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Effect of Difference in Diet Composition on the Toxicity of Polychlorinated Biphenyls

Noriko Yagi,[1] Kazutaka Kamohara,[2] and Yoshinori Itokawa[3]

[1]Department of Nutrition, Koshien University, Hyogo, [2]Kyoto City Institute of Public Health, Kyoto, and [3]Department of Hygiene, Faculty of Medicine, Kyoto University, Kyoto, Japan

Itokawa et al. (1976) reported that the weight of the liver of PCB poisoned rats increased significantly, compared with the liver weight of non-PCB poisoned rats, that the liver of rats given PCB and fed a commercial diet showed prominent dark-brawn pigmentation which was identified histochemically as a ceroid and that the liver of rats given PCB and fed a lab. made diet became pale yellow due to an accumulation of lipids.

However, how differences in diet composition affected PCB toxicity was not determined. Therefore, this paper examines the effects of differences in diet composition on the toxicity of PCB as to LD_{50} of mice.

MATERIALS AND METHODS

Male mice of DDY strain, weighing about 20g, were separated into two groups of 80 animals each; group 1 was fed a commercial diet and group 2 a lab. made diet. The composition of the diets are given in Table 1. After seven days of feeding, eight kinds of PCB (Kanechlor-500) concentrations of 0.89, 1.28, 1.55, 1.78, 2.19, 2.68, 3.08 and 3.55 g/kg mouse body weight were orally administered to ten mice each by a stomach sonde, and thereafter one group of mice was fed commercial diet and another group of mice was fed lab. made diet. When the mice died after administration of PCB, the dead mice and their livers were weighed. On the ninth day after the administration of PCB all the surviving mice were killed and their livers were weighed. The value of LD_{50} was obtained by the Litchfield and Wilcoxon method (Litchfield and Wilcoxon, 1949).

To compare the mice administered PCB orally to those given PCB in their diets, commercial and lab. made diets containing PCB were given to two groups of 25 mice each; group 1 was fed a commercial diet containing 2500 ppm (mg/kg) PCB and group 2 a lab. made diet containing 2500 ppm PCB. The number of days it took for the mice to die after being fed diets containing PCB and the number of

Table 1. Composition of diets.

Component	Commercial diet	Lab. made diet
	(%)	(%)
Protein	26.0	15.0 (Casein)
Fat	5.9	10.0 (Olive oil)
Carbohydrate	56.5	38.8 (Sucrose)
		30.0 (Starch)
Cellulose	4.8	2.0
Salt mixture	6.6[a]	4.0[b]
Vitamins	c	d

Fatty acid composition of diets (%)

Fatty acid	Palmitate	Stearate	Oleate	Linoleate
Commercial diet	16.08	1.78	23.43	53.20
Lab. made diet	10.93	2.26	81.68	4.29

a; Ca:1760mg, P:1040mg, Mg:300mg, Na:240mg, K:420mg, Fe:53mg, (per 100g of diet)

b; Ca:511mg, P:208mg, Mg:59mg, Na:127mg, K:211mg, Fe:19mg (per 100g of diet)

c; thiamin:900ug, riboflavin:800ug, pyridoxine:800ug, nicotinic acid:4mg pantothenic acid:2.5mg, ascorbic aicd:50mg, vitamin A:2000 I.U., vitamin D:400 I.U. (per 100g of diet)

d; thiamin:500μg, riboflavin:750μg, pyridoxine:500μg, nicotinic acid:5mg, pantothenic acid:2.5mg, ascorbic acid:18.8mg, vitamin A:6250 I.U., vitamin D:500 I.U. (per 100g of diet)

dead mice were examined.

RESULTS AND DISCUSSION

Figure 1 shows the relationship between the number of days it took for death to occur after administration of 3.05 g/kg body weight PCB and the liver weight of the dead mice. The liver weight of control groups (non PCB fed mice) was unchanged, while that of PCB administered groups increased noticeably for four days, and thereafter was almost unchanged.

Regarding the number of days before death of mice given PCB, all of the dead mice that had been fed the commercial diet died between the first and 4th days, and 90 percent of the dead mice that had been fed the lab. made diet died between the first and 4th days.

Figure 2 shows the effect of PCB concentration on the ratio of liver weight to body weight of mice on the 9th day after the administration of PCB. The ratio of liver weight to body weight increased with an increase in PCB concentration, and it was not affected by differences in diet composition.

Figure 3 shows the relationship between dose of PCB and percentage of dead mice. It is seen that the PCB concentration of LD_{50} was 2.1 g/kg body weight for commercial diet groups and 2.6 g/kg body weight for lab. made diet groups. That is, the PCB concentration of LD_{50} was lower for the commercial diet than for the lab. made diet. Thus, PCB toxicity was stronger in mice fed the commercial diet.

The liver of mice administered PCB and fed commercial diet had prominent dark-brown pigmentation, while those of mice administered PCB and fed the lab. made diet were pale yellow.

Figure 4 shows the relationship between the number of days it took for death to occur after mice were fed commercial and lab. made diets containing 2500 ppm PCB and the number of dead mice. The mice began to die on the 2nd day in the case of the commercial diet and on the 4th day in the case of lab. made diet. The mice fed the commercial diet died earlier than those fed the lab. made diet. Significant difference at $p < 0.005$ level was observed by analysis of Chi test (Remington and Schork, 1970). Thus, when the mice were fed commercial and lab. made diets containing PCB, the toxicity of PCB was stronger in the commercial diet, as in the case where PCB was administered orally to mice.

In Figure 2, the liver weight increased with an increase

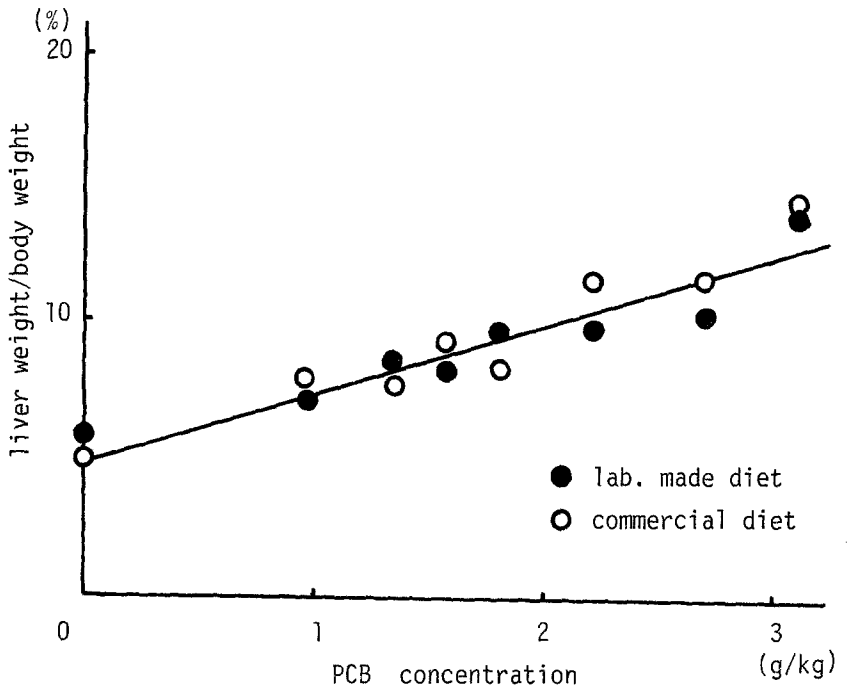

Fig. 1 RATIO OF LIVER WEIGHT TO BODY WEIGHT

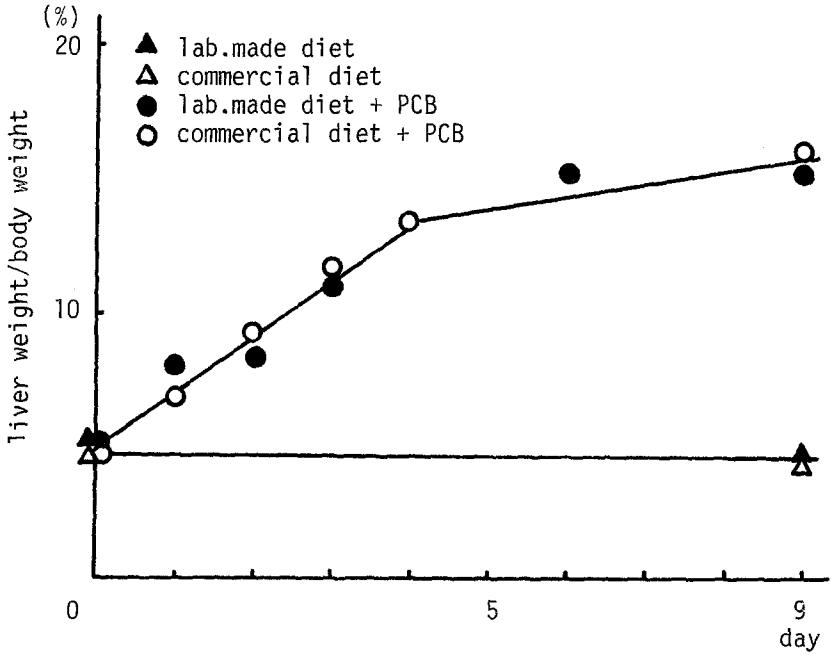

Fig. 2 RATIO OF LIVER WEIGHT TO BODY WEIGHT

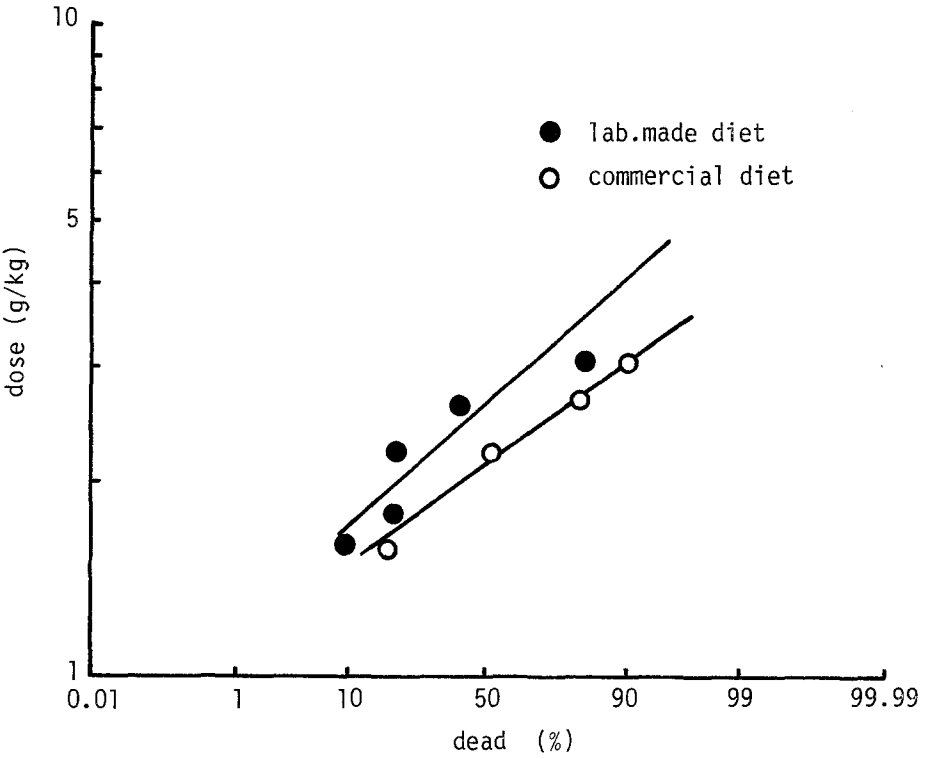

Fig. 3 DOSE EFFECT LINE

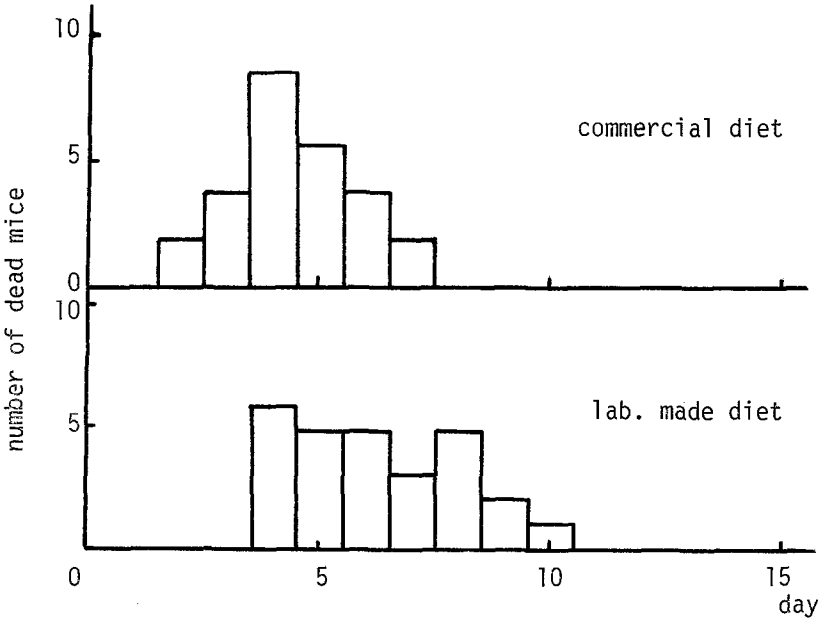

Fig.4 RELATION BETWEEN NUMBER OF DEAD MICE AND DAYS

in PCB concentration and this seems to mean that there is some relationship between increases in liver weight and PCB toxicity.

According to the results reported for rats (Itokawa et al., 1976), the livers of rats administered PCB and fed the commercial diet showed a dark-brown pigmentation which was identified as a ceroid which is produced by peroxidized lipid or lipoprotein (Endicott, 1944), while those of rats administered PCB and fed the lab. made diet became pale yellow due to an accumulation of lipids.

On the other hand, as was shown in Fig. 3, when the mice that were administered PCB were fed the same diets as the rats were fed, LD_{50} was smaller for mice fed the commercial diet than for mice fed the lab. made diet. Therefore, from these results, it is assumed that production of peroxidized lipid or lipoprotein is related to an increase in PCB toxicity. Further studies on these systems may elucidate the mechanism.

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