analysis since the methods for estimating PCBs in foods were not fully developed at the time."

The National Institute for Occupational Safety & Health (NIOSH) published a criteria document on PCBs in 1977. They say that:

The relevance of the Yusho episode to occupational PCB exposure is compromised because: (1) the oil was ingested; and (2) it contained large concentrations of dibenzofurans compared with those in the PCBs to which workers generally have been exposed in their occupations. Its relevance is further compromised because the effects observed from daily ingestion of 1-15 mg of PCBs (165, 167) were peculiar and excessive compared to those observed in workers exposed by inhalation of PCBs 1-5 mg/M³ (120, 125), notwithstanding that the amounts absorbed may have been similar.

Nevertheless, information obtained from the Yusho episode is relevant to the study of PCB toxicology and occupational exposure. The information established that PCBs can be transmitted from mother to fetus, and, in the milk, from mother to child. It also establishes that some PCB compounds are eliminated from the body relatively rapidly, and that others may require years for elimination."

After NIOSH came to this conclusion that Yusho could not be used to determine PCB toxicity, they turned around, and in the same document, state that the "toxic effects from ingestion of PCBs have been well documented in humans."[6]

The authority for this statement? Forty footnotes — every single footnote referring to the early Japanese studies of the Yusho incident, when all of the effects were erroneously attributed to PCBs.

EPA — THE AGENCY TASKED BY CONGRESS TO CONTROL PCBs

Finally, what has EPA said about Yusho? Please note the dates as you read the conclusions reached.

EPA 1978 — Subsequent investigations (have shown that the "Yusho Oil") had been more than half pyrolyzed to PCQs, and that this PCB-PCQ mixture contained 0.25% (2500 ppm) of PCDFs. It was the PCDFs that were chronically retained in the livers of chronically-ill victims.

The same oils were reanalyzed in 1976.

EPA 1977 — Because of the relatively large exposure of the Yusho victims to PCDFs it is inappropriate to use this data to make quantitative estimates to the TOXIC hazard posed by PCBs to humans.

Compare these first two statements with EPA's 1979 statement.

EPA 1979 — Knowledge of PCB toxicity is

based primarily on the Yusho Japan incident in 1969. (NOTE: Why has the EPA contradicted itself here, and take a position contrary to all recognized scientific evaluations of Yusho?)

Does not this last statement (when compared with EPA's earlier statements and the statement by all the "authorities" that a determination of PCB toxicity from Yusho is scientifically inappropriate) tell us that the case against PCBs, is "based primarily on no evidence at all?" This is what EPA has said, whether wittingly or not. The evidence is here. Our rules regulating PCBs are based on this fallacy! This is what the Congress must know; this is what the public should know: that PCBs are not a health problem to humankind!

In 1976-77, Congress passed the Toxic Substances Control Act (TOSCA). The only specific chemical listed in this law was PCBs — based upon one senator's amendment to the original TSCA law because of what happened in Japan in 1968 and to political pressure brought about by publicity of the event. This law prohibited the sale, distribution or manufacture of PCBs after 1979. Monsanto, the only U.S. manufacturer, ended its production of the chemical in 1977. After that time, PCBs were to be controlled. The EPA ended up with this regulation job, and set down rules which, in part, said that transformers with less than 50 ppm PCB could be considered a non-PCB object. The radical environmentalists, working through the Environmental Defense Fund (EDF) sued EPA for allowing any ppm of PCB. (EDF vs. EPA DC Cir No. 79-1580 Act 501980, October 30, 1980). The court ruled in favor of the EDF and said that Congress mandated a ban (0 ppm) on PCBs in transformers and capacitors. EPA has said that transformers and capacitors were "closed systems" but the court ruled in favor of EDF, recognizing that transformers and capacitors do sometimes leak or fail. EEI (Edison Electric Institute), as a friend of the court, told the court that the ruling left the electrical industry of America two choices; they could either operate illegally (which they would not do) or they would have to shut down all operations immediately. The court decided that this might not be such a good idea so it gave EPA 18 months, till October, 1982 to address these two questions:

1. PPM level of PCB to be regulated; and
2. Rules of operation of "totally enclosed" devices containing any PCBs.

Since Congress passed TOSCA, identifying PCBs as a priority pollutant to be regulated, the EPA must act on the mandate of Congress. Therefore, the solution to this problem must go back to the source of the problem: Congress. Congress must be made aware of the growing body of literature that completely exonerates electrical grade PCB from the health and environmental problems it has allegedly caused.

WHY ANIMAL STUDIES ARE USED

The consensus of those who study PCBs are in agreement that the Yusho event cannot, scientifically, be the basis for condemnation of PCBs. What, then, is the basis for the ban on PCBs? A review of the literature alludes to a lack of epidemiologic evidence to support their position. (Note: They are willingly ignorant of over 15 epidemiologic studies!!! And the reason why they are willingly ignorant of these 15 studies? Because they can not find any evidence — anywhere to support the conclusion they have predetermined to make! That's right. If they can't find evidence to support their conclusions — let's "create our own evidence." Hence — animal studies!) Therefore, the people fighting PCB turn to animal studies to build their case.[1] Due to the superabundance of animal studies, it would be repetitious and hence boring to cite all those studies here. We will concern ourselves, therefore, with looking at the results of the more representative and widely cited studies.

ANIMAL DOSAGES

In order to understand the results of these animal tests, a comparative knowledge of dosages is needed. The NIOSH criteria document cites a 1975 paper presented before the EPA in a national hearing on PCBs as estimating that the "general U.S. population has a dietary intake of PCBs of between 10-20 μg/day."[2] Inhalation of the air, NIOSH says, may add up to another 7/μg/day (100μg/m3 x 50 liters/min. . . .). Taking maximum figures, an average person in the U.S. will take in 27/μg/day, or for a 154 pound person, or approximately .39/μg/kg of body weight/day. Whichever figures are used, it still works out to be about 0.01 grams intake/year. Now compare this with the amounts fed these animals.

Dr. Renate Kimbrough, the toxicologist widely quoted by environmentalists concerned about PCBs, has done extensive testing on rats and mice. In one experiment in 1975, Dr. Kimbrough and Dr. Linder experimented on BALB/cJ male mice. The mice were fed Aroclor 1254 over eleven months. The average PCB intake was 49.8 mg/kg/day; the equivalent of over 2.5 pounds for an average person, or 129,111 times what one, at the maximum, would be expected to eat. Adenofibrosis was identified in the livers of these mice. "These lesions, according to the authors, may or may not be precursors of malignant lesions."[3]

In the following year, Dr. Kimbrough fed Aroclor 1260 (60% Chlorine, by weight; Aroclor 1242 would be 42% chlorine, by weight) to 200 rats in their diet for 21 months. The amount consumed was from 5-10 mg/kg/21 months.[4] This is 20-40 times what an average person (154 pounds) would consume in that same period. On 170 rats, elevated nodules were found on the livers. Twenty-six of these were hepatocarcinomic (compared to one in the control group) and the other 144 had "hepatocellular nodules that were described as characteristic of neoplastic nodules or synonymously, hyperplastic nodules. The authors said that 'neoplastic nodules are part of the spectrum of response to hepatocarcinogens and must

be included in the evaluation of tumorigenesis.' "[1] Hence came the cry that PCBs might cause cancer (Note: that is, cancer of the liver). (see page 10). Monsanto had previously conducted tests with Aroclor 1260, as well as with Aroclors 1242 and 1254 in which liver injury was produced, but no cancers were seen. They, therefore, did a reexamination "of the original liver slides from the Monsanto studies as well as additional liver sections from all rats in the Monsanto 2-year feeding studies."[1] The slides were independently examined by Prof. W. Richter of the University of Chicago, by Dr. D. Gordon of Industrial Bio-Test Labs, and by Prof. Parvis Pour of the Eppley Institute for Research in Cancer. Dr. Pour also examined sections of liver from the rats in Dr. Kimbrough's study. No carcinomas were seen in Monsanto's rats, and Dr. Pour did not agree with Dr. Kimbrough's diagnosis of liver cancer in her rats." So, at the very least, this study of Dr. Kimbrough's that has been cited as proof of the potential carcinogenicity of PCBs has to be viewed with serious reservations.

Another 1974 study done by Linder, Gaines and Kimbrough involved feeding rats Aroclor 1254 at 5 ppm or Aroclor 1260 at 100 ppm level. The study covered a "two-generation period" and no effects on reproduction were observed."

Another of the "big guns" of the environmental groups opposing PCB use is Dr. J. R. Allen. Although he has also done studies on rats, he and his co-workers are cited most often with respect to their experiments on rhesus monkeys. In their 1973 experiments, Aroclor 1248 or Aroclor 5460 was fed to the monkeys for 12 weeks. Each set of monkeys had access to 400g daily food supply laced with 300 ppm and 5000 ppm of PCB, respectively. This means that in a 23 week period, these monkeys received 10g and 168g, respectively. It would take a U.S. resident 1,014 and 17,047 years, respectively to consume this much PCB. The results for both groups will be put together for this one part. The monkeys lost, on an average, between 19 and 26% of their body weight, they lost hair, had puffy faces, edematous lips, and swollen eyelids with purulent exudates around the eyes. At necropsy, extensive alopecia, acneform lesions of the skin, subcutaneous edema and liver hypertrophy with fatty infiltration were evident."

Another study by Allen on 12 female rhesus monkeys fed 26 ppm Aroclor 1248 in an unspecified diet turned up some reproductive problems. The average weight of the monkeys was 5.6g. If these monkeys ate 300g of food a day, two-thirds of the amount used in the experiment on 1973 monkeys, then after the two month feeding time, they consumed about 457.5 mg, or 1.34 mg/kg/day. This is equivalent to 3472 times what a 154 pound person would normally eat."

Allen's 1976 study, done in conjunction with Deborah Barsotti, has been cited as an example of what will happen with low doses over a long time period. At first glance, it would seem to fit that category, for they fed the rhesus monkeys 2.5 and 5.0 ppm Aroclor 1248. But then it turns out that the total amount of PCBs fed the female monkeys was 180 mg and 364 mg respectively. This was over a six month period. Taking 6 kg as the average weight (in the 1974 experiment, the average weight was 5.6 kg — 6 kg will make the numbers more conservative). This works out to 30 mg/kg/6 mos, some 426 to 862 times the amount an average U.S. resident would consume. In fact, it would take a 154 pound woman 213 years to consume that much (adjusted for weight) given a normal diet — well past a woman's normal childbearing days. And that was what this study tested for: Reproductive problems. Conceptions did occur, some abortions, a still birth and other various problems also occurred. The babies that did survive showed signs of PCB toxicity after 2 months. (Acne, swollen eyelids, increased skin pigmentation)."

The following animal studies have been included in this paper not because their results or test methods show much deviation from the previously cited studies, but because NIOSH includes them in their criteria document and we wish to give our opponents' arguments enough exposure so that they cannot criticize our position by saying we blew down a straw man.

Allen, 1975, rhesus monkeys. Gave them a single 18 mg/kg dose of the above mixture. It would take an average U.S. resident 128 years to consume this much PCB. "No overt clinical effects were seen in the treated monkeys . . . Only the adipose tissue and adrenals had high PCB assays, and microscopically, except for a moderate proliferation of hepatocytic SER, all tissues were normal."[1]

Bruckner, 1973, rats. Gave them a single dose by oral intubation of either 2.5 g/kg or 6.0 g/kg Aroclor 1242. A 2.5 g/kg dose for a 70 kg man (154 pounds) is 4.6 ounces, or 175 g. Both groups got sick and the 6 g/kg group eventually died. "The conditions of the rats receiving the lower dosage gradually improved after the first 24 hours and was normal at the end of 72 hours."[1]

Barcay, Cobb and Cherry, 1974, rats. This was an inhalation study in which the rats inhaled air that contained 2.54 mg/liter PCB (2500 times the allowable amount for humans), for six hours. "During exposure there were repeated episodes of blinking and sneezing. Signs of irritation disappeared after cessation of the exposure. During the subsequent 14-day observation period, food and water consumption and growth of the rats were considered by the authors to have been similar to those of the controls. No gross pathological changes were seen when the rats were killed 14 days after removal from exposure."[1]

These next three studies can be found in a booklet published by the State of California. No mg/kg amounts were given in the reports, and no standard for determining that from PPM seems to work adequately in those cases where both are known, so only PPM figures will be given. Anyway it is the results of these tests that are important.

. . .

Conclusion to appear in September issue.