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Kinetics of Polychlorinated Biphenyls (Aroclor 1254) in Lactating Bovines and Their Distribution in Dairy Products

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ABSTRACT

Lactating cows were given orally, single or multiple graded doses of polychlorinated biphenyls (PCB) as Aroclor 1254 and the tissue distribution and excretion were measured. Persistence of PCB in milk was determined at all dose levels of PCB administered. A distinct predilection of PCB for tissues with high lipid content was noted. Similarly, PCB appeared in higher concentration in dairy products with high fat content.

It is now common knowledge that distribution of polyschlorinated biphenyls[3] (PCB) in the ecosytem is on a global scale (*14, 27*). These compounds have been detected in various forms of aquatic and terrestrial life (*5, 18 22*) and in human tissues and blood samples (*2, 4, 15, 26*). Contamination of agricultural commodities, including meat-producing animals as well as meat and milk products derived from these animals has been reported (*1, 3, 9, 17, 18, 31*). These biphenyls were used for improvement of chemical and water resistance, flexibility and adhesive properties of plastics, paints, lubricants, hydraulic fluids, etc. (*20*). Their present use is restricted to electrical and electronic industries in capacitors and transformers, especially because of their heat-transfer and dielectric properties.

The very properties of PCB that make them useful industrial applications also prevent them from being degraded once they reach the environment. Since their introduction to industry in 1930, PCB have been accumulating in the environment and have reached the latter from various sources. The largest amounts of PCB reaching the environment are estimated to occur from the industrial and municipal discharges into inland and costal waters (*21*).

Polychlorinated biphenyls possess strong lipophilic properties which, coupled with low biotransformation and excretion rates, result in their accumulation in animal lipids and consequent increase through the trophic levels of the food chain (*22, 27*). The toxicity of PCB in laboratory, wild, and domestic animals has been reviewed (*5, 17, 22*). Pathological lesions attributed to PCB in mammals and man consist mostly of liver lesions (*32*) whereas the most commonly observed lesions in birds are hydropericardium, kidney damage, and reduced spleen size (*32*).

Residues of PCB have been found in the milk of cows. The sources of milk contamination included feeding silage contaminated with PCB from silo sealants containing Aroclor 1254 (*9, 10, 11, 30, 31, 32*), use of discarded transformer oil for defoliant spraying (*8*), drinking water from contaminated streams (*24*), and feeding PCB-containing grain and cereal composites (*3*). The presence of PCB in milk is associated with the unsaponifiable fraction of anhydrous milk fat (*6*). The higher chlorinated isomers of PCB are more eliminated into the milk than are the lower chlorinated biphenyls (*16, 25*).

MATERIALS AND METHODS

Eight actively lactating Jersey cows were given the PCB as Aroclor 1254[4] in a single dose or in 10 consecutive daily doses. The PCB, dissolved in olive oil as a 10% solution, was mixed with the morning dairy concentrate. Animals were treated as follows.

Two cows were given a single dose of 10 mg PCB/kg body weight, the second pair of animals received a single dose of 100 mg/kg, and the third and fourth pairs of animals received 10 daily doses consecutively of 1 and 10 mg/kg, respectively. The first four animals were euthanized 10 days after the single treatment and the other four cows were euthanized 10 days after receiving the last dose.

Total urine, obtained by means of permanently installed catheters, and feces were collected for 2 days before, and 10 and 20 days after the single and first multiple administration of PCB, respectively. Milk was collected twice daily during the same time periods and held for later manufacturing purposes.

The animals were euthanized with sodium pentobarbital and immediately subjected to gross pathological examination. Tissues from selected organs were fixed in formaline and stained with hematoxylin-eosin according to routine procedures. Samples from various organs were removed and stored at − 20 C until analyzed. Half of the brain

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[3] *In North America PCB are manufactured exclusively by Monsanto Co. under the trademark of Aroclor. The Aroclors are complex mixtures of PCB varying from 21 to 70% in chlorine contents and their isomers ranging from mono- to decachlorobiphenyls.*
[4] *Aroclor 1254 is an industrial complex mixture of polychlorinated biphenyls containing 54% chlorine by weight.*

was homogenized before PCB analyses. The PCB were extracted from tissues and various dairy products and the extract cleaned using FDA multipesticide residue method (7) and subsequently detected using a Micro-Tek, Model MT-220 gas chromatograph (Tracor Inc., Augusts, Texas, U.S.A.) equipped with a ^{63}Ni high-temperature electron capture detector. The quantitation of PCB was done by measuring total peak area as the detector response, using Infotronics, Model 208 (Infotronics Ltd., Shannon, Ireland) automatic digital integrator equipped with a baseline tracking and drift corrector.

Dairy products were manufactured from control milk collected before treatment, and from milk of low and high dosed cows. The milk of each treatment was bulked and part of each lot separated for manufacture of spray-dried nonfat dry milk. Skimmilk was given several time-temperature treatments to determine the effect on PCB. These treatments included 1 min exposures at 71, 77, and 82 C, 10 min at 82 C, and 10 min at 82 C with gradual cooling to 55 C over 60 min (the time required to draw the skimmilk into the evaporator). Concentration was in a Rogers laboratory evaporator at 45 C vapor temperature. Concentrate of 32.5% total solids was dried in a Swenson laboratory research spray dryer with inlet air temperature of 188 C and outlet temperature of 88 C. Separated cream which was used to manufacture butter was pasteurized at 71 C for 30 min, cooled, and churned in a 10-liter paddle churn. Separated cream was standardized to approximately 16% milkfat for cultured cream manufacture. It was pasteurized at 71 C for 30 min, homogenized, cooled, inoculated with *Streptococcus cremoris*, and incubated at 21 C. Milk for yogurt manufacture was pasteurized at 71 C for 30 min, cooled to 42 C, inoculated with a mixed culture of *Lactobacillus bulgaricus* and *Streptococcus thermophilus*, and incubated for 3 h at 42 C.

RESULTS AND DISCUSSION

Experimental animals did not show any clinical signs of intoxication and upon necropsy, pathological examination failed to reveal any anomalies. Subsequent histological investigation confirmed gross pathology observations.

The concentrations of PCB in tissues of animals are given in Table 1. The highest concentrations of PCB occurred in the adipose tissues of all animals. In cows given a single dose of PCB, the psoas and diaphragmatic muscles contained the second and third highest level of PCB, respectively. The levels of PCB in the kidney of all animals were low, which indicates a low renal excretion of PCB compounds in milking cows. Brain concentrations, however, were always higher than they were in renal tissues. The relatively high PCB levels in brain are an indication of an important transfer of PCB across the blood-brain barrier. Uterus and ovary were low in PCB but the levels of these pollutants were substantially higher in the adrenal gland.

TABLE 1. *Concentrations*[1] *(in μg/g of fresh weight) of PCB in various tissues of milking cows given single or multiple doses of PCB as Aroclor 1254*

Tissue	Single dose		Ten daily doses	
	10 mg/kg	100 mg/kg	1 mg/kg	10 mg/kg
Brain	0.26	1.47	0.64	0.98
Perirenal fat	9.34	43.69	4.32	63.72
Kidney	0.14	0.49	0.09	0.22
Heart	0.27	1.63	0.76	0.19
Liver	0.48	5.64	0.18	3.30
Diaphragmatic muscle	1.01	7.21	0.04	0.84
Psoas muscle	1.70	8.48	0.25	—
Ovary	0.14	0.07	0.18	0.37
Uterus	0.08	0.09	0.14	<0.01
Adrenal gland	0.40	0.98	0.19	2.32

[1]Mean values of two animals

No attempt was made to detect the presence of metabolites in tissues, milk, feces, or urine. The parent compounds were not, however, detected in urine of all animals analyzed.

The highest concentrations of PCB in milk of cows given a single dose of 10 mg/kg and 100 mg/kg occurred on the second day and were 1.9 ppm and 4.6 ppm, respectively. When the multiple administration of PCB stopped, the average concentrations of PCB in milk of animals given 1 mg/kg and 10 mg/kg were 2.1 ppm. and 5.6 ppm, respectively.

The decline of PCB concentrations in blood and milk is depicted in Fig. 1 and 2. The decline portions of these curves indicate two compartment systems. The blood half-lives of PCB in animals given a single dose of 10 mg/kg or 100 mg/kg were in the first portion of the curve, 0.8 and 1.1 days, respectively. The corresponding blood half-life following the cessation of PCB administration in animals given miltiple doses of 1 mg/kg was 0.7 day and in those given 10 mg/kg was 1.2 days.

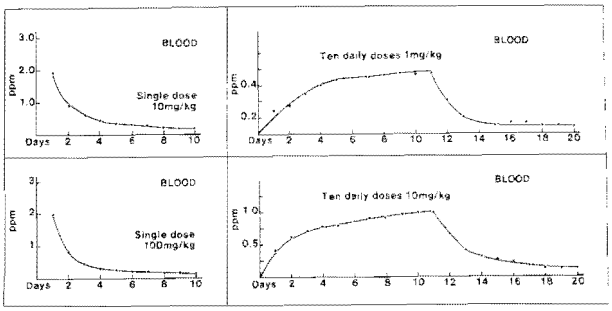

Figure 1. *Concentrations of PCB in the blood of cows as a function of time following the administration of single dose of 10 mg/kg or 100 mg/kg or multiple administration of 1 mg/kg/day or 10 mg/kg/day of Aroclor 1254. Data for each dose level were obtained from two animals.*

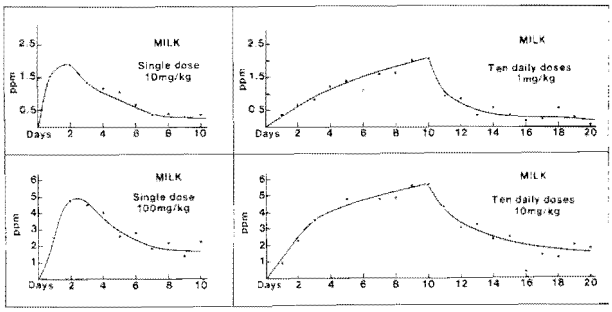

Figure 2. *Concentrations of PCB in the milk of cows as a function of time following the administration of single dose of 10 mg/kg or 100 mg/kg or multiple administration of 1 mg/kg/day or 10 mg/kg/day of Aroclor 1254. Data for each dose level were obtained from two animals.*

The declines in the milk PCB concentrations were somewhat slower than in the blood. The milk PCB half-lives of the first portion of the elimination curve were 1.4 and 1.8 days in animals given a single dose of 10 mg/kg and 100 mg/kg, respectively, and 1.1 and 1.4 days in cows given multiple doses of 1 mg/kg and 10 mg/kg, respectively.

Cumulative eliminations of PCB into milk and feces of animals given single and multiple daily doses of PCB are depicted in Fig. 3 and 4, respectively. In cows given single

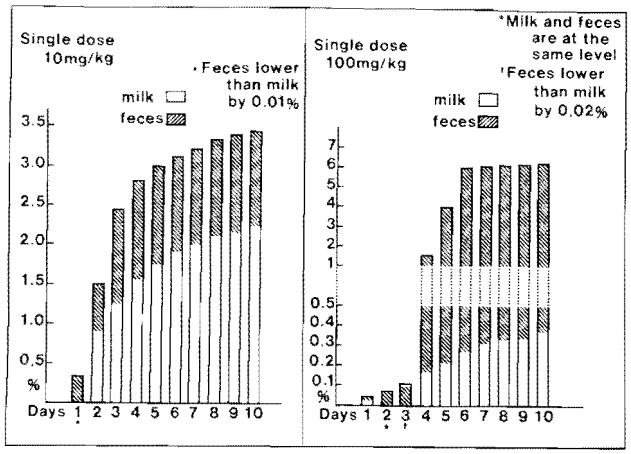

Figure 3. *Percentages of PCB excreted into milk and feces and collected for 10 consecutive days in animals given single dose of 10 mg/kg or 100 mg/kg of PCB. Data are given as cumulative percent of dose administered and obtained from two animals per dose level. Note that both the milk and feces bars begin from the base line.*

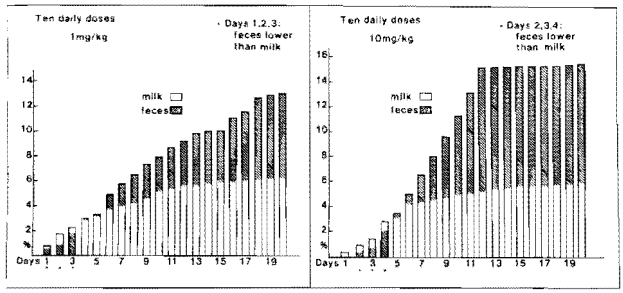

Figure 4. *Percentages of PCB excreted into milk and feces and collected for 20 consecutive days in animals given 10 consecutive daily doses of 1 mg/kg/day or 10 mg/kg/day of PCB. Data are given as cumulative percent of the amount administered and obtained from two animals per dose level. Note that both the milk and feces bars begin from the base line.*

doses of 10 mg/kg or 100 mg/kg, more PCB was eliminated into feces than into milk. Low dosage, however, resulted in proportionally more PCB being eliminated into milk than when cows were given high single doses. Similar results were obtained in cows given multiple doses of PCB. This observation indicates that PCB absorption and consequent elimination in milk is not necessarily proportional to the dose administered to the lactating bovine.

Because of diminishing PCB concentration in the milk of cows given single doses, only the first 4 days' milk from treated cows was used for dairy product manufacture. The bulked milk from cows receiving the higher PCB dosage showed evidence of churning during handling and processing. This sometimes led to difficulties in obtaining representative samples for testing. The same was true to a lesser extent for low dosage milk. The results for PCB and milkfat tests are in Table 2. As expected the PCB followed the fat phase of the milk and dairy products and in all instances the PCB levels were

TABLE 2. *Fat and PCB levels in several dairy products manufactured from milk of control and treated cows*

	Control		Low dose		High dose	
Product	Fat (%)	PCB (μg/g)	Fat (%)	PCB (μg/g)	Fat (%)	PCB (μg/g)
Composite milk	3.83	<0.01	4.38	1.30	3.54	5.90
Skimmilk	0.11	<0.01	0.09	0.04	0.36	0.28
Cream	40.22	<0.01	38.90	12.32	30.60	19.11
Butter	80.14	<0.01	86.14	19.78	85.97	34.10
Buttermilk	0.82	<0.01	0.87	0.61	4.11	5.19
Skimmilk						
71 C-1 min	0.11	<0.01	0.09	0.04	0.36	0.45
77 C-1 min	0.11	<0.01	0.09	0.03	0.36	0.27
82 C-1 min	0.11	<0.01	0.09	0.03	0.36	0.20
82 C-10 min	0.11	<0.01	0.09	0.05	0.36	0.23
82 C-10 min plus 60 min to 55 C	0.11	<0.01	0.09	0.05	0.36	0.16
Nonfat dry milk	1.05	<0.01	1.17	0.15	3.75	0.90
Standardized cream	15.40	<0.01	16.30	6.76	17.52	—
Past'd, homo'd cream + culture	14.47	<0.01	15.34	2.07	16.64	7.02
Cultured cream	14.47	<0.01	15.34	3.97	16.64	8.84
Milk + yogurt culture	3.70	<0.01	3.26	1.58	1.19	1.37
Yogurt	3.70	<0.01	3.26	2.72	1.19	3.20

greater in the higher dose milk. There appeared to be a decline in PCB concentration when skimmilk was heated to 77 C or higher. This supported previous results (*23*). The PCB levels in yogurt and cultured cream appeared to increase in each instance following fermentation. An explanation of this anomaly may be that the conjugated metabolites of PCB and/or the PCB bound to lipoproteins are non-extractable by the method used in the present experiment, but during the fermentation these PCB forms were broken down to their parent compound forms which therefore increased the total PCB present. The low level of fat in the yogurt made from high dose milk is explained by the removal of churned fat before fermentation.

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REFERENCES

1. Bradley, R. L., Jr. 1973. Polychlorinated biphenyls in man's food-a review. J. Milk Food Technol. 36:155-159.
2. Burns, J. E. 1974. Organochlorine pesticide and polychlorinated biphenyl residues in biopsied human adipose tissue, Texas 1969-72. Pesticide Monitor. J. 7:122-126.
3. Department of Health, Education and Welfare, Food and Drug Administration 1973. Polychlorinated biphenyls. Fed. Reg. 38: 18096.
4. Finkles, J., L. E. Priester, J. P. Creason, T. Hauser, T. Hinner, and D. I. Hammer. 1972. Polychlorinated biphenyl residues in human plasma expose a major urban pollution problem. Am. J. Public Health. 6:645-651.

5. Fishbein, L. 1974. Toxicity of chlorinated biphenyls. Ann. Rev. Pharmacol. 14:139-156.
6. Flanagan, V. P., and A. Ferretti. 1973. Hydrocarbons and polychlorinated biphenyls from the unsaponifiable fraction of anhydrous milk fat. J. Lipid Res. 14:306-311.
7. Food and Drug Administration. 1969. Pesticide analytical manual. Department of Health, Education and Welfare. Washington, D.C.
8. Food and Drug Administration. 1970. Status report on chemistry and toxicology of PCB's. Suppl. Washington, D.C.
9. Fries, G. F. 1972. Polychlorinated biphenyl residues in milk of environmentally and experimentally contaminated cows. Environ. Health Perspect. 1:55-59.
10. Fries, G. F., J. Marrow, and C. H. Gordon. 1971. Similarity in behavior of DDE and polychlorinated biphenyl (Aroclor 1254) residues in an environmentally contaminated herd of dairy cows. J. Dairy Sci. 54:796.
11. Fries, G. F., J. Marrow, and C. H. Gordon. 1972. Similarity of a polychlorinated biphenyl (Aroclor 1254) and DDE in rate of elimination from cows. Bull. Environ. Contam. Toxicol. 7:252-256.
12. Furr, A. K., D. R. Mertens, W. H. Gutenmann, C. A. Bache, and D. J. Lisk. 1974. Fate of polychlorinated biphenyls, metals, and other elements in papers fed to lactating cows. J. Agric. Food Chem. 22:954-959.
13. Gardner, A. M., H. F. Righter, and J. A. G. Roach. 1976. Excretion of hydroxylated polychlorinated biphenyl metabolites in cow's milk. J. Assoc. Off. Anal. Chem. 59:273-277.
14. Hutzinger, O., S. Safe, and V. Zitko. 1974. The chemistry of PCB's. CRC Press. Cleveland, Ohio.
15. Inove, Y., S. Abe, H. Esaki, and M. Takamutsu. 1973. Polychlorinated biphenyls in human blood. Kurume Med. J. 20:83-86.
16. Jan. J., M. Komar, and M. Milohnoja. 1975. Excretion of some pure PCB isomers in milk of cows. Bull. Environ. Contam. Toxicol. 13:313-315.
17. Jones, D. H., N. S. Platonow, and S. Safe. 1975. Contamination of agricultural products by halogenated biphenyls. Canad. Vet. J. 16: 349-356.
18. Khan, M. A., R. M. Rao, and A. F. Novak. 1976. Polychlorinated biphenyls (PCBs) in food. Crit. Rev. Food Sci. Nutr. pp. 103-145.
19. Mojonnier Bros. Co. Instruction manual for setting up and operating the Mojonnier milk tester. Bull. 101. Mojonnier Bros. Co., Chicago, Illinois.
20. Monsanto. 1968. Aroclor plasticizers. Technical Bulletin O/PL-306. St. Louis, Missouri.
21. Nisbet, C. T., and A. F. Sarofim. 1972. Rates and routes of transport of PCBs in the environment. Environ. Health Perspect. 1:21-38.
22. Peakall, D. B. 1975. PCB's and their environmental effects. CRC Critical Rev. Environ. Control 5:469-508.
23. Platonow, N. S., H. S. Funnell, D. H. Bullock, D. R. Arnott, P. W. Saschenbrecker, and D. G. Grieve. 1971. Fate of polychlorinated biphenyls in dairy products processed from the milk of exposed cows. J. Dairy Sci. 54:1305-1308.
24. Platonow, N. S., P. W. Saschenbrecker, and H. S. Funnell, 1971. Residues of polychlorinated biphenyls in cattle. Canad. Vet. J. 12: 115-118.
25. Platonow, N. S., and E. B. Meads. 1975. Distribution and excretion of two chlorinated biphenyl isomers: 4-chlorobiphenyl and decachloro-biphenyl in lactating bovine. Canad. J. Comp. Med. 39:104-106.
26. Price, H. A., and R. L. Welch. 1972. Occurrence of polychlorinated biphenyl in humans. Environ. Health Perspect. 1:73-78.
27. Risebrough, R. W., and B. de Lappe. 1972. Accumulation of polychlorinated biphenyls in ecosystems. Environ. Health Perspect. 1: 39-45.
28. Safe, S., N. Platonow, and O. Hutzinger. 1975. Metabolism of chlorobiphenyls in the goat and cow. J. Agric. Food Chem. 23:259-261.
29. Saschenbrecker, P. W., H. S. Funnell, and N. S. Platonow. 1972. Persistance of polychlorinated biphenyls in the milk of exposed cows. Vet. Rec. 90:100-101.
30. Savage, E. P., J. D. Tessari, and J. W. Malberg. 1973. The occurrence of polychlorinated biphenyls (PCB's) in silage stored in pit and upright silos. Bull. Environ. Contam. Toxicol. 10:97-100.
31. Skrentny, R. F., R. W. Hemken, and H. W. Dorough. 1971. Silo sealants as a source of polychlorobiphenyl (PCB) contamination of animal feed. Bull. Environ. Contam. Toxicol. 6:409-416.
32. Vos. J. G. 1972. Toxicology of PCBs for mammals and for birds. Environ. Health Perspect. 1:105-117.
33. Willett, L. B., and Hess, J. F. 1975. Polychlorinated biphenyl residues in silos in the United States. Residue Rev. 55:135-144.