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Fish toxicology

**ACCUMULATION DYNAMICS OF PCBs IN SELECTED ORGANS
AND TISSUES OF CARP (*CYPRINUS CARPIO* L.)**

**DYNAMIKA KUMULACJI PCB W WYBRANYCH NARZĄDACH
I TKANKACH KARPIA (*CYPRINUS CARPIO* L.)**

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The study was aimed at the PCB (Clophen A50) accumulation dynamics in the gill filaments, muscle tissue, perin-testinal adipose tissue, alimentary tract and the liver of cultured carp, *Cyprinus carpio* L. when taken, only, either from contaminated water or contaminated diet.

The highest accumulation dynamics was observed in first 5–10 days of intoxication followed by a visible decrease resulted partly from increase in a growth rate of the tested organs and tissues as well as from the smaller daily intake per weight unit of the tested fish.

INTRODUCTION

Presence of polychlorinated biphenyls in natural environment, their durability and toxicity pose a serious threat to biosphere. Among other synthetic chemical compounds PCBs dominate in marine environment, many freshwater basins and rivers. Up to 1980 an estimated global production ranged from 1 200 000 (Tanabe 1988) to 2 000 000 tonnes (Anonymous 1979). Since 1980 the annual production has been estimated at ~50 000 tonnes. As early as the 1930s the toxic effects of these compounds for humans were determined. In Japan, in 1968, poisoning symptoms in 1291 persons due to consumption of rice oil contaminated with a PCB compound (Kanechlor 400) were recorded (Kuratsune et al. 1976; Popov 1983).

For many years, due to exceptional durability of PCB compounds, the problem of PCBs presence and an ecological threat it posed were out of sight. An interest in PCBs presence in the environment dates from 1966, when polychlorinated biphenyls were identified in bodies of fishes (Jensen 1966) and then in tissues of humans, animals, and plants, in air samples, water, soil, and in feed and food products (Wildish 1970; Holden 1975; Marchand 1977; Smoczyński et al. 1984).

A long lasting production and broad, common usage of PCBs in various branches of economy contributed to penetration of these compounds into environment (Waish 1972; Tutsakava 1973; Marchand 1977). According to Tanaby (1988) 60% of, to-date, global PCBs production entered seas and oceans, 30% of which has been harboured within in-shore bottom sediments.

While in water basins, lipophilic by nature and of poor water solubility ($0.04\text{--}0.20\text{ mg}\cdot\text{dm}^{-3}$) PCB compounds enter easily the trophic chain and show high bioaccumulation coefficient (Tatsakava 1973; Harvey et.al. 1974; Dexter and Field 1989).

PCBs penetrate fish body mainly through the gills (O'Connor 1991). Large surface, numerous blood vessels and continuous water flow through the gill cavity contribute to such penetration. Feeding is also one of more important ways of PCBs uptake by fishes. In general, PCBs accumulation coefficients in fishes are very high (Skea et al. 1979; Roots 1984). Besides particular organs and tissues of fishes have different bioaccumulation coefficients (Gooch and Hamdy 1983; Solbakken et al. 1984).

The objective of this study was to determine accumulation dynamics of total PCBs in selected organs and tissues of cultured carp when acquired from polluted water or diet.

MATERIAL AND METHODS

The present study was conducted on cultured carp (*Cyprinus carpio* L.), 210 to 230 g of weight, free of PCB contamination. The experiment was carried out under laboratory conditions in glass aquaria— 100 dm^3 in volume—in tap water with forced aeration. There were 20 fish in each aquarium. Fish, upon arrival to the laboratory, had been adapted, for 10 days, to a new environment. Carp were fed granulated feed containing 40% of total protein and 6% of lipids. Clophen A50 standard was used in assessment of the accumulation dynamics.

Three experimental treatment were designed for the 40-day project:

1. Fish were kept in water with Clophen A50 at concentration of $40\text{ }\mu\text{g}\cdot\text{dm}^{-3}$ and fed PCB-free diet.
2. Fish were kept in clean PCB-free water and fed feed containing of $100\text{ }\mu\text{g}\cdot\text{kg}^{-1}$ Clophen A50.
3. Control; fish were kept in clean, PCB-free water and fed feed containing no PCB compounds.

In all the experimental treatments, in order to dechlorinate the water and adjust the water temperature, the aquaria were filled up with tap water and aerated for 24 hours before fish introduction. Water temperature throughout the experiment ranged from 18 to 24°C , oxygen content oscillated between 7.6 and $10.2\text{ mg}\cdot\text{dm}^{-3}$ and pH was 6.4–7.4. Fish were transferred to new aquaria with clean water every day. In first experimental treatment, prior

to fish transfer, 4 ml of Clophen A50 acetone solution, in concentration of $1.0 \text{ mg}\cdot\text{ml}^{-1}$, was added to the aquaria water and mixed intensively.

The assumed PCBs concentration in feed ($100 \text{ mg}\cdot\text{g}^{-1}$) was achieved by submerging 500 g of feed in 1.0 dm^3 acetone solution of Clophen A50 (50.5 mg of Clophen A50) and then evaporation of acetone by a rotary vacuum evaporator. Every day, when transferred to aquaria with clean water, carp were fed 3 times, in 2-hour intervals, with contaminated feed—each fish receiving daily 2 g of contaminated diet—followed by 3 servings of PCB-free diet until satiation. A daily intake of PCBs with feed was equal to the PCBs content per one fish in polluted water (5 dm^3). To facilitate accurate weighing, carp were divided into groups of five, at random, and marked.

The PCBs concentration in tested fish was estimated after 1, 2, 3, 5, 10, 20, 30, and 40 days of intoxication. Each time 5 fish were tested, and samples of muscle tissue (filets), liver, perintestinal adipose tissue, alimentary tract and gill filaments were collected and weighed. Each sample type was homogenised separately. Subsamples of 10 g of blended muscle tissue, 3 g of liver and alimentary tract, 2 g of gill filaments, and 1 g of perintestinal adipose tissue subsamples were collected for analyses. Analyses were carried out in 5 repetitions according to Jensen et al. (1983) method. Analytical procedure included extraction of lipids together with associated compounds first with acetone-hexane (2.5 : 1) mixture, then with hexane-ethyl ether (9 : 1) and purification of extracts with 7% SO_3 in H_2SO_4 and 5% KOH in 96% $\text{C}_2\text{H}_5\text{OH}$. Qualitative and quantitative analyses of PCBs were carried out by a "Pye-Unicam" gas chromatograph equipped with ^{63}Ni electron capture detector (ECD) under following conditions:

- glass column was 3.75 m long and of 1.8 mm in diameter,
- column filler: 2.8% QF and 0.6% SF 96 on Chromosorbent W mesh: 100–200,
- temperature of column 170–210°C programmed on $1^\circ\text{C}/\text{min}$; feeder temp.: 200°C ; detector temp.: 300°C ,
- carrying gas: 10% methane in argon; flow rate: 30 ml/min.

Qualitative PCBs readings were done by comparing retention times for peaks in the samples tested against Clophen A50 standard, while quantity of the PCB was calculated from comparison of sums of the peak heights.

Conclusions on changes in PCBs contents were based on statistics (Czermiński et al. 1974). Variances and equality hypothesis verification with the Hartley test, with confidence limit $\alpha = 0.01$ were calculated. To assess correlation between the PCB content within the tested organs and tissues and exposure time the correlation coefficients and equations of regression curves were counted.

RESULTS

No visible symptoms of poisoning in tested carp subjected to Clophen A50 intoxication were observed throughout the experiment. However weight of the tested organs and tissues were higher when compared with the same organs and tissues of control fishes. It was particularly noticeable for perintestinal adipose tissue and liver (Table 1). Moreover, the liver and muscle tissue had higher lipid contents (Table 2).

After 24-hour exposure of fish to a dose of $40 \mu\text{g}\cdot\text{dm}^{-3}$ of Clophen A50 in water, the highest accumulation of PCBs was noted in the gill filaments and the lowest one in the alimentary tract (Fig. 1). When converted to lipids of the tested organs and tissues, the highest concentration was found in the gill filaments and the smallest in perintestinal adipose tissue (Table 3). With the prolonging exposure to PCBs in the water, the highest growth dynamic in PCBs content, in wet weight, within first 5–10 days was noted for the gill filaments. In other organs and tissues differences were not significant (Fig. 1). After 40 days of exposure, the highest PCBs accumulation was recorded in perintestinal adipose tissue and the smallest one—in the alimentary tract. Again when converted to lipids the highest increment in PCBs concentration growing in direct proportion to the exposure time was for the gill filaments and the lowest one—in perintestinal adipose tissue. There was a drop of PCBs in lipids of muscle tissue after 30 days of exposure.

Daily intake of $200 \mu\text{g}$ of Clophen A50 in feed per one carp resulted in its highest accumulation, per wet weight, within the perintestinal adipose tissue with the lowest accumulation noted for the muscle tissue (Table 3). Again, lipids of gill filaments had the highest and perintestinal adipose tissue the lowest concentration of PCB compounds (Table 3). Accumulation dynamics of the PCBs in wet weight of the perintestinal adipose tissue, alimentary tract, liver, and the muscle tissue was observed during the first 10 days of fish exposure to the PCB compounds (Fig. 1). Such differences for gill filaments were less pronounced. After 40 days of intoxication the highest accumulation of Clophen A50 was registered for perintestinal adipose tissue and the lowest one for gill the filaments (Table 3). However, when converted to lipids of the tested organs and tissues the opposite effect was noted. The highest concentration of PCBs in the muscle tissue and liver, followed by a drop in PCBs concentration, was reached on day 10 and on day 20 of intoxication, respectively.

Table 1

Average increase in the body weight of whole fishes, tested organs, and tissues (%) during intoxication

Subject of analysis	Samples	Initial weight (g)	Intoxication time (days)							
			1	2	3	5	10	20	30	40
			weight increment (%)							
Gill filaments	control surveys	3.11 ±0.11	0.09	0.09	0.14	0.44	0.86	1.43	3.48	8.44
	absorption of PCB from water	3.21 ±0.17	0.28	0.34	0.42	0.56	1.57	2.77	6.42	12.83
	intake of PCB with feed	3.27 ±0.21	0.18	0.21	0.35	0.42	1.39	2.18	6.11	11.27
Muscle tissue	control surveys	84.17 ±3.74	0.12	0.14	0.31	0.50	2.08	8.72	14.43	19.09
	absorption of PCB from water	83.33 ±3.41	0.00	0.00	0.09	0.33	3.34	8.17	14.89	22.17
	intake of PCB with feed	84.79 ±4.11	0.00	0.00	0.10	0.21	3.72	8.84	15.14	23.49
Perintestinal adipose tissue	control surveys	1.74 ±0.15	1.11	1.29	1.64	1.72	1.83	2.43	6.15	10.43
	absorption of PCB from water	1.63 ±0.27	1.34	1.73	2.19	2.56	6.85	10.44	23.51	38.56
	intake of PCB with feed	1.84 ±0.32	1.41	1.81	2.76	3.11	10.03	21.16	38.09	59.41
Alimentary tract	control surveys	3.71 ±0.27	1.44	1.66	1.90	2.00	2.15	2.35	4.58	7.11
	absorption of PCB from water	3.68 ±0.17	1.17	1.70	1.76	1.93	2.07	2.98	6.15	10.43
	intake of PCB with feed	3.74 ±0.29	1.29	2.11	2.14	2.44	2.93	3.92	8.09	14.08
Liver	control surveys	2.12 ±0.18	0.00	0.00	0.00	1.57	1.95	2.43	3.48	6.98
	absorption of PCB from water	2.26 ±0.21	1.34	1.78	1.89	3.19	7.32	13.42	24.82	33.27
	intake of PCB with feed	2.32 ±0.33	1.49	2.09	2.63	4.81	15.11	32.19	41.14	58.62
Whole fish	control surveys	221.50 ±9.21	0.32	0.53	1.27	2.84	4.21	7.12	12.92	16.04
	absorption of PCB from water	219.32 ±8.36	-0.41	-1.11	-0.23	1.43	3.82	7.52	12.78	17.83
	intake of PCB with feed	223.27 ±6.33	-0.44	-0.93	-0.37	1.28	4.11	7.94	13.28	18.14

Table 2

Changes in lipids content (%) in carp organs and tissues during 40 days of intoxication

Organs and tissues	Samples	Intoxication time (days)								
		0	1	2	3	5	10	20	30	40
		lipids content (%)								
Gill filaments	control surveys	1.08	1.08	1.08	1.08	1.09	1.12	1.12	1.14	1.14
	absorption of PCB from water	1.08	1.09	1.09	1.09	1.11	1.17	1.24	1.28	1.31
	intake of PCB with feed	1.08	1.08	1.08	1.08	1.08	1.08	1.09	1.11	1.11
Muscle tissue	control surveys	1.51	1.51	1.53	1.57	1.61	1.93	2.31	3.29	3.96
	absorption of PCB from water	1.51	1.54	1.56	1.59	1.71	2.18	3.09	4.41	5.18
	intake of PCB with feed	1.51	1.52	1.51	1.51	1.52	1.60	3.16	4.50	5.15
Perintestinal adipose tissue	control surveys	84.03	83.99	84.06	84.08	84.71	85.01	85.17	85.42	85.61
	absorption of PCB from water	84.03	83.93	83.82	83.97	84.52	84.95	85.61	86.07	86.21
	intake of PCB with feed	84.03	83.91	83.89	84.08	84.11	84.86	87.19	86.92	87.29
Alimentary tract	control surveys	5.11	5.08	5.06	5.11	5.14	5.23	5.29	5.38	5.56
	absorption of PCB from water	5.11	5.09	5.08	5.09	5.11	5.31	5.27	5.41	5.48
	intake of PCB with feed	5.11	5.22	5.18	5.01	5.09	5.15	5.23	5.30	5.30
Liver	control surveys	4.38	4.36	4.59	4.41	4.44	4.42	4.52	4.63	4.94
	absorption of PCB from water	4.38	4.38	4.47	4.58	4.92	5.41	6.33	7.47	10.08
	intake of PCB with feed	4.38	4.37	4.59	4.69	4.97	6.63	7.23	8.84	11.69

Table 3

Amount of PCBs in lipids of carp organs and tissues during 40 days of intoxication

	Samples	Intoxication time (days)							
		1	2	3	5	10	20	30	40
		PCB content ($\mu\text{g}/\text{kg}$)							
Absorption of PCB from water	Gill filaments	91807.3	149990.8	207165.1	280900.9	370692.3	442064.5	475664.1	489740.5
	Muscle tissue	33305.2	55166.7	77094.3	105321.6	131839.4	145835.0	129449.0	128608.1
	Perintestinal adipose tissue	726.6	1233.2	1730.1	2345.5	3781.2	5950.6	8067.3	10043.0
	Alimentary tract	3546.2	5478.3	7353.6	10904.1	18905.8	28992.4	36055.5	42715.3
	Liver	8347.8	12248.4	16683.4	23859.8	36687.9	49690.4	50704.1	51561.5
Intake of PCB with feed	Gill filaments	66722.0	75564.8	91675.9	110453.7	139213.0	177412.8	200576.6	255675.9
	Muscle tissue	18026.3	28801.3	41655.6	69421.1	115343.8	88335.4	77897.8	81417.5
	Perintestinal adipose tissue	1105.4	1973.4	3374.3	5793.8	8743.0	9533.8	10603.7	11500.7
	Alimentary tract	12865.9	18666.0	26686.6	35473.5	53398.1	68831.7	81435.8	96560.4
	Liver	17972.5	27843.1	37096.6	63066.8	60392.2	68820.2	68263.6	58396.1

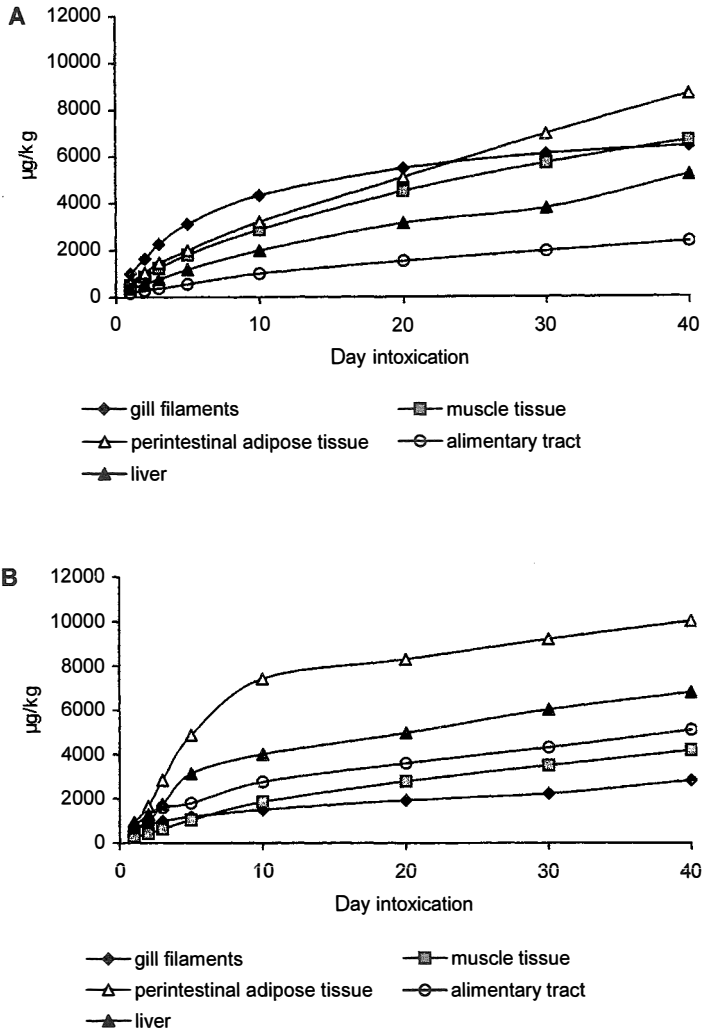


Fig. 1. Dynamics of Clophen A50 accumulation within gill filaments, muscle tissue, perintestinal adipose tissue, alimentary tract, and liver during 40-day intoxication; A, absorption of PCB from water; B, intake of PCB with feed

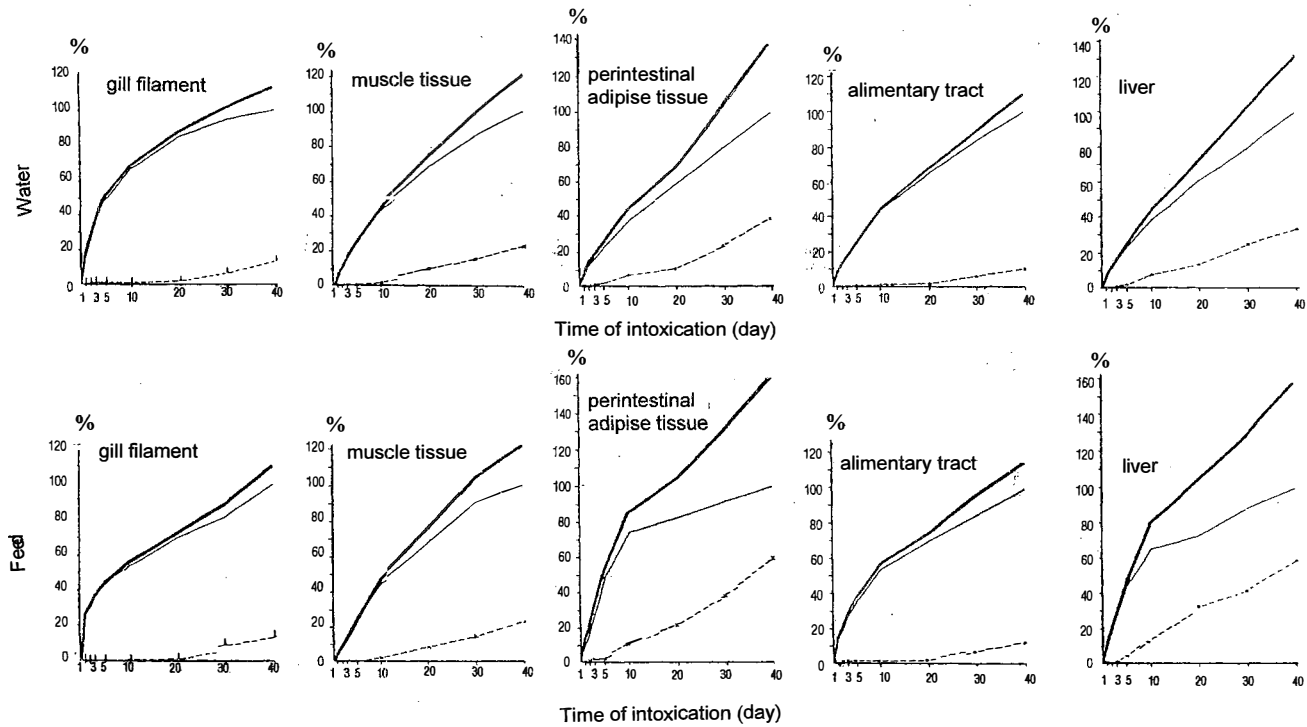


Fig. 2. Comparison (%) of PCBs accumulation dynamics (—) and PCBs content (—) per weight of whole organs and tissues of the carp (---) during 40-day of fish intoxication

DISCUSSION

Results of the present study demonstrated that polychlorinated biphenyls, when present in water and fish diet, penetrate fast into carp body and accumulate in individual organs and tissues. This is consistent with the results of O'Connor (1991) who confirmed presence of PCB in all fish organs and tissues 6 hours after its administration to fish with diet. In experimental carp PCBs were present in tested organs and tissues as early as in 24 hours after the exposure. Also differences were observed in accumulation dynamics in relation to intake routes of toxic compounds. The highest accumulation dynamics in wet liver weight, with PCBs delivered with feed, was noted within first 5 to 10 days of intoxication, which was not the case for the PCBs incorporated from water. It can be explained by faster increase in liver weight in carp when intoxicated via oral route than if adsorbing Clophen A50 from water. In liver lipids the highest increase in PCBs concentration was noted after first 20–30 days of exposure followed by a visible drop in concentration. Such drop in accumulation dynamics of the tested compounds within lipids of both, muscle tissue and liver, were probably due to essential weight increase of these organs as well as increase of lipids content along with the time of intoxication. Similar conclusions were obtained by Hummel et al. (1990). In general, during 40 days of exposure to intoxication, the highest level of PCBs, in wet weight, was found in perintestinal adipose tissue. It was confirmed previously by many authors (Delbeke and Joiris 1988; Sanders and Haynes 1988; Kulharni and Kavara 1990).

Obtained results proved length of intoxication to effect visibly concentration of tested PCB compounds in carp organs and tissues. In general, the highest accumulation dynamics was observed within first 5–10 days of intoxication. It was followed by a drop in accumulation dynamics, which can be explained by essential increase of weight of the tested organs and tissues with the exposure time. When converted to weight of particular organs and tissues increase in PCBs concentrations were essentially higher than accumulation levels (Fig. 2). Besides along with the time of intoxication there was a drop in daily intake of the tested PCB per unit of body weight of the tested carp.

Generally, after 40 days of exposure in water polluted with toxic compounds gill filaments and muscle tissue of carp harboured more PCBs than carp fed with contaminated feed where higher accumulation rate was typical for perintestinal adipose tissue, alimentary tract and liver. The above is consisted with earlier work of Courtney and Langston (1980).

CONCLUSIONS

1. Polychlorinated biphenyls present in water and fish diet penetrate fast into carp body and accumulate in individual organs and tissues.
2. Tested organs and tissues show various accumulation dynamics due to penetration route, intoxication time, and growth rate of weight.
3. Gill filaments and muscle tissue of carp harboured more PCBs, in wet weight, when adsorbed from water than when fed with PCBs contaminated feed, in which case higher concentrations were noted in the perintestinal adipose tissue, alimentary tract, and the liver.
4. In general, after 40 days of intoxication, the perintestinal adipose tissue showed highest PCBs accumulation; with the lowest cumulation noted for alimentary tract and gill filaments, when absorbing PCBs, respectively, from water and the diet.

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DYNAMIKA KUMULACJI PCB W WYBRANYCH NARZĄDACH
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STRESZCZENIE

Obecność polichlorowanych bifenyli (PCB) w środowisku, ich trwałość i toksyczność stanowią poważne zagrożenie dla biosfery. W środowisku wodnym związki te jako nierozpuszczalne w wodzie kumulują się głównie w osadach dennych i organizmach wodnych.

Badania dotyczyły dynamiki kumulacji PCB (Clophen A50) w listkach skrzelowych, tkance mięśniowej, okołojelitowej tkance tłuszczowej, przewodzie pokarmowym i wątrobie karpi hodowlanych (*Cyprinus carpio* L.) przy wchłanianiu go tylko z wody i przy pobraniu z paszą.

Badania analityczne wykonano metodą chromatografii gazowej. Wykazano, że obecne w wodzie i paszy PCB przenikają do organizmu ryb i charakteryzują się zróżnicowaną dynamiką kumulacji w poszczególnych narządach i tkankach w czasie 40 dni ekspozycji. Stwierdzono różnice w dynamice kumulacji w zależności od dróg pobrania toksykantów. Najwięcej PCB w ciągu 40 dni intoksykacji, niezależnie od dróg pobrania kumulowała w mokrej masie okołojelitowa tkanka tłuszczowa. W przeliczeniu na lipidy największy wzrost poziomu koncentracji obserwowano w listkach skrzelowych, a najmniejszy w okołojelitowej tkance tłuszczowej.

Największą dynamikę kumulacji obserwowano w pierwszych 5–10 dniach intoksykacji, a następnie wyraźny jej spadek, który wynikał częściowo z przyrostu masy analizowanych narządów i tkanek oraz spadku podaży skażonej paszy na jednostkę masy ciała analizowanych ryb

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