CARCINOGENIC RISK ASSESSMENTS
OF
POLYCHLORINATED BIPHENYLS (PCBs)

Executive Summary

This document presents the HERD risk assessment for carcinogenicity of PCBs and summarizes the results of four previous PCB risk assessments for cancer conducted by FDA, OTA, and CAG/EPA, and OTS. (Unfortunately, no consolidated assessment can be developed from these sources, because of the different units and different techniques used. However, the results from these various risk assessments are consistent, though not directly comparable).

In light of these earlier assessments we chose studies by NCI (three positive dose levels) and Kimbrough (one positive dose level), respectively, from which to extrapolate carcinogenic risks at low exposures. The NCI study has been criticized by other authors as having too few animals in each sex-dose group; however, the study was used because no consistent differences or trends in responses by sex were found

and with pooled data for males and females it allows the best characterization of dose-response, unlike the Kimbrough study with only one dose level. Our results are within a factor of three of the FDA risk assessment of carcinogenicity of PCB, which used both Kimbrough's data and the NCI data.

Estimates of human exposure come from the Exposure Evaluation Division of OTS. Other relevant features of the risk assessment are: 1) The extrapolation distance between the lowest experimental dose (1.25 mg/kg/day) in the NCI study and many of the exposure estimates can be very large. The larger the distance the less confidence one might have in the assessment. 2) The dose-response data for total malignanciess are also linear, this corresponds well with the "linearized" upper 95% confidence limits from the CAG model.

For regulatory purposes and for the purposes of comparing risks of PCBs with those for other chemicals, it is suggested that the number from the 95% upper confidence bound found in the multistage model in that table be used. This model is routinely used by CAG to set air and water quality criteria and standards. The estimates of exposure for which carcinogenic risks are calculated are based on conservative assumptions. Few, if any, individuals will be exposed to these relatively large amounts of PCBs. The carcinogenic risks calculated for

typical members of exposed groups are believed to be no higher and probably much lower than those presented in this document.

Previous Quantitative Cancer Risk Assessment

This memorandum summarizes and pulls together for the first time the information available from previous quantitative risk assessments of PCBs, and updates and fills in gaps in that area. It concludes with on an HERD risk assessment of PCBs.

The first risk assessment to be summarized is that conducted by the EPA Carcinogen Assessment Group (CAG) for the Office of Water Regulations and Standards, Criteria and Standards Division (EPA, 1980). This risk assessment was used to set an ambient water quality criteria level as shown in Table 1. (CAG Table page C-84). In Table 1 are the estimated concentrations of PCBs (or virtually safe doses) corresponding to lifetime cancer risk levels of 10^{-7}, 10^{-6}, and 10^{-5}. These concentrations are, as CAG said, "exceedingly low." The cancer risk levels stand for additional incidence of cancer in an exposed population. CAG used the "linearized" multistage model (Crump, 1981) which is linear at low doses or exposures so that the additional lifetime risk is directly proportional to PCB intake. CAG's risk assessment considered only the bioaccumulation of PCBs in fish and shellfish, plus exposure

through drinking water. Exposures from other food sources, air, or occupational exposures were not included in the criteria level derived by CAG using Crump's model.

For experimental data, CAG chose the study by Kimbrough et al. (1975) of Sherman rats, although CAG indicated that rats appear to be much less sensitive to the acute and subacute effects of PCBs than man or non-human primates. In 184 rats treated with 100 ppm PCB in their diet, Kimbrough observed 26 hepatocellular carcinomas compared to 1/173 hepatocullar carcinomas in controls. CAG considered all of the mouse studies of PCBs unsuitable for a quantitative risk assessment of cancer because none of them involved feeding for most or all of an animal lifetime. They also criticized the NCI study of PCBs (NCI, 1978), the only other long-term major study of PCB exposure that suggested a carcinogenic effect, as using too few experimental animals to establish a basis for assessming carcinogenic risk.

Crump conducted the PCB risk assessment (conversation with Chao Chen, November 18, 1982) but documentation of some details is incomplete. He combined the Kimbrough animal data for hepatocellular carcinoma and liver neoplastic nodules, the two tumor types observed in the Kimbrough study. At control the proportion responding was 1/173, but at the high dose the proportion responding was 170/184. Crump assumed that the

average weight of a test rat was 400 grams resulting in a conversion from 100 ppm to 4.42 mg/kg/day. (CAG normally assumes rats weigh 350 grams).

The formula for calculating VSD's for humans is then

$$VSD = \frac{70 \times 10^{-5}}{q_2 \times (2 + 0.0065kg \times 46{,}000)}$$

where 2 is a daily consumption of 2 liters of drinking water, 6.5g is an assumed daily consumption of fish and shellfish, 46,000 is the bioconcentration factor (because of the size of this factor all of risk comes from comsumption of fish and shellfish, not water), 10^{-5} is the presumed acceptable lifetime risk, and 70kg is the weight of a human. The above formula appears on p.353 of the Friday, November 28, 1980, Federal Register EPA Water Quality Criteria Document; Availability (U.S. EPA 1980), and q_2 is the potency calculated from Crump's model using 1/173 at 0 mg/kg/day and 170/184 at 4.42 mg/kg/day. Thus, q_2 for rats = .69 and q_1 for humans is achieved by multiplying by a species conversion of 5.85. This gives 4.01. Thus VSD = 5.8×10^{-7} mg/l or 0.58ng/l. This is close to .79ng/l, the value derived by CAG for 10^{-5} risk in (Table 1). The small difference between CAGs risk assessment

and our reconstruction of it is unexplained, perhaps Crump made an adjustment for the number of days in the experiment (730) versus the numer of days the animals were exposed (645).

CAG noted several drawbacks of the Kimbrough study. Since it had only one dose level it could not provide any evidence of the shape of the dose-response curve and it tested one species, one sex, and only one commercial mixture of PCBs. CAG added that Kimbrough's experimental design was good in that the dosing was for a large proportion of the lifespan of the animals, the food route was appropriate, and the dose over time was uniform. Also, there was good documentation of the intake dose, a sufficient number of test animals were used for statistical tests of increases in tumors, and a thorough description of the pathology was provided. Finally, CAG noted that an "acceptable no carcinogenic level" could be established with greater certainty if better quantitative data on carcinogenicity had been available. They also felt that studies with larger numbers of animals designed to measure relatively small effects were needed.

The second risk assessment to be summarized is that conducted by the PCB Risk Assessment Work Force of the Food and Drug Administration (Cordell et al., 1982). This group used both the Kimbrough (1975) data and the NCI (1978) data to estimate carcinogenic risk to humans from consumption of fish

contaminated with PCBs. FDA used linear extrapolation from high doses in the animal studies to the lower exposures of consumers of PCB contaminated fish and water. Linear extrapolation was defended by the authors on the grounds that one cannot deduce or directly observe the shape of the low end of the dose response curve with precision, and among the available methods, linear extrapolation from high to low doses appears to be consistent with what is known about the biological mechanisms of carcinogenesis as well as least likely to underestimate cancer risk. They also point out that linear extrapolation as used by Crump is the limiting case (upper confidence limit on risk) on his multistage model at low doses. Hence, "linearized" multistage model (Crump, 1981). Instead of using the entire Crump's model, however, the authors placed 99% upper confidence bounds on the animal response data to control for the effect of sample size so that they could make comparisons between experiments. Use of upper bounds on risk along with linear extrapolation involved an additional degree of conservatism in their risk assessment.

An additional element of uncertainty was brought out by the FDA that was not considered in the CAG assessment. That is, there is an absence of toxicity data on the particular set of PCBs that occur as residues in fish. Due to environmental transformation, the chemical composition of PCB residues found in fish differs from that of industrial PCB products (e.g.,

Aroclor 1254 and 1260), although a typical PCB residue in fish resembles the Aroclor 1254 mixture more closely that it does the other Aroclors. For this reason it was uncertain that the available toxicity data from Kimbrough and NCI represented the toxicity of the PCB mixture ingested by humans who consume fish.

FDA's upper confidence limits on risk were thus computed from the NCI Aroclor 1254 Fisher 344 rat data, for both sexes combined, on total malignancies and liver carcinomas plus adenomas. These data are shown in Table 2 (FDA Table 5). However, estimated risks from the one sex Sherman rat data on Aroclor 1260 from the Kimbrough study were also computed. Because the relative susceptibilities of humans and experimental animals to the carcinogenic effects of PCBs were not known, FDA noted that the risk estimates could obviously severely over-estimate or under-estimate human risks. The FDA risk estimates from the NCI and Kimbrough data, however, demonstrated remarkable agreement showing that the various rat strains used reacted similarly to PCB carcinogenic insult. These risks are presented as rates per 100,000 in Table 3 (FDA Table 6) for various tolerances or "acceptable daily intakes" of PCBs and percentiles of fish-eating populations. Exposures in ng/l or ppb are not directly given. Therefore it is difficult to compare the result of this risk assessment with that of CAG's (already discussed) or that of OTA's (to be

discussed). Table 4 (FDA Table 7) was derived by assuming that the carcinogenic risk is evenly distributed over a 70-year period and by multiplication of the size of the fish-eating population at risk times the risk estimates shown in Table 2. FDA also noted that our Table 3 (from their Table 6) may underestimate risks to neonates who receive PCBs in mother's milk, but who also continue to consume food contaminated with PCBs after childhood and throughout their lives.

The third risk assessment to be summarized was prepared for the U.S. Congressional Office of Technology Assessment by Kenneth Crump, who was involved in the first risk assessment discussed here (Crump and Masterman, 1979). Crump selected three data sets to use in his computation of virtually safe doses by extrapolation of the results of animal experiments to low dose levels. These data sets are shown in Table 5 (OTA Table 11). Crump's rationale for selection of data sets as the basis for risk extrapolation was as follows. First, Crump agreed with CAG in not using the NCI 1978 study was not used because it involved relatively few animals (only 24 per dose group, whereas the usual NCI bioassay uses 50 (NCI, 1978)). Second, the Kimbrough study was used because it provided the most convincing evidence of the carcinogenicity of PCBs (Kimbrough et al., 1975). The study involved a relatively large number of animals and the increased incidence of hepatocellular carcinomas in the treated group was highly

significant. This study was used with two endpoints, hepatocellular carcinomas and liver neoplastic nodules. Third, Crump used the Industrial Biotest study (Industrial Biotest Laboratories, 1971 from an unpublished report. Reasons for this choice are not clear (Some of the data from this testing company have been found to be fradulent). The slides from this study were examined at least twice, with markedly different results. Even so, the rediagnosis of the liver pathology had indicated a significant tumorigenic effect. There was also unusually high mortality and interim sacrifices further reduced the numbers of animals on test.

He then selected several high-to-low dose extrapolation procedures: the probit model (Mantel, et al., 1975), the one-stage or one-hit model (Crump et al., 1977) and the multistage model (Crump, 1981). The gamma multihit model (Rai and Van Ryzin, 1978) was also considered but could not be used: it requires data with at least two positive experimental doses and thus could not be applied to either of the Kimbrough and datasets, and when the computer algorithm for computing the estimates failed to converge for the third data set (Industrial Biotest).

Table 6 (OTA Table 12) presents the computed maximum likelihood estimates in ppb of the lifetime dose required to produce additional risks over background of 10^{-8}, 10^{-6}, and

10^{-5}. Recall that CAG considered risk levels of 10^{-7}, 10^{-6}, and 10^{-5} for its virtually safe doses (Table 1). Lower 95% confidence bounds for each of these virtually safe doses were also computed from various models. Likewise, as mentioned earlier, the gamma multihit model was applied only to the third data set since this model could not be applied to data containing only one experimental dose. In the case of one experimental dose, however, the one-hit model and multistage model produced identical results.

Crump believed that the Kimbrough study provided the most convincing evidence of the carcinogenicity of PCBs, he used these data to calculate risks for estimated PCB exposures. The fact that only one dose level of PCB was tested in the Kimbrough study meant that the one-hit model was the only one that could be utilized. Crump then converted PCB exposure levels from the the FDA Total Diet Study (Johnson and Marshe, 1977) to equivalent exposures in rats. The FDA Total Diet Study exposure levels come from a market basket of food (representing the basic 2-week diet of a 16-to-19 year old male) which was collected in each of several geographic areas. The various foods were prepared in the manner in which they could normally be eaten and were then analyzed for the presence of various substances, including PCBs. The estimates of dose from this study were felt to be tenuous, however. Exposures to PCBs in Michigan sport fish came from a 2-year

(1973-1974) study made under an FDA contract (Humphrey, 1977) of persons who regularly consumed PCB-contaminated Lake Michigan sport fish and randomly selected persons who did not consume such fish (Humphrey, 1977).

Crump's then fitted a one-step model to the animal data to estimate the extra risk of hepatocellular carcinomas at this dose. Crump did not mention allowing for any difference between the composition of PCBs in food to which humans are exposed being and the composition of Aroclor 1260 used in the experiment. But, neither CAG nor FDA considered the difference between the types of PCBs in fish and Aroclor 1254 and 1260. Crump did comment that PCB levels in human adipose tissue are likely to be much greater than corresponding levels in rats for a given level of dietary exposure, but this also was not used in the risk estimates. The estimates are given in Table 7 (OTA Table 13). Upper confidence bounds on these risks are not listed, but could as the author said that they could be obtained by multiplying all corresponding risks by 1.5.

From the estimates in Table 7 Crump computed that nationwide exposure at the dietary level detected in the 1976 Total Diet Study (3.3 µg/day to 8.7 µg/day) would cause 4.1 to 11.1 extra hepatocellular carcinomas in the U.S. Crump also used the one-hit model with estimates of PCB levels in Lake Michigan fish, to find an average excess of 0.22 hepatocellular

carcinomas among residents of Michigan County exposed to PCBs in Lake Michigan. Crump did not indicate hepatocellular carcinomas in rats might translate into cancer at other sites in man. Crump went on to estimate the risk of hepatocellular carcinoma in breast-fed infants and concluded that the risk could be at much higher than for the U.S. population in general.

Comparison of results from the FDA, OTA, and CAG risk assessment is difficult due to the different units in which risk is expressed in each assessment and the paucity of explanation in each risk assessment of some crucial assumptions that may have been made in each risk assessment. Table 8 presents the extra lifetime risks of cancer associated with consumption of PCBs in food calculated by FDA and OTA in their separate risk assessments. CAG's risk assessment is not compared here as they presented their data only in terms of virtually safe dose (Table 1). Differences are readily seen. These are primarily due to the different assumptions made in the amount of PCBs that would be ingested and in the size of the exposed population. FDA's risk assessment applies to the 15% of the total U.S. population expected to consume the fish species known to be the most highly contaminated with PCBs. The OTA calculations are based on FDA's Total Diet Study (3.3 and 8.7 µg/day) and on estimates of the PCB intake of people who consumed more than 24 lbs. of Lake Michigan sport fish per year.

CAG and OTA both used the Kimbrough experimental data. However, CAG used conversions for exposure concentrations due to bioaccumulation levels in aquatic organisms which OTA did not use. All three risk assessments are in general poorly documented and justified. It is not always clear whether estimated virtually safe doses or increases in risk apply to for animals or humans. None of the three published risk assessments explicitly refers to species-to-species conversions (or any other conversions) applied to either the exposure estimates or the risks from the model. Though we learned from personal communication that CAG did use species-to-species conversions.

FDA used linear extrapolation while OTA (Crump) used the one-hit model, but this difference, is not great because the one-hit model is nearly linear at low doses. Another difference is that FDA used the NCI study on Aroclor 1254 and while OTA used the Kimbrough study on Aroclor 1260. Not only were the number of dose levels different, but the responses and tumor types were different. Finally, CAG, FDA, and OTA probably used different exposure assumptions about the bioaccumulation or bioconcentration levels in aquatic organisms. It is at least known that CAG used a bioconcentration factor of 46,000.

HERD Risk Assessment

We now updated and expanded the quantitative risk assessments conducted by CAG, FDA, and OTA. Oncologists from the HERD Oncology Branch reviewed the NCI report "Bioassay of Aroclor 1254 for Possible Carcinogenicity" and extracted the detailed tumor data presented in Table 9. Categories for leukemias, malignant lymphomas, liver carcinomas, stomach adenocarcinoma and gastrointestinal tract malignancies, and all malignant tumors combined were established. The data are presented for males, females, and sexes combined.

The categories of stomach adenocarcinoma and gastrointestinal tract malignancies have an additional combined subcategory, labelled "Morgan, et al.". Morgan et al., 1981 reviewed the specimens of the stomachs of the Fischer 344 rats in the NCI study of PCBs and found an incidence of adenocarcinoma of the glandular stomach of 0/47, 1/48, 3/48 and 2/48. The "Morgan, et al." subcategory for stomach adenocarcinoma includes the tumors in the combined subcategory because Morgan included but did not review the adenocarcinomas of the glandular stomach already noted by NCI scientists. The Morgan, et al." subcategory for gastrointestinal malignancies is also a summation of the combined subcategory alone and the Morgan, et al. data.

The tumor categories in Table 9 correspond with the categories in Table 2 (used by FDA for animal data in risk extrapolation to humans) except that FDA combined liver adenomas with carcinomas, and leukemias with malignant lymphomas labelled hematopoietic neoplasms by FDA). Liver adenomas are frequently defined to be benign. (Personal conversation with scientists in the Oncology Branch). Note also that a summation of all the tumor categories in Table 9 beginning with leukemias would not be expected to add to the category of any malignancy. This is because any animal may have had more than one type of tumor. Thus, perhaps any malignancy would best be called "at least one malignancy." Also, not all the tumor types observed in the NCI study are listed in Table 9, only those that were malignant.

In parentheses after some of the tumor incidences in Table 9 are p-values derived using the Fisher Exact Test (Cox, 1970). In the cases where more than one Fisher Exact Test p-value has been calculated in a given row, a simultaneous comparison using the same control group has taken place and the Bonferroni inequality was used to maintain a significance of $p = .05$. This occurs for the following reason. When results for a number of treated groups are compared simultaneously with those for a control group, a correction is usually employed to ensure the overall significance level that one has chosen. The Bonferroni inequality requires that for k treated groups the p-

value for such simultaneous comparisons be less than or equal to p-value/k. In the case of Table 9 a p-value or α-level of 0.05 was chosen. The only dose levels or categories which were statistically significant at $\alpha = 0.05$ were the combined data by sex at 100 ppm in feed (or 5.00 mg/kg/day) for any malignancy.

NCI's report on their PCB bioassay (NCI, 1978) states on pp. 25-26,

> "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, [hyperplastic nodules, adenomas, and carcinomas] in both male and female rats was related to treatment. In addition, the carcinomas of the gastrointestinal tract may be associated with treatment in both males and females [none of these lesions were found in control animals in this study."]

NCI did not establish a category of "any malignancy'; therefore they did not conduct any test of statistical significance for such a category. However, as shown in Table 9 the p-value for the statistical significance of the 21/48

animals with any malignancy at 5 mg/kg/day compared with 9/48 animals in control was highly significant at 0.0074. The data for female rats at 1.25 mg/kg/day were also significant; it is not clear whether this is a statistical aberration, as none of the higher doses was significant.

Leukemias and malignant lymphomas are an important tumor category in this study. 66.1 percent (41/62) of the animals with any malignancy had one of these tumor types. However, this category is difficult to study because of the high background incidence of these malignancies in Fisher 344 rats. Historically (up to 1979, at least), 6.5 percent of the male rats and 5.4 percent of the females have had spontaneous occurrences of leukemias or lymphomas (Gart et al., 1979). Similar rates in the 1978 NCI bioassay were 20.0 (19/96) percent in males, and 23.0 percent in females. These differences suggest that the significant trend observed in the NCI study reflected a real increase, but additional work would be needed to settle the matter.

Two additional tests were conducted of the data at each subcategory, males, females, and combined sexes. The technique used to derive both tests comes from Armitage (1955). The first test is a test for departure from linear trend. If the p-value for departure from linear trend is small, then the null hypothesis of linearity (whether the association between dose

and response is a linear one) is rejected and one concludes that dose affects cancer incidence but in a manner more complete than linear. Otherwise, not significant (N.S.) is indicated. For the NCI data only, the female data for any maligancy is rejected at $p < .05$ as being linear in dose-response. However, small numbers of animals and respondents in some tumor categories make interpretation of this test difficult. The second test determines whether the (linear) slope of the dose-response curve is different from zero at a one-tailed level of significance. If the p-value for slope is small then the inference is that the slope is significantly different from zero (in a positive direction), indicating a tendency for increasing values of dose to be associated with increasing values of response. Not significant (N.S.) is indicated if the p-value is greater than 0.05. The categories of "any malignancy" for males and combined sexes are highly significant for slope at $p = 0.002$ and 0.005, respectively. For the other tumor categories small numbers of animals and responding animals present a problem of interpretation, though high significance was observed for slope in the data for males with leukemias and combined sexes with malignant lymphomas.

One additional test was conducted on the dose-response data for any malignancy to determine whether there was a significant difference between the male and female responses across dose levels. The method used was Cochran's method of

standardized difference (Cochran, 1954). No statistically differences (at an α-level of 0.05) were found. This test was primarily conducted to see if there would be problems in using the combined data for any malignancy as a basis for risk extrapolation, and it was determined there would be none from a statistical viewpoint.

There are several reasons for using the "any malignancy" data as a basis for risk assessment. First, they are markedly statistically significantly higher at the high dose, the test for positive slope is highly significant and there is no significant departure from a linear trend. These data also have denominators of 48, a larger number than the 24 animals of each sex, so that confidence limits an extrapolated dose-response curve would be narrow. FDA used the identical categories of total malignancies in Table 2 as a basis for part of their risk assessment. Available risk extrapolation models do not preclude the use of data where there are no statistically significant increases over control at any (or even all) dose levels, though there may be a substantial impact on p-values and confidence limits. Better discrimination between these models also can be obtained if there is an ample range of doses over which the response is noted. Measures of goodness-of-fit of the models to the available data are also more meaningful with a larger number of different dose-response data points.

NCI did not analyze the data for "total malignancies" or "any malignancy". Since NCI stated that "under the conditions of this bioassay, Aroclor 1254 was not carcinogenic ..." the establishment of a tumor category where significance occurs may seem to be like "fishing" for statistical significance. We do not regard this as a serious objection because there is solid independent confirmation of carcinogenicity from the Kimbrough study (1975), presented in Table 5, where the high incidence of hepatocellular carcinomas was observed 26/184 at 5 mg/kg/day compared to 1/173 at control (highly significant by the Fisher Exact Test at $p < 0.0001$).

A high incidence of liver neoplastic nodules was also observed by Kimbrough with 146/184 at 5 mg/kg/day compared to 0/173 at control (so obvious that no statistical test of significance was reported). However, like adenomas, neoplastic nodules are not malignant and the significance of their appearance is not entirely clear. (Personal conversation with scientists in the Oncology Branch).

A question arises here: why were the tumor types between the NCI study and the Kimbrough study so different? The main tumor types observed in the National Cancer Institute (NCI) Bioassay on PCBs were, in order of decreasing incidence, hematopoietic neoplasms (leukemias and malignant lymphomas), liver carcinomas, stomach adenocarcinomas, and gastrointestinal

tract malignancies. On the other hand, hepatocellular carcinomas were the main tumor type identified by Kimbrough. A possible explanation for this difference is that two different Aroclors were tested in two different strains. The NCI studied Aroclor 1254 in Fisher 344 rats and Kimbrough used Aroclor 1260 on Sherman rats. We do not know what the polychlorinated dibenzoturon concentrations were in these two studies. (Kimbrough, 1975 and NCI, 1978) The degree of variability between different strains within a species can be as high as the degree of variability between different species. Also, while the Aroclors are mixtures they differ in both the degree and structural location of chlorination; for Aroclor 1254 the major component (53%) is $C_{12}H_5Cl_5$ and for Aroclor 1260 the major component (42%) is $C_{12}H_4Cl_6$.

The Kimbrough study will allow only the crudest forms of risk modelling as it has only one nonzero dose level. However, there is no question as to the statistical significance of the increase in hepatocellular carcinomas observed by Kimbrough. The NCI study will allow much more mathematically sophisticated risk modeling and measures of goodness-of-fit of the models to the data; but, standing alone is difficult to interpret, because of the question of statistical significance of the various tumor types. Indeed, NCI concluded that its study did not establish carcinogenicity. There is also some question about the validity or advisability of using the category of

"any malignancy". FDA combined conclusions from the two studies to estimate cancer risks to humans. That is also the course that this assessment follows.

Table 10 presents maximum likelihood estimates and lower 95 percent confidence limits on dose for a range of excess risks from $1x10^{-1}$ (1 in 10) to $1x10^{-8}$ (1 in 100,000,000) (hereafter called virtually safe dose). The data used here are the Kimbrough data for hepatocellular carcinomas. Because Kimbrough tested only one nonzero dose level, the only models that could be used were one-hit or simple linear extrapolation. As linear regression gives nearly the same results as the one hit models at low doses, only one-hit extrapolation was employed. The computer program used was that for the multistage model, which degenerates to a one-hit model when only one positive dose level is available. The confidence limits on these virtually safe doses are tight, roughly within a factor of 1.5 of the point estimates at a risk of $1x10^{-6}$ (1 in 1,000,000).

Table 11 presents a similar range of excess risks of any malignancy over background based on the NCI PCB Bioassay, derived using five of the most widely known and used extrapolation models: The multistage, logit, probit, Weibull, and gamma-multihit. The logit and the probit ordinarily are expected to give similar results, because of their similar functional form. Estimates from the one-hit model were similar

to those from the multistage model. Simple linear extrapolation is approximated by the lower confidence limits on virtually safe doses (that is, upper confidence limits on risk) from the multistage model. Two types of background were assumed for incorporation in all models except the multistage. Independent background implies that any ongoing background carcinogenic processes are independent of the carcinogenic response induced by the administration of the carcinogenic agent under study. Additive background implies, on the other hand, that cancers induced by administration of the chemical under study add to already on-going carcinogenic processes, as if arising from an effective background dose, acting by the same mechanism and producing similar types of tumors. It is not clear at this time which of these two is the more valid assumption, therefore for the four models available that consider both types of background, both are presented. The multistage model is consistent with both assumptions so, traditionally, one does not speak of it as either additive or independent. Goodness-of-fit p-values were also calculated to give an idea of the fit of the model to the data.

It can be seen from of Table 11 that the estimates of virtually safe dose at a risk of 1×10^{-6} (1 in 1,000,000) from the independent background models vary a million fold from 10^{-2} for the probit to 10^{-8} for the gamma-multihit. The additive background models, including the multistage model, are all in

the area of 0.16×10^{-4} to 0.17×10^{-3} mg/kg/day. The goodness-of-fit test results show the multistage model to be the model with the best fit to the data ($p > 0.9000$) and the additive gamma-multihit, the worst fit (although it is still quite adequate) at $p = 0.5391$.

Tables 12 and 13 present the results of two suggested methods for placing confidence limits on virtually safe dose, one by using the variance of log-dose, the other by using the variance of the reciprocal of dose. The confidence limits in Table 12, based on the variance of log dose are generally much wider than those in Table 13, based on the variance of the reciprocal of dose. Confidence limits from the independent models vary widely, while those from the additive models are within factors of 5 of each other by the two methods and are within factors of 4 to 20 of their maximum likelihood estimates at a risk of 1×10^{-6}.

The confidence limits for the multistage model, calculated by a method unique to that model, are within a factor of 1.8 of its maximum likelihood estimate of virtually safe dose at a risk of 1×10^{-6}. Thus the multistage confidence limits are much more narrow or "tight." See Figure 1, which shows upper confidence limits on risks, rather than lower confidence limits on dose.

Table 14 presents, for a range of excess risks from $1x10^{-1}$ to $1x10^{-8}$, a table of maximum likelihood estimates for leukemia alone over background based on the NCI PCB bioassay. Leukemia alone was chosen because of the high percentage of pathology of this type in the NCI bioassay. The risks predicted by the same range of independent and additive background models are within factors of 5 of similar estimates based on any malignancy. Again estimates of confidence limits on virtually safe dose based on the variance of log dose vary more widely than estimates based on the variance of the reciprocal of dose. The multistage estimates are also narrow and this model produces the best fit among the models examined. Overall there is much consistency between the estimates based on any malignancy and those based on leukemia alone.

We now consider how estimates of virtually safe doses for risk of hepatocellular carcinoma from the Kimbrough study compare with estimates of virtually safe dose based on any malignancy or leukemia alone from the NCI study. Based on the multistage, the best fitting model, the maximum likelihood estimates from Table 10 compare very well (within a factor of 1 to 2) with each other, the confidence limits within a factor of 1 to 3. It seems then that it makes little difference which study is used, Kimbrough or NCI, to base quantitative estimates of cancer risk to humans. The decision on which study to choose for extrapolation might be based on the likelihood of

exposure to a particular type of Aroclor since apparently the tumor types were different for tests of Aroclor 1254 and 1260, though strain of animal may have been just as important. The extent to which one expects perfect site concordance would also dictate the choice of a study. Remember that FDA used both studies in its risk assessment of PCBs.

With these preliminary remarks and tabulations, we use for exposures of various subpopulations provided by the Exposure Assessment Branch, Exposure Evaluation Division of OTS to estimate additional or excess lifetime risk. The NCI data (used by FDA) and the Kimbrough data (used by FDA, CAG, and OTA) are used as the bases for extrapolation.

Table 17 presents the excess or additional lifetime risk estimates based on total malignancies in the NCI study. The model used to derive these excess risks was the Crump multistage model and program (used by FDA, CAG, and OTA in their assessments). A species conversion factor of approximately 5.85 was used for the transformation of rat risks to human risks; i.e., humans are presumed to be roughly six times as sensitive to carcinogenic effects of chemicals as rats. The estimated exposure duration in years was used to modify the risk estimates for the proportion of an average human lifespan of 70 years that an individual in a given exposure category might be exposed to PCBs. If the exposure

duration was 38.5 years, as in the case of exposure loading/unloading a liquid assuming PCBs are present in the liquid at 25 mg/kg then the cancer risks were reduced by a factor times 38.5/70 or 0.55 of their original size. (It would be best to base such proportion of lifetime calculations on results from a differential exposure study; however, none was available and this simplistic multiplication factor is currently the state-of-the-art in this area.) Both "most likely" or point estimates of excess cancer risk and 95 percent upper confidence limits on excess cancer risk are presented. The upper confidence limits from the multistage model exhibit linearity at low exposures. They also assume additivity. Additivity, as mentioned earlier, assumes that the effect of a carcinogenic agent is to act through the same mechanisms as that operating for background process. The upper confidence limits are not markedly more conservative than the point estimates of risk in the case of the NCI total malignancies category. Many other factors mentioned in the other Agencies risk assessments influence the results. Among these are the dissimilarity between residues in fish and water and Aroclor 1245, the levels of PCBs in human adipose tissue, and the small sample sizes in the NCI study.

The extrapolation distance between the lowest experimental dose in the NCI study (1.25 mg/kg/day, the lowest of any level in either the NCI dataset), and the highest and lowest exposure

levels is on the order of from 6 to 45,000,000,000. This is a very wide range. Keep in mind that generally the further the distance over which risks are extrapolated the less confidence one could have in those risk estimates, and that errors could go in either direction.

The assumptions used to derive the exposure levels shown in Table 17 are discussed in the exposure assessment for incidentially - produced PCBs (Versar 1983). One purpose of the exposure and risk assessments is to provide guidance in establishing a permissable level of PCBs in other chemicals. Using conservative assumptions, the exposure assessment estimated the upper limits of exposures that may result when PCBs are present as impurities in a variety of chemicals and products at levels of 50 ppm, 25 ppm, and 2 ppm. The exposure levels in Table 17 correspond to the upper limits of exposure for the various scenarios when the concentration of PCBs is 25ppm. Few, if any, individuals will be exposed to these relatively large amounts of PCBs. Thus, the maximum likelihood of excess risk in Table 17 applies to highly exposed individuals. The excess carcinogenic risks to typical members of the exposed groups are believed to be no higher than those in Table 17 and are probably much lower.

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FIGURE 1

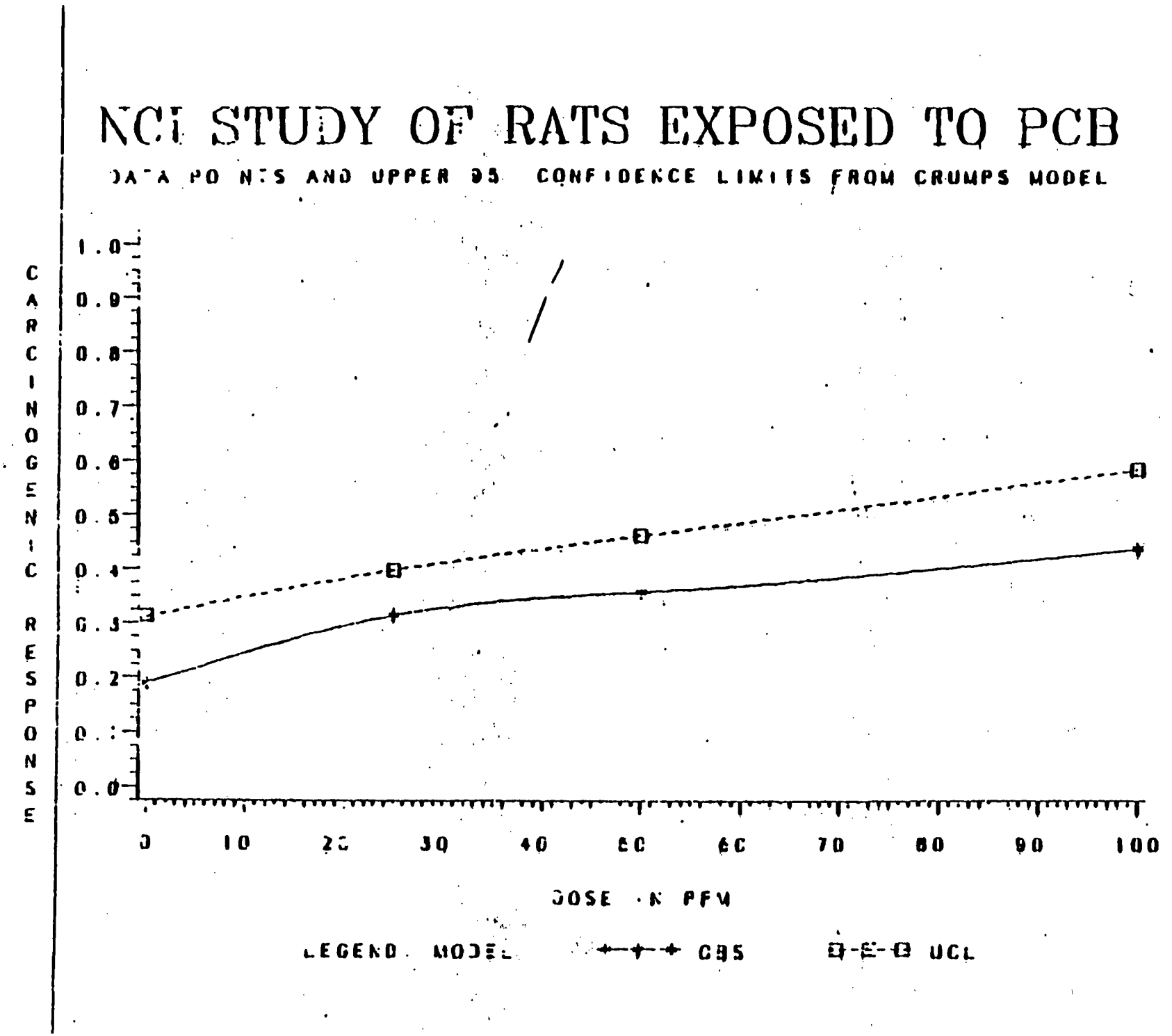
NCI STUDY OF RATS EXPOSED TO PCB
DATA POINTS AND UPPER 95 CONFIDENCE LIMITS FROM CRUMPS MODEL
CARCINOGENIC RESPONSE
1.0
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0.0
0
10
20
30
40
50
60
70
80
90
100
DOSE IN PPM
LEGEND: MODEL
CBS
UCL

FIGURE 2

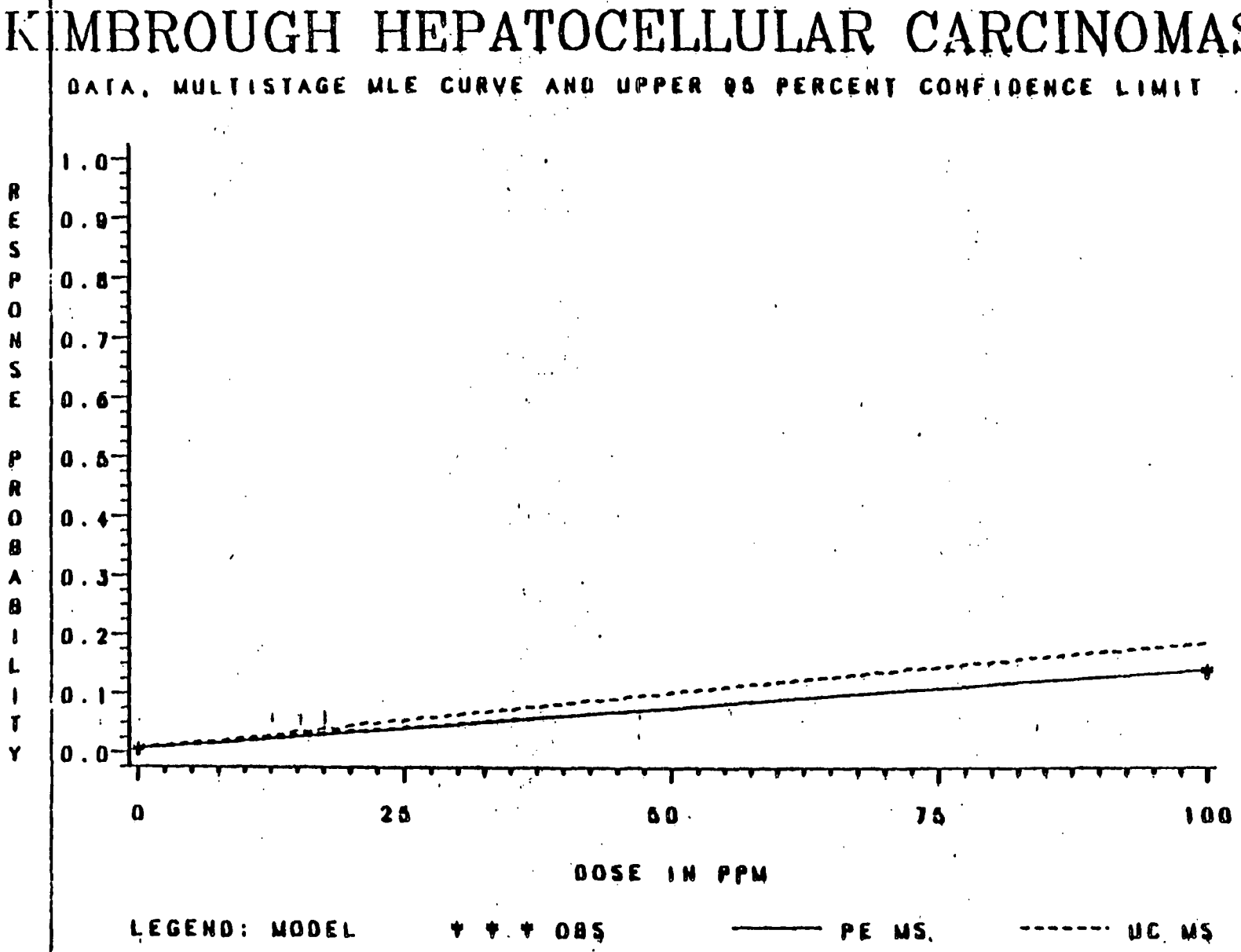
KIMBROUGH HEPATOCELLULAR CARCINOMAS
DATA, MULTISTAGE MLE CURVE AND UPPER 95 PERCENT CONFIDENCE LIMIT
RESPONSE PROBABILITY
1.0
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0.0
0
25
50
75
100
DOSE IN PPM
LEGEND: MODEL * * * OBS —— PE MS. ------ UC MS

Table 1. CAG's Table for Water Quality Criterion of PCBs

PCB Exposure	Lifetime Risk Levels and Water Quality Crite		
	10^{-7}	10^{-6}	10^{-5}
2 liters of drinking water and consumption of 6.5g contaminated fish and shellfish per day*	0.0079 ng/l	0.079 ng/l	0.79 ng/l
Consumption of fish and shellfish only*	0.0079 ng/l	0.079 ng/l	0.79 ng/l

*Approximately 99% of the PCB exposure results from aquatic organisms, which exhibit an average bioaccumulation potential of 31,200-fold. The remaining 1% of OCB exposure results from drinking water.

Table 2: Animal Data Used for Risk Extrapolation to Humans in FDA Assessment

Animal Studies	Dose of Aroclor 1254 fed			
	ppm 0	25	50	100
	mg/kg/day 0	1.25	2.50	5.00
NCI Bioassay - Fischer Fischer Rats fed Total Malignancies				
Males	5/24	2/24	9/24	12/24
Females	4/24	13/24	8/24	9/24
Combined	9/48	15/48	17/48	21/48
Liver Carcinoma & Adenomas				
Males	0/24	0/24	1/24	2/24
Females	0/24	0/24	1/24	2/24
Combined	0/48	0/48	2/48	4/48
Hematopoetic Neoplasms				
Males	3/24	2/24	5/24	9/24
Females	4/24	6/24	6/24	6/24
Combined	7/48	8/48	11/48	15/48

Table 3: Upper Confidence Limits (99%) on Lifetime Risks* of Cancer in Eaters of Fish Spe

Animal Studies on which risks are based	50th Percentile Eaters					90	
	Assuming No Tolerance		Assuming Tolerance = 5 ppm	Assuming Tolerance = 2 ppm	Assuming Tolerance = 1 ppm	Assuming No Tolerance	
	USA	Lake** Michigan				USA	Michiga
Kimbrough - Rats Liver Carcinoma	1.3	18.4	1.2	0.8	0.5	3.4	41.4
NCI Bioassay - Total Malignancies for Male and Female	4.1	58.0	3.7	2.7	1.6	10.6	

ble 3: Upper Confidence Limits (99%) on Lifetime Risks* of Cancer in Eaters of Fish Species of Interest in FDA Assessment

imal Studies which risks based	50th Percentile Eaters					90th Percentile Eaters				
	Assuming No Tolerance		Assuming Tolerance = 5 ppm	Assuming Tolerance = 2 ppm	Assuming Tolerance = 1 ppm	Assuming No Tolerance		Assuming Tolerance = 5 ppm	Assuming Tolerance = 2 ppm	Assuming Tolerance = 1 ppm
	USA	Lake** Michigan				USA	Lake** Michigan			
abrough - Rats ver Carcinoma	1.3	18.4	1.2	0.8	0.5	3.4	41.4	3.1	2.3	1.4
Bioassay - Total lignancies for Male d Female	4.1	58.0	3.7	2.7	1.6	10.6	129.2	9.8	7.2	4.4
Bioassay - Liver cinomas and Adenomas Male and Female	0.9	12.75	0.9	0.6	0.4	2.5	30.5	2.3	1.7	1.0
Bioassay matopietic for le and Female	2.7	38.25	2.4	1.8	1.1	7.0	85.3	6.5	4.7	2.9

All risks are lifetime risks computed as rates per 100,000 of the population at risk.

Risk calculated for Lake Michigan sportsfish eaters who consume an average of 1.7 µg/kg/day PCB or 3.9 µg/kg/day at the 90th percentile. Risks in other areas having similar sportsfish consumption and PCB contamination are probably similar.

able 4: Upper Confidence Limits on Number of New Cancers per Year in Eaters of Fish Species of Interest in FDA Assessment

imal studies on which isks are based	50th Percentile Eaters					90th Percentile Eaters				
	Assuming No Tolerance		Assuming Tolerance = 5 ppm	Assuming Tolerance = 2 ppm	Assuming Tolerance = 1 ppm	Assuming No Tolerance		Assuming Tolerance = 5 ppm	Assuming Tolerance = 2 ppm	Assuming Tolerance = 1 ppm
	USA	Lake** Michigan				USA	Lake** Michigan			
Imbrough - Rats iver Carcinoma	6.2	10.4	5.8	3.8	2.4	16.3	23.4	14.7	10.0	6.7
CI Bioassay - Total alignancies for Male nd Female	19.6	32.8	17.6	12.9	7.6	50.6	73.1	46.8	34.3	21
CI Bioassay - Liver arcinomas and Adenomas or Male and Female	4.3	7.2	4.2	2.9	2.0	12.0	17.3	10.9	8.0	4.7
CI Bioassay ematopoietic or Male and Female	12.9	21.6	11.4	8.6	5.3	33.4	48.3	31.0	22.5	13.8

All risks are the increased number of cancers per year for the population at risk (15.2% of U.S. population) considering a 70 year life span.

*Risk calculated for Lake Michigan sportsfish eaters who consume an average 1.7 µg/kg/day PCB or 3.9 µg/kg/day at the 90th percentile. (4,000,000 people assumed exposed). Risks may be similar for sportsfish eaters in other areas; but data not available to make estimate.

Table 5: Data sets used by Crump for OTA in calculating virtually safe doses

Data Set No.—I Kimbrough et al. (1975) rat study with Aroclor 1260—

Hepatocellular carcinomas

Dietary level (ppm)	(mg/kg/day)	No. of animals	No. animals with Hepatocellular carcinomas
0	0	173	1
100	5.0	184	26

Data Set No. II-Kimbrough et al (1975) rat study with Aroclor 1260—

Liver neoplastic nodules

Dietary level (ppm)	(mg/kg/day)	No. of animals	No. of animals with Neoplastic nodules
0	0	173	0
100	5.0	184	146

Data Set No. III-Industrial Bio-Test rat experiment with Aroclor 1260—

Liver neoplastic nodules

Dietary level (ppm)	(mg/kg/day)	No. of animals	No. of animals with Neoplastic nodules
0	0	23	1
1	0.05	25	0
10	0.5	23	9
100	5.0	27	7

Table 6: Virtually safe doses computed by Crump for OTA from data in Table 5

Data Set	Analytic Method	Maximum Likelihood estimates of dose in ppb. corresponding to extra risk of			Virtually safe doses (lower 95% confidence bounds for dose) in ppb. corresponding to		
		10^{-8}	10^{-6}	10^{-5}	10^{-8}	10^{-6}	10^{-5}
Ia	Probit	—	—	—	1.96	14.2	43.6
Ib	One-Hit and Multistage	.0069	.686	6.86	.0051	.511	5.11
IIa	Probit	—	—	—	.025	.180	.552
IIb	One-Hit and Multistage	.00063	.063	.634	.00055	.0511	.511
IIIa	Probit	—	—	—	.176	1.29	3.97
IIIb	One-Hit	.0046	.465	4.65	.00235	.235	2.35
IIIc	Multistage	.0046	.465	4.65	.00205	.205	2.05

Table 7. Estimates of lifetime extra risk to humans of hepatocellular carcinoma based on a one-hit model (2) to the Kimbrough et al. (1975) rat study (appeared in OTA document)

Human PCB Dosage	Risks calculated from converting human dose to animal dose on the basis of ppm in diet	mg/kg/day
3.3 µg/day (1976 Total Diet Study)	1/328,000	1/764,000
8.7 µg/day (1975 Total Diet Study)	1/123,000	1/288,000
127 µg/day (Avg. intake of people consuming more than 24 lbs./yr. Lake Michigan fish, Humphrey (1977)	1/8,000	1/20,000

Table 8. Extra lifetime risks of cancer associated with consumption of PCBs in food (appeared in OTA document)

Dose (ug/day)	Extra lifetime risk/100,000	Upper limit of new cancers/year
FDA[a]		
9.2[c]	4.4	21
14.9[c]	7.2	34
20.1[c]	9.8	47
OTA[b]		
3.3[d]	0.13	4
8.7[e]	0.35	11
127[f]	5	—

[a]Based on NCI bioassay and total malignancies for males and females.
[b]Based on Kimbrough (1975) study and hepatocellular carcinomas.
[c]Based on highest consumers (90th percentile) of fish species contaminated with PCBs if tolerance established at 1.2 of 5 ppm.
[d]Based on FDA Total Diet Study. 1976.
[e]Based on FDA Total Diety Study. 1975.
[f]Based on average intake of people consuming more than 24 lbs/year Lake Michigan fish.

ɔle 9: Animal Data Used for OTS Risk Extrapolation to Humans*

lignancy Category	ppm in feed	Animals Per Dose Level**				p-Value***	
		0	25	50	100	Positive Slope	p-value for departure from linear trend
	mg/kg/day	0	1.25	2.50	5.00		
/ Malignancy							
les		5/24	2/24 (N.S.)	9/24 (N.S.)	12/24 (N.S.)	0.002	N.S.
nales		4/24	13/24 (.0093)	8/24 (N.S.)	9/24 (N.S)	(N.S.)	p < .05
mbined		9/48	15/48 (N.S.)	17/48 (N.S.)	21/48 (.0074)	0.005	N.S.
ukemias							
les		3/24	2/24	5/24	8/24 (N.S.)	0.014	N.S.
males		4/24	6/24	6/24	4/24 (N.S.)	N.S.	N.S.
mbined		7/48	8/48	11/48	12/48 (N.S.)	0.080	N.S.
lignant Lymphomas							
les		0/24	0/24	0/24	1/24 (N.S.)	0.063	N.S.
males		0/24	0/24	0/24	2/24 (N.S.)	0.015	N.S.
mbined		0/48	0/48	0/48	3/48 (N.S.)	0.004	N.S.
ver Carcinoma							
les		0/24	0/24	1/24	2/24 (N.S.)	0.030	N.S.
males		0/24	1/24	0/24	0/24 (N.S.)	N.S.	N.S.
mbined		0/48	1/48	1/48	2/48	0.016	N.S.
omach Adenocarcinoma							
les		0/24	0/24	1/24	0/24 (N.S.)	N.S.	N.S.
males		0/24	1/24	1/24	0/24 (N.S.)	N.S.	N.S.
mbined (original report)		0/48	1/48	2/48	0/48 (N.S.)	N.S.	N.S.
mbined (Morgan et al.)		0/47	1/48	3/48	2/48 (N.S.)	N.S.	N.S.

Table 9: Animal Data Used for OTS Risk Extrapolation to Humans* (Continued)

Malignancy Category	ppm in feed	Animals Per Dose Level** 0	25	50	100	Positive Slope	P-Value*** p-value for departure from linear trend
	mg/kg/day	0	1.25	2.50	5.00		
Gastrointestinal Tract Malignancies (Including Jejunum, Cecum, and Stomach							
Males		0/24	0/24	2/24	1/24 (N.S.)	N.S.	N.S.
Females		0/24	1/24	1/24	0/24 (N.S.)	N.S.	N.S.
Combined (original report)		0/48	1/48	3/48	1/48 (N.S.)	N.S.	N.S.
Combined (Morgan et al.)		0/48	1/48	4/48	3/48 (N.S.)	0.044	N.S.

*From the National Cancer Institute bioassay of Aroclor 1254 except as noted. Morgan et al. (Cancer Research 41, 5052-5059, December 1981) re-examined the whole-tissue specimans of stomach taken from the NCI bioassay and presented malignancy data per combined sexes only.

**Numbers in parenthesis are Fisher Exact Test p-values for statistical significance over control. The Bonferroni inequality was employed to correct for lowered p-values due to multiple comparisons with the same control group. N.S. indicates not significant at the $\alpha = 0.0500$ level.

***The technique used to derive these numbers comes from Cochran (1954) and Armitage (1955). If the p value for positive slope is small the inference is that the slope is significantly different from zero (in a positive direction), indicating that there is a tendency for dose to be associated with increasing values of response. Otherwise N.S. (not significant) is indicated if the p-value is greater than 0.10. If the p-value for departure from linear trend is small the null hypothesis of linearity (or whether the association between dose and response is a linear one) is rejected. Otherwise N.S. is indicated.

Table 10

Excess Risk to Rats of Hepatocellular Carcinoma Over Background
(Based on Kimbrough PCB Bioassay)

Model	1.000E-01	1.000E-02	1.000E-03	1.000E-04	1.000E-05	1.000E-06	1.000E-07	1.000E-08
	Maximum Likelihood Estimates of Virtually Safe Dose for Rats (mg/kg/day)							
Multistage (One-stage)[a]	0.723180E+02	0.689833E+01	0.686703E+00	0.686392E-01	0.686361E-02	0.686358E-03	0.686358E-04	0.686358E-05
	95% One-Sided Lower Confidence Limits on Virtually Safe Doses for Rats (mg/kg/day)							
Multistage (One-stage)[a]	0.524392E+02	0.500293E+01	0.498026E+00	0.497801E-01	0.497779E-02	0.497776E-03	0.497776E-04	0.497776E-05

[a]The multistage model degenerates to the specific case of the one-hit model when only one positive dose level is available.

Table 11

Excess Risk to Rats of Any Malignancy Over Background
(Based on NCI PCB Bioassay)

Model	1.000E-01	1.000E-02	1.000E-03	1.000E-04	1.000E-05	1.000E-06	1.000E-07	1.000E-08	Goodness of fit test p-value*
	Maximum Likelihood Estimate of Virtually Safe Dose for Rats (mg/kg/day)								
Multistage	0.358387E+02	.3373694E+01	0.335445E+00	0.335254E-01	0.335235E-02	0.335233E-03	0.335233E-04	0.335233E-05	>0.9000
Independent Probit**	0.188359E+02	0.111017E+01	0.145657E+00	0.276896E-01	0.638583E-02	0.182654E-02	0.567512E-03	0.192601E-03	0.8652
Independent Logit**	0.183615E+02	0.486139E+00	0.151306E-01	0.478073E-03	0.151281E-04	0.478783E-06	0.151530E-07	0.479580E-09	0.8801
Independent Weibull**	0.179320E+02	0.327504E+00	0.654608E-02	0.131959E-03	0.266236E-05	0.537192E-07	0.108392E-08	0.218707E-10	0.8937
Independent Gamma Multihit**	0.959199E+01	0.185339E-01	0.358741E-04	0.694380E-07	0.134404E-09	0.260153E-12	0.503553E-15	0.974677E-18	0.6784
Additive Probit**	0.193480E+02	0.125692E+01	0.120492E+00	0.119985E-01	0.119934E-02	0.119929E-03	0.119929E-04	0.119928E-05	0.8536
Additive Logit**	0.192963E+02	0.123398E+01	0.117993E+00	0.117465E-01	0.117413E-02	0.117407E-03	0.117407E-04	0.117407E-05	0.8549
Additive Weibull**	0.190640E+02	0.115365E+01	0.109503E+00	0.108931E-01	0.108874E-02	0.108869E-03	0.108868E-04	0.108868E-05	0.8632
Additive Gamma Multihit**	0.215479E+02	0.166645E+01	0.162010E+00	0.161548E-01	0.161502E-02	0.161498E-03	0.161497E-04	0.161499E-05	0.5391

*Any p-value greater than 0.50 indicates an adequate fit of the model to the data.

**In many experiments the response of interest also occurs spontaneously in control animals. This background may be assumed to be either independent of the induced responses or additive in a mechanistic manner. If the spontaneous and induced responses are assumed to be independent, then the probability of observing a response of either type at dose d is given by: $P^*(d) = q + (1 - q) P(d)$, where $0 < q < 1$ denotes the spontaneous background rate. Under the additivity assumption, the background response may be considered as arising from an effective background dose $w > 0$, with $P^*(d) = P(d + w)$. Although the manner in which background response is accommodated is crucial, the extent to which independence or additivity is indicated by either biological theory or experimental data is somewhat unclear at this time.

Table 12

95.0% One-Sided Lower Confidence Limits on Virtually Safe Doses for Rats in mg/kg/day in Table 11 Based on the Variance of Log-Dose (Based on HCI PCB Bioassay)

Model	1.000E-01	1.000E-02	1.000E-03	1.000E-04	1.000E-05	1.000E-06	1.000E-07	1.000E-08
Multistage	0.201936E+02	0.190809E+01	0.189785E+00	0.189683E-01	0.189673E-02	0.189672E-03	0.189678E-04	0.189672E-05
Independent Probit*	0.220746E+01	0.346488E-02	0.294015E-04	0.586098E-06	0.197079E-07	0.950177E-09	0.598362E-10	0.463900E-11
Independent Logit*	0.189026E+01	0.394130E-03	0.999379E-07	0.256971E-10	0.658461E-14	0.168250E-17	0.429200E-21	0.109377E-24
Independent Weibull*	0.165192E+01	0.129025E-03	0.105210E-07	0.858352E-12	0.697306E-16	0.565007E-20	0.457138E-24	0.369536E-28
Independent Gamma Multihit*	0.169541E+00	0.421417E-08	0.851322E-16	0.168444E-23	0.331172E-31	0.649296E-39	0.127115E-46	0.248643E-54
Additive Probit*	0.280330E+01	0.634469E-01	0.545471E-02	0.537295E-03	0.536484E-04	0.536403E-05	0.536396E-06	0.536393E-07
Additive Logit*	0.274554E+01	0.570838E-01	0.484797E-02	0.476930E-03	0.476150E-04	0.476072E-05	0.476064E-06	0.476063E-07
Additive Weibull*	0.254950E+01	0.441988E-01	0.367362E-02	0.360619E-03	0.359952E-04	0.359885E-05	0.359878E-06	0.359878E-07
Additive Gamma Multihit*	0.497889E+01	0.190933E+00	0.171554E-01	0.169708E-02	0.169525E-03	0.169506E-04	0.169505E-05	0.169507E-06

*In many experiments the response of interest also occurs spontaneously in control animals. This background may be assumed to be either independent of the induced responses or additive in a mechanistic manner. If the spontaneous and induced responses are assumed to be independent, then the probability of observing a response of either type at dose d is given by: $P^*(d) = q + (1 - q) P(d)$, where $0 < q < 1$ denotes the spontaneous background rate. Under the additivity assumption, the background response may be considered as arising from an effective background dose $w > 0$, with $P^*(d) = P(d + w)$. Although the manner in which background response is accommodated is crucial, the extent to which independence or additivity is indicated by either biological theory or experimental data is somewhat unclear at this time.

Table 13

95.0% One-Sided Lower Confidence Limits on Virtually Safe Doses for Rats in mg/kg/day in Table II Based on the Variance of the Reciprocal of Dose (Based on HCl PCB Bioassay)

Model	1.000E-01	1.000E-02	1.000E-03	1.000E-04	1.000E-05	1.000E-06	1.000E-07	1.000E-08
Multistage	0.201936E+02	0.190809E+01	0.189785E+00	0.189683E-01	0.189673E-02	0.189672E-03	0.189672E-04	0.189672E-05
Independent Probit*	0.599122E+01	0.163994E+00	0.153195E-01	0.235394E-02	0.480037E-03	0.118077E-03	0.332556E-04	0.103875E-04
Independent Logit*	0.560906E+01	0.598872E-01	0.117040E-02	0.269306E-04	0.670718E-06	0.174903E-07	0.470663E-09	0.129557E-10
Independent Weibull*	0.529803E+01	0.370511E-01	0.456459E-03	0.664758E-05	0.104959E-06	0.173944E-08	0.297762E-10	0.521701E-12
Independent Gamma Multihit*	0.190484E+01	0.113728E-02	0.129198E-05	0.176877E-08	0.264809E-11	0.417802E-14	0.662721E-17	0.1143 19E-19
Additive Probit*	0.659938E+01	0.315316E+00	0.294234E-01	0.292219E-02	0.292019E-03	0.291999E-04	0.291997E-05	0.291995E-06
Additive Logit*	0.654126E+01	0.302931E+00	0.281468E-01	0.279417E-02	0.279213E-03	0.279193E-04	0.279190E-05	0.279190E-06
Additive Weibull*	0.632956E+01	0.270683E+00	0.249166E-01	0.247118E-02	0.246914E-03	0.246894E-04	0.246892E-05	0.246892E-06
Additive Gamma Multihit*	0.874128E+01	0.526271E+00	0.499207E-01	0.496567E-02	0.496303E-03	0.496277E-04	0.496275E-05	0.496281E-06

*In many experiments the response of interest also occurs spontaneously in control animals. This background may be assumed to be either independent of the induced responses or additive in a mechanistic manner. If the spontaneous and induced responses are assumed to be independent, then the probability of observing a response of either type at dose d is given by: $P^*(d) = q + (1 - q) P(d)$, where $0 < q < 1$ denotes the spontaneous background rate. Under the additivity assumption, the background response may be considered as arising from an effective background dose $z > 0$, with $P^*(d) = P(d + z)$. Although the manner in which background response is accommodated is crucial, the extent to which independence or additivity is indicated by either biological theory or experimental data is somewhat unclear at this time.

Table 14

Excess Risk to Rats of Leukemia Over Background
(Based on NCI PCB Bioassay)

Model	1.000E-01	1.000E-02	1.000E-03	1.000E-04	1.000E-05	1.000E-06	1.000E-07	1.000E-08	Goodness of fit test p-value[a]
	Maximum Likelihood Estimate of Virtually Safe Dose to Rats (mg/kg/day)								
Multistage	0.875538E+02	0.827988E+01	0.823603E+00	0.823171E-01	0.823127E-02	0.823123E-03	0.823122E-04	0.823122E-05	>0.7500
Independent Probit**	0.839838E+02	0.726541E+01	0.124448E+01	0.293427E+00	0.839835E-01	0.274622E-01	0.991062E-02	0.386172E-02	0.6768
Independent Logit**	0.847370E+02	0.516213E+01	0.353861E+00	0.245262E-01	0.170178E-02	0.118093E-03	0.819503E-05	0.568692E-06	0.6616
Independent Weibull**	0.851187E+02	0.481628E+01	0.289734E+00	0.175308E-01	0.106135E-02	0.642593E-04	0.389061E-05	0.235559E-06	0.6577
Independent Gamma Multihit**	0.848662E+02	0.447594E+01	0.243522E+00	0.1327111E-01	0.723295E-03	0.394208E-04	0.214850E-05	0.117097E-06	0.6562
Additive Probit**	0.837553E+02	0.566387E+01	0.544626E+00	0.542495E-01	0.542282E-02	0.542261E-03	0.542258E-04	0.542255E-05	0.6739
Additive Logit**	0.838657E+02	0.564823E+01	0.542071E+00	0.539835E-01	0.539612E-02	0.539589E-03	0.539587E-04	0.539587E-05	0.6731
Additive Weibull**	0.840371E+02	0.561226E+01	0.537097E+00	0.534719E-01	0.534482E-02	0.534458E-03	0.534456E-04	0.534455E-05	0.6718
Additive Gamma Multihit**	0.831950E+02	0.615610E+01	0.594977E+00	0.592923E-01	0.592717E-02	0.592697E-03	0.592694E-04	0.592690E-05	0.6670

[a]Any p-value greater than 0.50 indicates an adequate fit of the model to the data.

**In many experiments the response of interest also occurs spontaneously in control animals. This background may be assumed to be either independent of the induced responses or additive in a mechanistic manner. If the spontaneous and induced responses are assumed to be independent, then the probability of observing a response of either type at dose d is given by: $P^*(d) = q + (1 - q) P(d)$, where $0 < q < 1$ denotes the spontaneous background rate. Under the additivity assumption, the background response may be considered as arising from an effective background dose $w > 0$, with $P^*(d) = P(d + w)$. Although the manner in which background response is accommodated is crucial, the extent to which independence or additivity is indicated by either biological theory or experimental data is somewhat unclear at this time.

Table 15

95.0% One-Sided Lower Confidence Limits on Virtually Safe Doses for Rats in mg/kg/day in Table 14 Based on the Variance of Log-Dose (Based on NCI PCB Bioassay)

Model	1.000E-01	1.000E-02	1.000E-03	1.000E-04	1.000E-05	1.000E-06	1.000E-07	1.000E-08
Multistage	0.375313E+02	0.355084E+01	0.354086E+00	0.353907E-01	0.353890E-02	0.353888E-03	0.353888E-04	0.353888E-05
Independent Probit[a]	0.178401E+02	0.250757E-01	0.997456E-04	0.103528E-05	0.196042E-07	0.563538E-09	0.220975E-10	0.110397E-11
Independent Logit[a]	0.184791E+02	0.527052E-02	0.775747E-06	0.112539E-09	0.161647E-13	0.230974E-17	0.329112E-21	0.468183E-25
Independent Weibull[a]	0.188841E+02	0.367066E-02	0.315628E-06	0.263[illegible]E-10	0.217855E-14	0.179115E-18	0.146879E-22	0.120262E-26
Independent Gamma Multihit[a]	0.188499E+02	0.257715E-02	0.126783E-06	0.587324E-11	0.268350E-15	0.121977E-19	0.553026E-24	0.250367E-28
Additive Probit[a]	0.158943E+02	0.967604E-01	0.665965E-02	0.641349E-03	0.638936E-04	0.638696E-05	0.638671E-06	0.638665E-07
Additive Logit[a]	0.160531E+02	0.905683E-01	0.610410E-02	0.586531E-03	0.584192E-04	0.583959E-05	0.583936E-06	0.583933E-07
Additive Weibull[a]	0.162702E+02	0.844160E-01	0.558973E-02	0.536121E-03	0.533885E-04	0.533662E-05	0.533640E-06	0.533637E-07
Additive Gamma Multihit[a]	0.183917E+02	0.111336E+00	0.754068E-02	0.724782E-03	0.721912E-04	0.721625E-05	0.721596E-06	0.721589E-07

[a] In many experiments the response of interest also occurs spontaneously in control animals. This background may be assumed to be either independent of the induced responses or additive in a mechanistic manner. If the spontaneous and induced responses are assumed to be independent, then the probability of observing a response of either type at dose d is given by: $P^*(d) = q + (1 - q) P(d)$, where $0 < q < 1$ denotes the spontaneous background rate. Under the additivity assumption, the background response may be considered as arising from an effective background dose $w > 0$, with $P^*(d) = P(d + w)$. Although the manner in which background response is accommodated is crucial, the extent to which independence or additivity is indicated by either biological theory or experimental data is somewhat unclear at this time.

Table 16

95.0% One-Sided Lower Confidence Limits on Virtually Safe Doses for Rats in mg/kg/day in Table 14 Based on the Variance of the Reciprocal of Dose (Based on NCI PCB Bioassay)

Model	1.000E-01	1.000E-02	1.000E-03	1.000E-04	1.000E-05	1.000E-06	1.000E-07	1.000E-08
Multistage	0.375313E+02	0.355884E+01	0.354086E+00	0.3539076-01	0.353890E-02	0.353888E-03	0.353888E-04	0.353888E-05
Independent Probit*	0.327277E+02	0.108943E+01	0.119299E+00	0.216476E-01	0.516174E-02	0.146842E-02	0.473707E-03	0.160080E-03
Independent Logit*	0.335870E+02	0.654514E+00	0.252207E-01	0.121419E-02	0.645106E-04	0.362634E-05	0.211465E-06	0.126535E-07
Independent Weibull*	0.339697E+02	0.588631E+00	0.196698E-01	0.822457E-03	0.380248E-04	0.186185E-05	0.946204E-07	0.495586E-08
Independent Gamma Multihit*	0.337409E+02	0.529085E+00	0.157434E-01	0.588822E-03	0.244170E-04	0.107379E-05	0.490482E-07	0.230066E-08
Additive Probit*	0.314640E+02	0.111722E+01	0.100781E+00	0.997641E-02	0.996631E-03	0.996530E-04	0.996519E-05	0.996512E-06
Additive Logit*	0.316079E+02	0.110038E+01	0.980021E-01	0.977571E-02	0.976533E-03	0.976429E-04	0.976419E-05	0.976418E-06
Additive Weibull*	0.318136E+02	0.107991E+01	0.965092E-01	0.954421E-02	0.953361E-03	0.953255E-04	0.953245E-05	0.953244E-06
Additive Gamma Multihit*	0.331448E+02	0.122811E+01	0.110833E+00	0.109712E-01	0.109600E-02	0.109589E-03	0.109588E-04	0.109587E-05

*In many experiments the response of interest also occurs spontaneously in control animals. This background may be assumed to be either independent of the induced responses or additive in a mechanistic manner. If the spontaneous and induced responses are assumed to be independent, then the probability of observing a response of either type at dose d is given by: $P^*(d) = q + (1 - q)\,P(d)$, where $0 < q < 1$ denotes the spontaneous background rate. Under the additivity assumption, the background response may be considered as arising from an effective background dose $w > 0$, with $P^*(d) = P(d + w)$. Although the manner in which background response is accommodated is crucial, the extent to which independence or additivity is indicated by either biological theory or experimental data is somewhat unclear at this time.

Table 17: Excess Lifetime Cancer Risk From PCB Exposure Derived Using the Multistage Model.
Basis: NCI Total Malignancies Category

Exposure type	Exposure scenario	Estimated individual exposure* During period of exposure (mg/yr)	Estimated individual exposure* During period of exposure (mg/kg/day)	Estimated individual exposure* Lifetime average (mg/kg/day)	Duration of exposure** (years)	Maximum Likelihood Estimates of Excess Risk	95% Upper Confidence Limit on Excess Risk
Ambient Inhalation	o Reference scenarios						
	o Exposure at the average urban atmospheric level (5 ng/m^3)	3.5×10^{-2}	1.4×10^{-6}	1.4×10^{-6}	70	6.2×10^{-7}	1.0×10^{-6}
	o Exposure at the average rural atmospheric level (0.05 ng/m^3)	3.5×10^{-4}	1.4×10^{-8}	1.4×10^{-8}	70	6.2×10^{-9}	1.0×10^{-8}
	o Exposure at the PCB level of quantitation for air (10 ug/m^3)	69	2.7×10^{-3}	2.7×10^{-3}	70	1.2×10^{-3}	2.0×10^{-3}
	o Exposure at a distance of 800 m (0.5 mile) downwind of a large capacity chemicals manufacturing plant with PCBs present in the process stream at 25 mg/kg	2.9×10^{-2}	1.1×10^{-6}	1.1×10^{-6}	70	4.9×10^{-7}	8.1×10^{-7}
	o Exposure at a distance of 800 m (0.5 mile) downwind of a large capacity industrial incinerator burning wastes containing 50 mg/kg PCBs	$<2.4\times10^{-2}$	$<9.4\times10^{-7}$	$<9.4\times10^{-7}$	70	$<4.2\times10^{-7}$	$<6.9\times10^{-7}$
Ambient Ingestion	o Reference scenarios						
	o Average adult intake of PCBs via food during 1978 as reported by FDA	$<6.9\times10^{-1}$	$<2.7\times10^{-5}$	$<2.7\times10^{-5}$	70	$<1.2\times10^{-5}$	2.0×10^{-5}
	o Ingestion of fish containing 2 ppm of PCBs (i.e., the 1977 proposed FDA tolerance level for PCBs in the edible portion of fish)	4.75	1.9×10^{-4}	1.9×10^{-4}	70	8.4×10^{-5}	1.4×10^{-4}

Table 17 (Continued)

Exposure type	Exposure scenario	Estimated individual exposure* During period of exposure (mg/yr)	Estimated individual exposure* During period of exposure (mg/kg/day)	Estimated individual exposure* Lifetime average (mg/kg/day)	Duration of exposure (years) **	Maximum Likelihood Estimates of Excess Risk	95% Upper Confidence Limit on Excess Risk
Ambient Ingestion	o Ingestion of fish or water obtained from water bodies down stream of chemical plants discharging wastewater containing 100 ug/l of PCBs	2.7×10^{-4} to 130	1.1×10^{-8} to 5.1×10^{-3}	1.1×10^{-8} to 5.1×10^{-3}	70	4.9×10^{-9} to 2.3×10^{-3}	8.1×10^{-9} to 3.7×10^{-3}
	o Ingestion of groundwater drawn from wells located down gradient from a landfill receiving wastes containing 50 mg/kg PCBs	$<7.7\times10^{-6}$	$<3.0\times10^{-10}$	$<3.0\times10^{-10}$	70	$<1.3\times10^{-10}$	$<2.2\times10^{-10}$
	o Consumers of water contaminated by discharges from a typical aluminum forming plant with 66 general hydraulic systems assuming						
	- All systems contain 50 mg/kg PCBs in July 1984 and thereafter	4.5×10^{-6} to 4.5×10^{-3}	1.8×10^{-10} to 1.8×10^{-7}	1.8×10^{-10} to 1.8×10^{-7}	70	8.0×10^{-11} to 8.0×10^{-8}	1.3×10^{-10} to 1.3×10^{-7}
	- All systems contain 1,752 mg/kg PCBs from July 1984 to July 1985 and contain 50 mg/kg thereafter -July 1984 to July 85	1.1×10^{-4} to 1.1×10^{-1}	4.3×10^{-9} to 4.3×10^{-6}	2.3×10^{-10} to 2.3×10^{-7}	1	1.0×10^{-10} to 1.0×10^{-7}	1.7×10^{-10} to 1.7×10^{-7}
	-after July 85	4.5×10^{-6} to 4.5×10^{-3}	1.8×10^{-10} to 1.8×10^{-7}	2.3×10^{-10} to 2.3×10^{-7}	69	1.0×10^{-10} to 1.0×10^{-7}	1.7×10^{-10} to 1.7×10^{-7}
	o Consumers of water contaminated by dischargers from a typical petroleum refinery with 8 heat transfer systems assuming						
	- All systems contain 50 mg/kg PCBs July 1984 to July 1985 and contain 50 mg/kg thereafter	3.0×10^{-7} to 1.3×10^{-3}	1.2×10^{-11} to 5.1×10^{-8}	1.2×10^{-11} to 5.1×10^{-8}	70	5.3×10^{-12} to 2.3×10^{-8}	8.8×10^{-12} to 3.8×10^{-8}
	- All systems contain 176 mg/kg PCBs from July 1984 to July 1985 and contain 50 mg/kg thereafter -July 1984 to July 85	5.0×10^{-7} to 2.3×10^{-3}	2.0×10^{-11} to 9.0×10^{-8}	1.2×10^{-11} to 5.1×10^{-8}	1	5.3×10^{-12} to 2.3×10^{-8}	8.8×10^{-12} to 3.8×10^{-8}
	-after July 85	3.0×10^{-7} to 1.3×10^{-3}	1.2×10^{-11} to 5.1×10^{-8}	1.2×10^{-11} to 5.1×10^{-8}	69	5.3×10^{-12} to 2.3×10^{-8}	8.8×10^{-12} to 3.8×10^{-8}
	- All systems contain 441 mg/kg PCBs from July 1984 to July 1985 and contain 50 mg/kg thereafter -July 1984 to July 85	1.2×10^{-6} to 5.6×10^{-3}	4.7×10^{-11} to 2.2×10^{-7}	1.2×10^{-11} to 5.3×10^{-8}	1	5.3×10^{-12} to 2.3×10^{-8}	8.8×10^{-12} to 3.9×10^{-8}
	-after July 85	3.0×10^{-7} to 1.3×10^{-3}	1.2×10^{-11} to 5.1×10^{-8}	1.2×10^{-11} to 5.3×10^{-8}	69	5.3×10^{-12} to 2.3×10^{-8}	8.8×10^{-12} to 3.9×10^{-8}

Table 17 (Continued)

Exposure type	Exposure scenario	Estimated individual exposure[a] During period of exposure (mg/yr)	Estimated individual exposure[a] During period of exposure (mg/kg/day)	Estimated individual exposure[a] Lifetime average (mg/kg/day)	Duration of exposure (years) [b]	Maximum Likelihood Estimates of Excess Risk	95% Upper Confidence Limit on Excess Risk
Occupational inhalation	o Reference scenarios						
	o Exposure at the OSHA standard for PCBs in air (1,000 ug/m^3)	2.3×10^{-3}	9.0×10^{-2}	5.0×10^{-2}	38.5	2.2×10^{-2}	3.6×10^{-2}
	o Exposure at the level of quantitation for PCBs in air (10 ug/m^3)	23	9.0×10^{-4}	5.0×10^{-4}	38.5	2.2×10^{-4}	3.7×10^{-4}
	o Exposure at the NIOSH recommended standard for PCBs in air (1 ug/m^3)	2.3	9.0×10^{-5}	5.0×10^{-5}	38.5	2.2×10^{-5}	3.7×10^{-5}
	o Transfer and handling operation scenarios						
	o Loading/unloading a liquid assuming PCBs are present in the liquid at 25 mg/kg.	6.0×10^{-2}	2.3×10^{-6}	1.3×10^{-6}	38.5	5.7×10^{-7}	9.6×10^{-7}
	o Loading/unloading a powder assuming PCBs are present in the powder at 25 mg/kg	2.9×10^{-2}	1.1×10^{-6}	6.2×10^{-7}	38.5	2.7×10^{-7}	4.6×10^{-7}
	o Loading/unloading a powder assuming compliance with the OSHA nuisance dust standard and assuming PCBs are present in the powder at 25 mg/kg	8.8×10^{-1}	3.4×10^{-5}	1.9×10^{-5}	38.5	8.4×10^{-6}	1.4×10^{-5}
	o Process operation scenarios						
	o Exposure to background levels of fugitive emissions in enclosed chemical manufacturing plants assuming PCBs are present in the process stream at 25 mg/kg	2.6	1.1×10^{-4}	5.6×10^{-5}	38.5	2.5×10^{-5}	4.1×10^{-5}

Table 17 (Continued)

Exposure type	Exposure scenario	Estimated individual exposure[a] During period of exposure (mg/yr)	Estimated individual exposure[a] During period of exposure (mg/kg/day)	Estimated individual exposure[a] Lifetime average (mg/kg/day)	Duration of exposure (years) [b]	Maximum Likelihood Estimates of Excess Risk	95% Upper Confidence Limit on Excess Risk
Occupational inhalation	o Exposure to fugitive emissions for a worker stationed six meters downwind of leaking equipment assuming PCBs are present in the emitted chemical at 25 mg/kg	9.2×10^{-1}	3.6×10^{-5}	2.0×10^{-5}	38.5	8.8×10^{-6}	1.5×10^{-5}
	o Exposure during open-surface tank operations (e.g., degreasing tank) assuming tank liquid temperature of 75°C and assuming PCBs are present in the liquid a 25 mg/kg	6.5×10^{-2}	2.5×10^{-6}	1.4×10^{-6}	38.5	6.2×10^{-7}	1.0×10^{-6}
	o Exposure to evaporative emissions during non-spray coating operations assuming a coating temperature of 75°C and assuming PCBs are present in the coating at 25 mg/kg	4.6×10^{-1}	1.8×10^{-5}	9.9×10^{-6}	38.5	4.4×10^{-6}	7.3×10^{-6}
	o Exposure to paint mists during spray painting assuming PCBs are present in the binder at 25 mg/kg	5.5×10^{-1}	2.2×10^{-5}	1.2×10^{-5}	38.5	5.3×10^{-6}	8.8×10^{-6}
	o Exposure to paint mists during spray painting assuming PCBs are present in the solvent at 25 mg/kg	6.4×10^{-1}	2.5×10^{-5}	1.4×10^{-5}	38.5	6.2×10^{-6}	1.0×10^{-5}
	o Exposure to paint mists during spray painting assuming PCBs are present in the pigment toner at 25 mg/kg	3.2×10^{-2}	1.3×10^{-6}	6.9×10^{-7}	38.5	3.0×10^{-7}	5.1×10^{-7}
	o Exposure to evaporation emissions during liquid product formulation assuming a liquid temperature of 25°C, open formulation tanks, and PCB concentrations in the liquid at 25 mg/kg	2.5×10^{-3}	9.8×10^{-8}	5.4×10^{-8}	38.5	2.4×10^{-8}	4.0×10^{-8}

Table 17 (Continued)

Exposure type	Exposure scenario	Estimated individual exposure[a] During period of exposure (mg/yr)	Estimated individual exposure[a] During period of exposure (mg/kg/day)	Estimated individual exposure[a] Lifetime average (mg/kg/day)	Duration of exposure (years) [b]	Maximum Likelihood Estimates of Excess Risk	95% Upper Confidence Limit on Excess Risk
Occupational Inhalation	o Exposure to mists during air-blast pesticide spraying assuming PCBs are present in the active pesticide ingredient at 25 mg/kg	8.6×10^{-4}	3.3×10^{-8}	1.8×10^{-8}	38.5	8.0×10^{-9}	1.3×10^{-8}
	o Exposure to evaporative emissions during grain fumigation assuming PCBs are present in the fumigant at 25 mg/kg	$<1.7 \times 10^{-1}$	$<6.5 \times 10^{-6}$	$<3.6 \times 10^{-6}$	38.5	$<1.6 \times 10^{-6}$	$<2.6 \times 10^{-6}$
	o Exposure to oil mists during operations such as printing and metalworking assuming compliance with the OSHA standard for mineral oil mist and assuming PCBs are present in the oil at 25 mg/kg	2.9×10^{-1}	1.1×10^{-5}	6.2×10^{-6}	38.5	2.7×10^{-6}	4.6×10^{-6}
	o Exposure to evaporative emissions during foamed plastics manufacturing operations assuming PCBs are present in the blowing agent (which constitutes approximately 17 percent by weight of the foam formulation) at 25 mg/kg	1.0×10^{-1}	3.9×10^{-6}	2.2×10^{-6}	38.5	9.7×10^{-7}	1.6×10^{-6}

Table 17 (Continued)

Exposure type	Exposure scenario[b]	Estimated individual exposure[a] During period of exposure (mg/yr)	Estimated individual exposure[a] During period of exposure (mg/kg/day)	Estimated individual exposure[a] Lifetime average (mg/kg/day)	Duration of exposure (years) [c]	Maximum Likelihood Estimates of Excess Risk	95% Upper Confidence Limit on Excess Risk
Occupational inhalation	o Exposure to evaporative emissions during plastic manufacturing operations assuming PCBs are present in the plastic at 25 mg/kg	7.1×10^{-1}	2.8×10^{-5}	1.5×10^{-5}	38.5	6.6×10^{-6}	1.1×10^{-5}
	o Exposure during manufacture of asphalt roofing products	$<2.3\times10^{-1}$ to 6.4	$<9.0\times10^{-6}$ to 2.5×10^{-4}	$<5.0\times10^{-6}$ to 1.4×10^{-4}	38.5	$<2.2\times10^{-6}$ to 6.2×10^{-5}	$<1.7\times10^{-6}$ to 1.0×10^{-4}
	o Exposure to evaporative emissions during paper manufacturing assuming PCBs are present in waste-paper furnish at:						
	- 12 mg/kg	9.2×10^{-1}	3.6×10^{-5}	2.0×10^{-5}	38.5	8.8×10^{-6}	1.5×10^{-5}
	- 5 mg/kg	3.9×10^{-1}	1.5×10^{-5}	8.4×10^{-6}	38.5	3.7×10^{-6}	6.2×10^{-6}
	- 2.5 mg/kg	2.0×10^{-2}	7.8×10^{-7}	4.3×10^{-7}	38.5	1.9×10^{-7}	3.2×10^{-7}
	o Exposure to evaporative emissions during paper manufacturing assuming PCBs are present in the printed ink of wastepaper furnish at:						
	- 2 mg/kg	6.0×10^{-4}	2.4×10^{-8}	1.3×10^{-8}	38.5	5.7×10^{-9}	9.6×10^{-9}
	- 25 mg/kg	7.5×10^{-3}	2.9×10^{-7}	1.6×10^{-7}	38.5	7.1×10^{-8}	1.2×10^{-7}
	- 50 mg/kg	1.5×10^{-2}	5.9×10^{-7}	3.2×10^{-7}	38.5	1.4×10^{-7}	2.4×10^{-7}
	o Exposure during rerefining of waste oil assuming PCBs are present in the waste oil at:						
	- 2 mg/kg	7.8×10^{-3}	3.0×10^{-7}	1.7×10^{-7}	38.5	7.5×10^{-8}	1.3×10^{-7}
	- 25 mg/kg	9.7×10^{-2}	3.8×10^{-6}	2.1×10^{-6}	38.5	9.3×10^{-7}	1.5×10^{-6}
	- 50 mg/kg	1.9×10^{-1}	7.4×10^{-6}	4.1×10^{-6}	38.5	1.8×10^{-6}	3.0×10^{-6}

Table 17 (Continued)

Exposure type	Exposure scenario	Estimated individual exposure[a] During period of exposure (mg/yr)	Estimated individual exposure[a] During period of exposure (mg/kg/day)	Estimated individual exposure[a] Lifetime average (mg/kg/day)	Duration of exposure (years) [b]	Maximum Likelihood Estimates of Excess Risk	95% Upper Confidence Limit on Excess Risk
Occupational inhalation	o Exposure during garment dry cleaning operations assuming PCBs are present in the cleaning fluid at 25 mg/kg	1.6×10^{-2}	6.3×10^{-7}	3.4×10^{-7}	38.5	1.5×10^{-7}	2.5×10^{-7}
	o Sampling and maintenance operation scenarios						
	o Exposure during on-line repair of equipment leaking gas/fluid containing PCBs at 25 mg/kg	1.7×10^{-1}	6.6×10^{-6}	3.6×10^{-6}	38.5	1.6×10^{-6}	2.6×10^{-6}
	o Exposure during sampling assuming the process stream contains PCBs at 25 mg/kg:	3.6	1.4×10^{-4}	7.7×10^{-5}	38.5	3.4×10^{-5}	5.7×10^{-5}
	o Exposure while cleaning equipment containing a fluid in which PCBs are assumed to be present at 25 mg/kg	5.0×10^{-1}	2.0×10^{-5}	1.1×10^{-5}	38.5	4.9×10^{-6}	8.1×10^{-6}
	o Exposure while repairing equipment off-line assuming the equipment contains a fluid in which PCBs are present at 25 mg/kg	2.5×10^{-1}	9.8×10^{-6}	5.4×10^{-6}	38.5	2.4×10^{-6}	4.0×10^{-6}
	o Exposure during filter removal assuming PCBs are present at 25 mg/kg	1.3×10^{-1}	5.1×10^{-6}	2.8×10^{-6}	38.5	1.2×10^{-6}	2.1×10^{-6}
	o Exposure during removal of still bottoms assuming PCBs are present in the still bottoms at:						
	- 200 mg/kg	9.8×10^{-1}	3.8×10^{-5}	2.1×10^{-5}	38.5	9.3×10^{-6}	1.5×10^{-5}
	- 2500 mg/kg	13	5.1×10^{-4}	2.8×10^{-4}	38.5	1.2×10^{-4}	2.1×10^{-4}
	- 5000 mg/kg	25	9.8×10^{-4}	5.4×10^{-4}	38.5	2.4×10^{-4}	4.0×10^{-4}
	o Exposure during cleaning of spilled liquids assuming PCBs are present in the liquid at 25 mg/kg	2.0×10^{-3}	7.8×10^{-8}	4.3×10^{-8}	38.5	1.9×10^{-8}	3.2×10^{-8}

Table 17 (Continued)

Exposure type	Exposure scenario	Estimated individual exposure* During period of exposure (mg/yr)	Estimated individual exposure* During period of exposure (mg/kg/day)	Estimated individual exposure* Lifetime average (mg/kg/day)	Duration of exposure (years) **	Maximum Likelihood Estimates of Excess Risk	95% Upper Confidence Limit on Excess Risk
Occupational Inhalation	o Exposure to evaporative emissions for a worker stationed one meter downwind of a leaking hydraulic system operating at 75°C assuming						
	- All systems contain 50 mg/kg PCBs in July 1984 and thereafter	1.5×10^{-1}	5.9×10^{-6}	3.2×10^{-6}	38.5	1.4×10^{-6}	2.4×10^{-6}
	- All systems contain 1,752 mg/kg PCBs from July 1984 to July 1985 and contain 50 mg/kg thereafter						
	-July 84 to July 85	5.3	2.1×10^{-4}	6.2×10^{-6}	1	2.7×10^{-6}	4.6×10^{-6}
	-after July 85	1.5×10^{-1}	5.9×10^{-6}	6.2×10^{-6}	37.5	2.7×10^{-6}	4.6×10^{-6}
	o Exposure to evaporative emissions for a worker stationed three meters downwind of a leaking heat transfer system assuming						
	- All systems contain 50 mg/kg PCBs July 1984 to July 1985 and contain 50 mg/kg thereafter	5.8×10^{-1}	2.3×10^{-5}	1.3×10^{-5}	38.5	5.7×10^{-6}	9.6×10^{-6}
	- All systems contain 176 mg/kg PCBs from July 1984 to July 1985 and contain 50 mg/kg thereafter						
	-July 84 to July 85	2.0	7.8×10^{-5}	1.3×10^{-5}	1	5.7×10^{-6}	9.6×10^{-6}
	-after July 85	5.8×10^{-1}	2.3×10^{-5}	1.3×10^{-5}	37.5	5.7×10^{-6}	9.6×10^{-6}
	- All systems contain 441 mg/kg PCBs from July 1984 to July 1985 and contain 50 mg/kg thereafter						
	-July 84 to July 85	5.1	2.0×10^{-4}	1.5×10^{-5}	1	6.6×10^{-6}	1.1×10^{-5}
	-after July 85	5.8×10^{-1}	2.3×10^{-5}	1.5×10^{-5}	37.5	6.6×10^{-6}	1.1×10^{-5}

Table 17 (Continued)

Exposure type	Exposure scenario	Estimated individual exposure[a] During period of exposure (mg/yr)	Estimated individual exposure[a] During period of exposure (mg/kg/day)	Estimated individual exposure[a] Lifetime average (mg/kg/day)	Duration of exposure (years) **	Maximum Likelihood Estimates of Excess Risk	95% Upper Confidence Limit on Excess Risk
Occupational dermal	o Exposure to hydraulic system operators and maintenance workers assuming						
	- All systems contain 50 mg/kg PCBs in July 1984 and thereafter	17.7	6.9×10^{-4}	3.8×10^{-4}	38.5	1.7×10^{-4}	2.8×10^{-4}
	- All systems contain 1,752 mg/kg PCBs from July 1984 to July 1985 and contain 50 mg/kg thereafter						
	-July 84 to July 85	620	2.7×10^{-2}	7.1×10^{-4}	1	3.1×10^{-4}	5.2×10^{-4}
	-after July 85	17.7	6.9×10^{-4}	7.1×10^{-4}	37.5	3.1×10^{-4}	5.2×10^{-4}
	o Exposure to heat transfer system operators and maintenance workers assuming						
	- All systems contain 50 mg/kg PCBs July 1984 to July 1985 and contain 50 mg/kg thereafter	17.7	6.9×10^{-4}	3.8×10^{-4}	38.5	1.7×10^{-4}	2.8×10^{-4}
	- All systems contain 176 mg/kg PCBs from July 1984 to July 1985 and contain 50 mg/kg thereafter						
	-July 84 to July 85	62.3	2.4×10^{-3}	4.0×10^{-4}	1	1.8×10^{-4}	2.9×10^{-4}
	-after July 85	17.7	6.9×10^{-4}	4.0×10^{-4}	37.5	1.8×10^{-4}	2.9×10^{-4}
	- All systems contain 441 mg/kg PCBs from July 1984 to July 1985 and contain 50 mg/kg thereafter						
	-July 84 to July 85	156	6.1×10^{-3}	4.6×10^{-4}	1	2.90×10^{-6}	4.83×10^{-6}
	-after July 85	17.7	6.9×10^{-4}	4.6×10^{-4}	37.5	1.09×10^{-4}	1.81×10^{-4}

Table 17 (Continued)

Exposure type	Exposure scenario	Estimated individual exposure[a] During period of exposure (mg/yr)	Estimated individual exposure[a] During period of exposure (mg/kg/day)	Estimated individual exposure[a] Lifetime average (mg/kg/day)	Duration of exposure (years) [b]	Maximum Likelihood Estimates of Excess Risk	95% Upper Confidence Limit on Excess Risk
Occupational dermal							
	o Transfer and handling operations assuming PCBs are present at 25 mg/kg						
	o Loading/unloading liquid	6.0	2.4×10^{-4}	1.3×10^{-4}	38.5	5.7×10^{-5}	9.6×10^{-5}
	o Loading/unloading powder	5.8	2.4×10^{-4}	1.2×10^{-4}	38.5	5.3×10^{-5}	8.8×10^{-5}
	o Processing operations assuming PCBs are present at 25 mg/kg						
	o Closed process operations	3.0	1.2×10^{-4}	6.4×10^{-5}	38.5	2.8×10^{-5}	4.7×10^{-5}
	o Open surface tank operations	15	5.9×10^{-4}	3.2×10^{-4}	38.5	1.4×10^{-4}	2.4×10^{-4}
	o Spray painting operations	15	5.9×10^{-4}	3.2×10^{-4}	38.5	1.4×10^{-4}	2.4×10^{-4}
	o Grain fumigation operations	15	5.9×10^{-4}	3.2×10^{-4}	38.5	1.4×10^{-4}	2.4×10^{-4}
	o Air-blast pesticide spraying operations	0.41	1.6×10^{-5}	8.8×10^{-6}	38.5	3.9×10^{-6}	6.5×10^{-6}
	o Non-spray coating operations	15	5.9×10^{-4}	3.2×10^{-4}	38.5	1.4×10^{-4}	2.4×10^{-4}
	o Product formulation operations	6.0	2.4×10^{-4}	1.3×10^{-4}	38.5	5.7×10^{-5}	9.6×10^{-5}
	o Product fabrication operations						
	o Metalworking operations	15	5.9×10^{-4}	3.2×10^{-4}	38.5	1.4×10^{-4}	2.4×10^{-4}
	o Newspaper production	15	5.9×10^{-4}	3.2×10^{-4}	38.5	1.4×10^{-4}	2.4×10^{-4}
	o Plastics manufacture	15	5.9×10^{-4}	3.2×10^{-4}	38.5	1.4×10^{-4}	2.4×10^{-4}
	o Dry cleaning of garments	15	5.9×10^{-4}	3.2×10^{-4}	38.5	1.4×10^{-4}	2.4×10^{-4}
	o Sampling and maintenance operations assuming PCBs are present at 25 mg/kg in the process stream						
	o On-line repair of leaking equipment	0.25	9.8×10^{-6}	5.4×10^{-6}	38.5	2.4×10^{-6}	4.0×10^{-6}
	o Sampling process stream	15	5.9×10^{-4}	3.2×10^{-4}	38.5	1.4×10^{-4}	2.4×10^{-4}
	o Cleaning equipment	3.0	1.2×10^{-4}	6.4×10^{-5}	38.5	2.8×10^{-5}	4.7×10^{-5}
	o Off-line repair of equipment	1.5	5.9×10^{-5}	3.2×10^{-5}	38.5	1.4×10^{-5}	2.4×10^{-5}
	o Removing filters	3.0	1.2×10^{-4}	6.4×10^{-5}	38.5	2.8×10^{-5}	4.7×10^{-5}
	o Removing still bottoms assuming PCBs are present in still bottoms at:						
	- 200 mg/kg	24	9.3×10^{-4}	5.1×10^{-4}	38.5	2.3×10^{-4}	3.8×10^{-4}
	- 2500 mg/kg	300	1.1×10^{-2}	6.0×10^{-3}	38.5	2.6×10^{-3}	4.4×10^{-3}
	- 5000 mg/kg	600	2.3×10^{-2}	1.3×10^{-2}	38.5	5.7×10^{-3}	9.5×10^{-3}
	o Spill cleanup	0.75	2.9×10^{-5}	1.6×10^{-5}	38.5	7.1×10^{-6}	1.2×10^{-5}

Table 17 (Continued)

Exposure type	Exposure scenario	Estimated individual exposure[a] During period of exposure (mg/yr)	Estimated individual exposure[a] During period of exposure (mg/kg/day)	Estimated individual exposure[a] Lifetime average (mg/kg/day)	Duration of exposure (years) [b]	Maximum Likelihood Estimates of Excess Risk	95% Upper Confidence Limit on Excess Risk
Consumer inhalation	o Exposure resulting from use of space deodorants assuming PCBs are present in the product at 25 mg/kg	1.7×10^{-1}	6.7×10^{-6}	6.7×10^{-6}	70	3.0×10^{-6}	4.9×10^{-6}
	o Exposure resulting from use of moth control products assuming PCBs are present in the product at 25 mg/kg	3.2×10^{-2}	1.3×10^{-6}	1.3×10^{-6}	70	5.7×10^{-7}	9.6×10^{-7}
	o Exposures resulting from painting the interior of a house assuming CB are present in the pigment at 25 mg/kg	1.7×10^{-4}	6.7×10^{-9}	1.3×10^{-9}	14	5.7×10^{-10}	9.6×10^{-10}
	o Exposures resulting from painting the interior of a house assuming PCBs are present in a resin intermediate at 25 mg/kg	7.7×10^{-4}	3.0×10^{-8}	6.0×10^{-9}	14	2.7×10^{-9}	4.4×10^{-9}
	o Exposures resulting from inhabiting a home with a newly painted interior assuming PCBs are present in the paint pigment at 25 mg/kg	2.6×10^{-2}	1.0×10^{-6}	4.1×10^{-7}	28	1.8×10^{-7}	3.0×10^{-7}
	o Exposures resulting from inhabiting a home with a newly painted interior assuming PCBs are present in a resin intermediate at 25 mg/kg	1.2×10^{-1}	4.7×10^{-6}	1.9×10^{-6}	28	8.4×10^{-7}	1.4×10^{-6}
	o Exposures resulting from use of spray paint assuming PCBs are present in a resin or solvent at 25 mg/kg	2.9×10^{-2}	1.1×10^{-6}	8.9×10^{-7}	55	3.9×10^{-7}	6.5×10^{-7}
	o Exposures resulting from use of spray paints assuming PCBs are present in the pigment at 25 mg/kg	2.2×10^{-3}	8.6×10^{-8}	6.8×10^{-8}	55	3.0×10^{-8}	5.0×10^{-8}

Table 17 (Continued)

Exposure type	Exposure scenario	Estimated individual exposure[a] During period of exposure (mg/yr)	Estimated individual exposure[a] During period of exposure (mg/kg/day)	Estimated individual exposure[a] Lifetime average (mg/kg/day)	Duration of exposure (years) **	Maximum Likelihood Estimates of Excess Risk	95% Upper Confidence Limits on Excess Risk
Consumer dermal	o Exposures resulting from painting the interior of a house assuming PCBs are present in a resin intermediate at 25 mg/kg	3.3×10^{-2}	1.3×10^{-6}	2.6×10^{-7}	14	1.1×10^{-7}	1.9×10^{-7}
	o Exposures resulting from use of spot removers assuming PCBs are present in the product at 25 mg/kg	1.0×10^{-1}	3.9×10^{-6}	3.1×10^{-6}	55	1.4×10^{-6}	2.3×10^{-6}
	o Exposures resulting from use of general household cleaners assuming PCBs are present in a detergent constituent (that accounts for 25 percent by weight of the product) at 25 mg/kg	2.0×10^{-1}	7.8×10^{-6}	6.2×10^{-6}	55	2.7×10^{-6}	4.6×10^{-6}
	o Exposures resulting from use of paint removers assuming PCBs are present in the product at 25 mg/kg	2.1×10^{-2}	8.2×10^{-7}	6.4×10^{-7}	55	2.8×10^{-7}	4.7×10^{-7}
	o Exposures resulting from use of dyed clothing and sheets assuming PCBs are present in the dye at 25 mg/kg	1.3×10^{-2}	5.1×10^{-7}	5.1×10^{-7}	70	2.1×10^{-7}	3.8×10^{-7}
Consumer ingestion	o Exposure to foods contaminated with pesticides that contain PCBs at 25 mg/kg	6.4×10^{-4}	2.5×10^{-8}	2.5×10^{-8}	70	1.1×10^{-8}	1.8×10^{-8}

References:

[a] Estimated individual exposures in terms of mg/yr were taken from Versar (1983). Average estimated exposures in terms of mg/kg/day were calculated for the period exposure and also for the lifetime of the individual (assumed to be 70 years) for a 70 kg individual averaged over 365 days/year. The absorbed dose equals the exposure because 100 percent absorption is assumed for all exposure routes.

** Duration of exposure refers to the number of years during a lifetime that the PCB exposure could be expected to occur. All occupational exposures are assumed to have a duration of 38.5 years. This is the average work life expectancy for males in the United States (personal communication between G. Schweer (Versar Inc.) and S. Smith (U.S. Department of Labor, Bureau of Labor Statistics), on April 7, 1983).

Exposure type	Exposure scenario	Estimated individual exposure* During period of exposure (mg/yr)	Estimated individual exposure* During period of exposure (mg/kg/day)	Estimated individual exposure* Lifetime average (mg/kg/day)	Duration of exposure (years) **	Maximum Likelihood Estimates of Excess Risk	95% Upper Confidence Limit on Excess Risk
Consumer inhalation	o Exposure resulting from use of pesticide sprays assuming PCBs are present in the active ingredient at 25 mg/kg	2.2×10^{-2}	8.6×10^{-7}	6.8×10^{-7}	55	3.0×10^{-7}	5.0×10^{-7}
	o Exposure resulting from use of pesticide sprays assuming PCBs are present in the inert ingredients at 25 mg/kg	1.1×10^{-1}	4.3×10^{-6}	3.4×10^{-6}	55	1.5×10^{-6}	2.5×10^{-6}
	o Exposures resulting from use of spray cleaning/disinfectant products assuming that PCBs are present in a constituent (that accounts for 50 percent of the weight of the product) at 25 mg/kg	2.3×10^{-1}	9.0×10^{-6}	7.1×10^{-6}	55	3.1×10^{-6}	5.2×10^{-6}
	o Exposures resulting from inhabiting a new home containing plastic building materials which are assumed to contain PCBs at 25 mg/kg	8.1	3.2×10^{-4}	2.7×10^{-5}	6	1.2×10^{-5}	2.0×10^{-5}
Consumer dermal	o Exposures resulting from use of deodorant soaps assuming PCBs are present in the surfactant at 25 mg/kg	2.1×10^{-2} to 19	8.2×10^{-7} to 7.4×10^{-4}	8.2×10^{-7} to 7.4×10^{-4}	70	3.6×10^{-7} to 1.3×10^{-4}	6.0×10^{-7} to 5.4×10^{-4}
	o Exposures resulting from use of skin lotions assuming PCBs are present in the surfactant at 25 mg/kg	6.4	2.5×10^{-4}	2.5×10^{-4}	70	1.1×10^{-4}	1.8×10^{-4}
	o Exposures resulting from handling of printed matter assuming PCBs are present in the ink pigment at 25 mg/kg	5.9×10^{-2}	2.3×10^{-6}	2.0×10^{-6}	60	8.8×10^{-7}	1.5×10^{-6}
	o Exposures resulting from painting the interior of house assuming PCBs are present in the pigment at 25 mg/kg	7.0×10^{-3}	2.7×10^{-7}	5.5×10^{-8}	14	2.4×10^{-8}	4.0×10^{-8}