This is your voucher copy;
please return the other set of proofs only.

Dieses Exemplar ist Ihr Beleg;
bitte senden Sie nur das andere Exemplar zurück.

Ceci est votre exemplaire justificatif;
veuillez seulement nous retourner l'autre épreuve.

Primatology 172

S. KARGER AG
Arnold-Böcklin-Strasse 25, CH–4011 Basel

Possible Polychlorinated Biphenyl Poisoning in Rhesus Monkeys (*Macaca mulatta*)

WILBUR P. MCNULTY and DONALD A. GRIFFIN

Oregon Regional Primate Research Center, Beaverton, Oreg., and Department of Agricultural Chemistry, Oregon State University, Corvallis, Oreg.

Key Words. Polychlorinated biphenyls · Poisoning · Rhesus monkey · Infant · Stomach · Sebaceous glands

Abstract. In 1967 68 rhesus monkeys that had been held in new pens for a month or more developed a chronic disease with high mortality. The principal postmortem findings were focal necrosis of the liver and hyperplasia of bile duct epithelium, invasive gastric mucosal metaplasia, and squamous metaplasia of the sebaceous glands. The monkeys were probably poisoned by polychlorinated biphenyls (PCBs), because the pathologic changes were identical with those occurring after experimental feeding of PCBs to monkeys, and because PCBs were found in the livers of the dead animals. The source of the contaminant was not identified.

Introduction

Then years ago polychlorinated biphenyls (PCBs), which had been increasingly used since 1930 in plastics, inks, paints, and sealers, were found to be a global pollutant [9, 15]. Because of their potentially harmful biologic effects, PCBs have since 1970 been manufactured in the United States (trade name 'Aroclors', Monsanto, Akron, Ohio) for only 'closed' uses, such as transformer and capacitor dielectrics and heat exchanger and hydraulic fluids, where the chance of escape to the general environment is small. Foreign manufacturers followed suit in 1971–72. Although environmental levels of PCBs are now said to be falling in some locations [17, 18], this

[1] Supported by NIH grants No. RR00163 and RR05694 to the Oregon Regional Primate Research Center and by ES00210 to the Environmental Health Science Center, Oregon State University.

Short Title: PCB Poisoning in Monkeys

MONS 082611

decline has not been shown to be general, and high levels can be expected to persist in those restricted environments where these chemicals are still used.

The effects in people of ingesting or absorbing PCBs have been described in reports of industrial exposures (reviewed by KIMBROUGH [11] and in the 'Yusho' epidemic in Japan, in which over 1,000 persons were made sick by cooking oil containing PCBs [12]. The victims suffered from chloracne, hyperpigmentation of the skin, and gastrointestinal and neurological disturbances. Squamous metaplasia of the sebaceous and esophageal glands were found at autopsy in two persons dying from unrelated causes [10].

Presumably, nonhuman primates can serve as models for human toxicity. ALLEN *et al.* [3], for example, have shown that low levels of PCBs in the diet of rhesus monkeys *(Macaca mulatta)* caused pathologic changes in the stomach, sebaceous glands, liver, and bone marrow.

In 1967-68 an epidemic disease with a high mortality struck rhesus monkeys kept for only a few weeks in newly constructed enclosures at the Oregon Regional Primate Research Center. The postmortem findings were similar to those later reported after experimental feeding of PCBs [1], and in 1974 PCBs were identified in the formalin-fixed livers of the Oregon animals that had died seven years earlier.

Methods

Before the onset of the epidemic, the small groups of monkeys had been placed for behavioral observations in four identical newly built indoor-outdoor enclosures for four to eight weeks. These pens had an inside space of 60 ft² and were connected by a small swinging door to an outdoor space of 100 ft². The floors of both parts of the enclosures were concrete, to which a commercial sealer had been applied. The walls were cyclone fence except for the partition between the inside and the outside areas; this wall, constructed of cinder block, was the wall of the building. A different sealer had been applied to the cinder block, and the swinging door was painted.

Because of the nature of the behavioral experiment, no detailed physical examinations of the animals were made. When a few of the monkeys became very ill, however, they were removed to cages, but no medications except antibiotics were given.

A conventional autopsy with paraffin sections was performed on most but not all of the animals that died. Samples of gastric mucosa and eyelids, which in retrospect were key sections, were sometimes not taken.

In 1974, stored formalin-fixed samples of liver from some of the monkeys which had died in the epidemics of 1967 and 1968 were analyzed for PCBs by electron capture gas chromatography after acetone extraction and adsorption-elution from alumina with hexane. The individual peak concentrations were determined by a method similar to that of WEBB and MCCALL [19].

MONS 082612

Clinical and Postmortem Observations

Mothers and Infants, 1967

Before the epidemic, 15 pregnant rhesus females, due to deliver within a few weeks, had been moved into the new enclosures. All the mothers delivered live infants, but within a month after birth the babies in all three pens were obviously ill. They were not gaining weight; their eyelids were red and swollen and covered with a crusty exudate; and most suffered from intermittent diarrhea. Except for one infant, which died at age ten days, probably of an unrelated cause, all infants died at ages 41 to 141 days, with a mean of 75 days. The length of exposure of mothers and infants and the times of death are shown in figure 1.

Fifty days after the mothers had first entered the pens, they and the surviving infants were removed from the pens to cages. These infants succumbed within a month, except the one which had been introduced at age two weeks to replace the infant that had died at ten days. This replacement, which had been in the pen for only 16 days, survived intermittent bouts of diarrhea for the next six months and grew normally. She is still alive and has delivered three infants in the breeding colony.

The postmortem findings in liver, stomach, and eyelids and the concentrations of total PCBs in those livers which were analyzed are listed in table 1. The anatomical changes – focal hepatic necrosis and biliary ductal hyperplasia, gastric mucosal mucous metaplasia with submucosal invasion, and squamous metaplasia of the Meibomian glands – are shown in figures 2-5.

When removed from the enclosures, two of the 16 mothers were ill and died after one and two months, respectively. Their pathologic findings were much the same as those of the infants, except that the invasive mucous metaplasia of gastric mucosa was more pronounced.

Juveniles, 1967

Immediately after the mothers and surviving infants had been removed, groups of four to six 2- and 3-years-old juvenile rhesus monkeys were introduced into four enclosures, including the three which had housed the mothers and infants (fig. 1). Two groups, which remained in the pens for 20 and 25 days, respectively, stayed well. All members of a third group, held for 46 days, became sick, three with diarrhea. These three died 13, 44, and 76 days after having been removed. All members of a group held for 62 days became ill, two dying 26 and 63 days after removal. The pathologic findings were the same as those in the infants and mothers (table 1), and PCBs were present in representative livers.

MONS 082613

Mothers and Infants, 1968

In March, 1968, two groups of five near-term pregnant mothers each were put into two enclosures. The babies were born within 1 to 30 days, and both mothers and infants remained in the pens until 78 days after the first entry (fig. 1). Nine of the ten babies died within 1 to 179 days after removal from the pens, with a mean survival time of 99 days. The pathologic findings were the same as before (table 1). All the mothers survived, and their records make no mention of clinical illness. PCBs were again present in preserved livers from these infants (table 1).

PCBs in Liver

The chromatographic profile of PCBs extracted from the liver of one of the infants which died in 1967 is illustrated in figure 6. The pattern was similar for all livers taken from animals dying in both years. The less chlorinated compounds are not as well represented as those of commercial PCBs [19], an indication that the more highly chlorinated homologs are not excreted as rapidly as those with low chlorine content. Two of these highly chlorinated compounds - peaks No. 39 and No. 41 - are not found in Aroclors with code numbers less that 1260 (the last two digits signify the weight percent of chlorine).

Discussion

In randomly selected liver samples of all groups which died in both years, PCBs were present at about half the concentration reported in the liver of an adult monkey dying 68 days after a two-month exposure to Aroclor 1248 at 25 ppm [3]. The levels were also much above that found in the liver of a 'control' monkey from another part of the colony in 1974 (0.18 ppm).

The pathologic lesions found in the stomach, sebaceous glands, and liver were the same as those caused by experimental feeding of monkeys with Aroclor 1248 [1, 3, 4] and Aroclor 1242 [unpublished]. However, these lesions are not pathognomonic for PCBs. The same changes were seen in stomach and sebaceous glands after ingestion of 'toxic fat', which contained 2, 3, 7, 8-tetrachlorodibenzo-*p*-dioxin (TCDD) [2]. Gastric mucosal metaplasia with submucosal invasion was also reported in rhesus monkeys after the ingestion of diesel oil [13] and in *Macaca cynomolgus (M. fascicularis)* in association with infestation by the nematode *Nochtia nochti* [6]. Chloracne, a human skin disease characterized by enlargement and squamous plugging of

MUNS 082614

sebaceous glands, has followed exposure to chlorinated napthalenes [8] and TCDD [16], as well as to PCBs [12, 14].

The case for PCB poisoning in these monkeys rests therefore only on the analytic findings in stored, fixed livers, assuming that contamination did not occur during seven years of storage, and on the parallelism of unusual though not diagnostic pathologic changes.

Although the etiology is not rigorously proven, the presumption is sufficiently strong that investigators with primate colonies should be alert for environmental PCB poisoning. Rhesus monkeys are more susceptible than laboratory rats to the toxic action of PCBs: they sicken and may die at a dietary intake of Aroclor 1248 of 2.5 ppm for two months [3], whereas rats remain in apparent health and show relatively minor biochemical and ultrastructural alterations in the liver after ingesting 25 ppm of Aroclor 1242 for six months [7]. The first clinical sign of intoxication in monkeys, both in the accidentally exposed cases reported here and in animals experimentally fed Aroclor 1242, was swelling and reddening of the eyelids. This was soon followed by lethargy and loss of appetite.

In both outbreaks, morbidity and mortality were highest in the infants. Newborns may be more susceptible, or they may have been exposed to a higher dose per kg through concentration of PCBs in the mothers' milk [5].

The source of the PCBs which we think poisoned these monkeys is unknown. The food as received was not contaminated because the same pelleted chow was used throughout the colony of 1,500 animals, including a breeding colony of 200, without any other untoward effects. It could not have been distributed as an aerosol during washing operations because cohort rhesus monkeys caged only a few feet away remained well. Teh monkeys in the enclosures were in constant contact with the concrete floor, as was their food, and therefore a sealer used on the concrete, or material incorporated in the concrete mix itself, is most suspect. However, the manufacturer of the sealer denied ever using PCBs in his formulation knowingly, and unfortunately no sealer was left for analysis when PCBs were first suspected three years after the outbreak of the disease. After the second outbreak in 1968 and before PCBs were suspected, the enclosures were dismantled and the floor, walls, doors, and fencing were unavailable for analysis.

References

1 Allen, J.R.; Abrahamson, L.J., and Norback, D.H.: Biological effects of polychlorinated biphenyls and triphenyls on the subhuman primate. Environ. Res. 6: 344–354 (1973).

MONS 082615

2 Allen, J.R. and Carstens, L.A.: Light and electron microscopic observations in *Macaca nudatta* monkeys fed toxic fat. Am. J. vet. Res. *28*: 1513–1526 (1967).
3 Allen, J.R.; Carstens, L.A., and Barsotti, D.A.: Residual effects of short-term, low-level exposure of nonhuman primates to polychlorinated biphenyls. Toxic. appl. Pharmac. *30*: 440–451 (1974).
4 Allen, J.R. and Norback, D.H.: Polychlorinated biphenyl- and triphenyl-induced gastric mucosal hyperplasia in primates. Science, N.Y. *179*: 498–499 (1973).
5 Barsotti, D.A.; Carstens, L.A., and Allen, J.R.: The association of transplacental and mammary movement of polychlorinated biphenyls with infant morbidity and mortality. Am. J. Path. *82*: 21a–22a (1976).
6 Bonne, C. and Sandground, J.H.: On the production of gastric tumors, bordering on malignancy, in Javanese monkeys through the agency of *Ancylostoma malchi*, a parasitic nematode. Am. J. Cancer *37*: 173–185 (1939).
7 Brückner, J.V.; Jhanna, K.L., and Cornish, H.H.: Effect of prolonged ingestion of polychlorinated biphenyls on the rat. Fd Cosmet. Toxicol. *12*: 323–330 (1974).
8 Good, C.K. and Penskey, N.: Halowax acne ('cable rash'). Archs Derm. Syph. *48*: 251–257 (1973).
9 Jensen, S.: Report of a new chemical hazard. New Scient. *32*: 612 (1966).
10 Kikuchi, M.; Miyagi, Y.; Hashimoto, M., and Kojima, T.: Two autopsy cases of chronic chlorobiphenyls poisoning. Fukuoka Acta med. *62*: 98–103 (1971).
11 Kimbrough, R.D.: The toxicity of polychlorinated polycyclic compound and related chemicals. CRC crit. Rev. Toxicol. *2*: 445–498 (1974).
12 Kuratsune, M.; Yoshimura, T.; Matsuzaka, J., and Yamaguchi, A.: Epidemiologic study on Yusho, a poisoning caused by ingestion of rice oil contaminated with a commercial brand of polychlorinated biphenyls. Environ. Hlth Perspect. *1*: 119–128 (1972).
13 Lushbaugh, C.C.: Infiltrating adenomatous lesions of the stomach, cecum, and rectum of monkeys similar to early human carcinoma and carcinoma in situ. Cancer Res. *9*: 385–394 (1949).
14 Meigs, J.W.; Albom, J.J., and Kaelin, B.L.: Chloracne from an unusual exposure to Aroclor. J. Am. med. Ass. *154*: 1417–1418 (1954).
15 Nisbet, I.C.T. and Sarofim, A.F.: Rates and routes of transport of PCBs in the environment. Environ. Hlth Perspect. *1*: 21–38 (1972).
16 Oliver, R.M.: Toxic effects of 2,3,7,8-tetrachlorodibenzo-1,4-dioxin in laboratory workers. Br. J. ind. Med. *32*: 49–53 (1975).
17 Sleinhafer, W.G. and Miklas, H.P.: Decline of PCB concentrations in North Atlantic surface water. Nature, Lond. *252*: 387–388 (1974).
18 Veith, G.D.: Recent fluctuations of chlorobiphenyls (PCBs) in the Green Bay, Wisconsin region. Environ. Hlth Perspect. *1*: 51–54 (1972).
19 Wise, R.G. and McCall, A.C.: Qualitative PCB standards for electron capture gas chromatography. J. chromat. Sci. *11*: 366–373 (1973).

Wilbur P. McNulty, Oregon Regional Primate Research Center, 505 N.W. 185th Avenue, *Beaverton, OR 97005* (USA)

MONS 082616

Table 1. Mortality and autopsy findings

	Exposed	Sick	Died	Positive autopsy findings			PCBs in liver
				liver	stomach	eyelids	ppm wet wt
1967 infants	16	16	15	8/8	1/6	3/3	3.30, 4.91, 5.96
mothers	16	2	2	2/2	2/2	1/1	4.35, 5.56
1967 juveniles	23	14	5	5/5	5/5	2/2	1.64, 3.28
1968 infants	10	10	9	8/9	3/7	7/7	0.84, 2.71, 3.47
mothers	10	0	0	–	–	–	–

Fig. 1. Durations of exposure (shaded areas) and survival times of rhesus monkeys placed in pens contaminated with PCBs.

Fig. 2. Focal parenchymal necroses in liver of newborn rhesus monkey infant dying after 67 days in a contaminated pen. HE. × 100.

Fig. 3. Hyperplasia of lining epithelium of medium-sized intrahepatic bile duct in newborn rhesus infant surviving 11 days after 69 days in a contaminated pen. HE. × 40.

Fig. 4. Mucous metaplasia and hyperplasia of gastric epithelium with invasion of the submucosa in a rhesus mother surviving 55 days after 113 days in a contaminated pen. HE. × 10.

Fig. 5. Transverse section of eyelid with squamous metaplasia of Meibomian glands in newborn rhesus infant surviving 73 days after 86 days in a contaminated pen. HE. × 10.

Fig. 6. Electron capture gas chromatograms of PCBs extracted from the liver of a newborn rhesus infant surviving 30 days after 54 days in a contaminated pen, and of stock Aroclor 1260. Coincident peaks 37, 39, and 41 are not present in Aroclors with code numbers less than 1260.

MUNS 082617

MUNS 082018

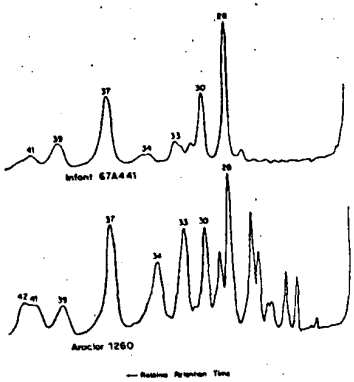

41
39
37
34
33
30
29
Infant 67A441
42 41
39
37
34
33
30
29
Aroclor 1260
Relative Retention Time

Fig 6

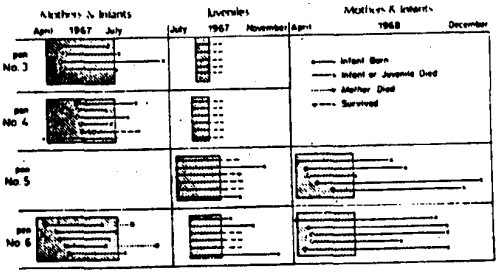

Mothers & Infants
Agmi 1967 July
Juveniles
July 1967 November
Mothers & Infants
April 1968 December
pen No. 3
pen No. 4
pen No. 5
pen No. 6
Infant Born
Infant or Juvenile Died
Mother Died
Survived

Fig 1

Appendix B

	Monkey	Aroclor 1242 Level in Food (ppm)	Est. Total Dose, mg/kg	Survival (days) Exposure	Survival (days) Recovery	Total PCB Level in Liver (ppm)
High Level						
	5454	100	170	34	--	129
	5466	100	200	40	609*	0.7
	5526	200	270	27	--	227
	5571	200	400	40	45	ND
	5558	400	800	40	382	24
	5464	400	800	40	184	ND
	5469	800	1,560	39	--	ND
	5591	800	1,600	40	2	478
Low Level						
	6388	3	37	245	--	7
	6422	10	73	146	--	51
	6427	30	136	91	--	35
	6398	100	685	137	--	160

*Clinically well, killed for autopsy.



Photomicrographs (35x magnification) of the wall of a normal rhesus monkey stomach (right) and the stomach of a rhesus monkey eating food with Aroclor 1242 (3 ppm) for 8 months. Arrows indicate normal plane of separation of mucous membrane and muscle.

MUNS 082621

APPENDIX D

Curriculum Vitae

Wilbur P. McNulty, M.D.

Social Security Number 315-20-2388

Born 9-23-25, Iowa City, Iowa, U.S. Citizen

Married, 5 children

Present Title: Chairman, Laboratory of Pathology, Oregon Regional Primate Research Center

Education:

Yale University, New Haven, Conn., B.S., Mathematics, 1947

Yale University School of Medicine, New Haven, Conn., M.D., Medicine, 1952

Positions held:

Scientist, Oregon Regional Primate Research Center, Chairman, Laboratory of Pathology, 1963 - present
Member, Scientific Advisory Board of the Wisconsin Regional Primate Research Center, 1975 -
Director, Automatic Data Processing Department, Oregon Regional Primate Research Center, 1967-1971
Assistant Professor, Pathology, Yale Medical School, 1963
Director, Clinical Laboratories, Griffin Hospital, Derby, Conn., 1959-1963
Instructor, Pathology, Yale Medical School, 1959-1963
Jr. Staff, Hepatic and Pediatric Pathology, Armed Forces Institute of Pathology, 1958-1959
Captain, U.S. Army, Walter Reed Army Institute of Research, Experimental Biology, 1956-1958
Intern and Resident, Pathology, Yale Medical School, 1954-1956
Instructor, Physiology, University of Pennsylvania, 1952-1954
Fellow, Biophysics, Yale University, 1950-1951

Academic and Professional Honors:

Phi Beta Kappa, 1947
Sigma Xi (Assoc., 1947; Full, 1952)
Alpha Omega Alpha, 1950
Certified, American Board of Pathology (anatomy) 1959

Membership in Societies:

American Association for the Advancement of Science (1961 - present)
New York Academy of Sciences (1962 - present)
Biophysical Society (1962 - present)
Tissue Culture Society (1965 - present)
Association for Computing Machinery (1967 - present)
International Academy of Pathology (1970 - present)

Publications

McNulty, W. P., Jr., and F. Hutchinson: Deuteron inactivation of bovine serum albumin. Arch. Biochem. Biophys. 50:92 (1954).

Schaeffer, K. E., W. P. McNulty, Jr., C. Carey, and A. A. Liebow: Mechanisms of development of interstitial emphysema and air embolism on decompression from depth. J. Appl. Physiol. 192:15 (1958).

Shively, J. N., H. L. Andrews, H. P. Miller, A. P. Warner, Jr., and W. P. McNulty, Jr.: Responses of swine to high doses of radiation. Proc. Soc. Exp. Biol. Med. 101:74 (1959).

Rothberg, H., E. B. Blair, A. C. Gomez, and W. P. McNulty, Jr.: Observations on chimpanzees after whole-body radiation and homologous bone marrow treatment. Blood 14:1302 (1959).

McNulty, W. P. Jr., and R. Linares: Hemorrhagic shock of germ-free rats. Am. J. Physiol. 198:141 (1960).

McNulty, W. P., Jr.: Tumor viruses. Adv. Biol. Skin 7:133 (1966).

Hall, A. S., and W. P. McNulty, Jr.: Contagious pox disease in monkey. J. Am. Veterinary Med. Assoc. 151:833 (1967).

Knezevich, A. L., and W. P. McNulty, Jr.: Tuberculosis in Lemur Mongoz. Folia Primatol. 6:152 (1967).

McNulty, W. P., Jr., W. Lobitz, F. Hu., C. A. Maruffo, and A. S. Hall: A pox disease in monkeys transmitted to man. Clinical and histological features. Arch. Dermatol. 97:286 (1968).

Nicholas, A., and W. P. McNulty, Jr.: In vitro characteristics of poxvirus isolation from rhesus monkeys. Nature 217 (5130):745 (1968).

Smith, A. A., and W. P. McNulty, Jr.: Salivary gland inclusion disease in the Tarsier. Lab. Anim. Care 19:479 (1969).

McNulty, W. P., Jr., and H. M. Wuest: Thalidomide, teratogeny, and structure: Teratogenic action of WU 385 on the rhesus. Teratology 2:265 (1969).

Knezevich, A. L., and W. P. McNulty, Jr.: Pulmonary acariasis in colony-bred rhesus monkey. Lab. Anim. Care 20:693 (1970).

McNulty, W. P., Jr., and Wuest, H. M.: The teratogenicity of a thalidomide analog in rhesus monkeys. In: Proceedings of the Third Congress of the International Society of Primatology. Vol. 2, pp. 238-245, Karger, Basel (1971).

McNulty, W. P., Jr., and M. R. Malinow: Cardiovascular pathology in primates. In: Pathology of Simian Primates (ed. by R. N. T-W-Fiennes). Part I, pp. 756-808, Karger, Basel (1972).

McNulty, W. P., Jr.: Pox diseases in primates. In: Pathology of Simian Primates (ed. by R. N. T-W-Fiennes). Part II, pp. 612-645, Karger, Basel (1972).

Hill, J. D., M. R. Malinow, W. P. McNulty, and A. J. Ochsner: Experimental myocardial infarction in unanesthetized monkeys. Am. Heart J. 84: 82 (1972).

McNulty, W. P.: Spontaneous cardiopulmonary disease in nonhuman primates: potential models. In: Research Animals in Medicine (ed. by L. T. Harmison). pp. 829-839, U. S. Government Printing Office, Washington, D. C. (1973).

Malinow, M. R., J. D. Hill, M. Lees, W. P. McNulty, and A. J. Ochsner, III: Experimental myocardial infarction in nonhuman primates. In: Research Animals in Medicine (ed. by L. T. Harmison). pp. 105-118, U. S. Government Printing Office, Washington, D. C. (1973).

McNulty, W. P. (editor) Nonhuman Primates and Human Diseases, Vol. 4 of Symposia of the Fourth International Congress of Primatology. Karger, Basel, 149 pp. (1973).

McNulty, W., F. Hagemenas, N. Beamer, and G. Kittinger: The effect of chronic decapitation on the development of the rhesus fetus (Macaca mulatta). Program for the Endocrine Society 55th Annual Meeting, p. A-175 (1973).

Pasztor, L. M., F. Hu, and W. P. McNulty, Jr.: 5-Bromodeoxyuridine-tolerant melanoma cells in vitro and in vivo. Yale J. Biol. Med. 46:397 (1973).

Ferron, R. R., R. S. Miller, and W. P. McNulty: Estimation of fetal age and weight from radiographic skull diameters in the rhesus monkey (Macaca mulatta). J. Med. Primatol. 5:41-48 (1976).

McNulty, W. P.: PCB toxicity in rhesus monkeys. National Conference on "Polychlorinated Biphenyls." Chicago, Illinois, November, 1975. pp. 347-350 (1976).

Malinow, M. R., P. McLaughlin, L. Papworth, H. K. Naito, L. Lewis, and W. P. McNulty: A model for therapeutic interventions on established coronary atherosclerosis in a nonhuman primate. In: Atherosclerosis Drug Discovery (ed. by C. E. Day). pp. 3-31, Plenum Publishing Corporation, New York (1976).

Malinow, M. R., P. McLaughlin, D. S. Dhindsa, J. Metcalfe, A. J. Ochsner, III, J. Hill, and W. P. McNulty: Failure of carbon monoxide to induce myocardial infarction in cholesterol-fed cynomolgus monkeys (Macaca fascicularis). Cardiovasc. Res. 10:101-108 (1976).

MUNS 082624

McNulty, W. P. and D. A. Griffin: Possible polychlorinated biphenyl poisoning in rhesus monkeys (Macaca mulatta). J. Med. Primatol. 172: (1976), in press.

McNulty, W. P.: Toxicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin for rhesus monkeys: Brief Report. Submitted to the Bulletin of Environmental Contamination and Toxicology.