THE PCB SITUATION IN GERMANY

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We have had a PCB problem in Germany since 1969. At the Swedish-German Symposium at Baden-Baden, Dr. Jensen, who published about this problem first in 1966, gave a report entitled "PCB-the DDT Problem of Industrial Countries."

In September 1970 there was a PCB conference at Stockholm. One of the reports given there mentioned that a fatal case of biphenyl intoxication occurred in Finland. The person was a 31 year old man who had worked in a plant which used biphenyl not chlorinated for impregnation. The exposure level was about 100 mg/m^3 - 10 times the MAC. He had been working in this industry for 11 years and suddenly developed liver insufficiency which appeared biologically as necroses, atrophy, and cirrhosis. A general atrophy of the brain cortex was also found. Medical examinations of the other 120 persons working in this plant revealed three additional cases with liver damage (biopsy) and two cases with pathological EEG and impaired peripheral nerve function.

In July 1970 Acker (University of Munster) was able to find PCB in human milk at a level of 0.103 ppm in milk which means 3.5 ppm in milk fat. In human fat tissue he found an average of 5.7 ppm.

A recent German investigation on rats (Benthe, University of Hamburg) reports about absorption and distribution of PCB after one inhalatory application of Pydroul A 200, a mixture of low chlorinated biphenyls. Absorption as well as distribution were studied by measurements of PCB concentration in different organs.

By this route of inhalatory application a very good absorption is shown. Depending on duration of PCB exposition, a fast increase of PCB concentration in liver was observed. The concentration after 15 minutes was more than 50 percent of the maximum concentration attained after two hours (70 ug/g tissue). Concentration in fat after 30 minutes of exposition is 14 ug/g tissue (about 27 percent of liver concentration) whereas in brain tissue only 9 ug/g tissue (17 percent of liver concentration) is found at this time.

Dependent on the amount of time which passed after the end of aerosol application, a fast decrease of PCB concentration in liver during 24 hours was measured and was accompanied by an increase in brain and fat. In the course of two days brain and liver concentration fell to a minimum value, whereas in fat a maximum value was attained (260 ug/g tissue) and remained at a constant level in the subsequent period. There was no toxic fatty degeneration of liver under these experimental conditions.

Seven weeks after exposition to low chlorinated PCB (Tetrachlor), the compounds were found in the fat of rats. After three weeks in the fat 70 percent of maximum value was detected, and it has to be taken into account that in a stress situation PCB moves from fat tissue into the liver. In addition to the inhalatory application other studies were made. The results are the following:

A stimulation of the microsomal enzyme system which is responsible for the degradation of drugs (phenobarbitals) was found. After intraperitoneal application of PCB there was found by measurement of O-demethylation an extreme high stimulation of enzyme activity for a long time. Yet after four weeks the values were

twice the value of the control animals. Because the microsomal enzymes are responsible to hormone system, an influence to the hormone systems of kidney-cortex and sexual hormone system can be expected.

After exposition for a long time (3 inhalations for 3 hours), a toxic effect on liver function was found: transaminase activity (SGOT, SGPT) had increased to an average of 50 percent. Parallel to this increase of transaminase activity, the liver fat increased from a normal 3.9 percent to 8.7 percent. We consider this a toxic effect.

After a subclinical dose of tetrachlorinated hydrocarbon (a dose which does not cause a measurable effect of liver damage) was given, the application of PCB caused a massive effect of liver intoxication; this means that in the case of a subclinical liver damage a PCB absorption in any way may be dangerous by causing liver dystrophy.

All these results will be published next time in Archives of Toxicology.