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Accumulation of Polychlorinated Biphenyls in Atlantic Tomcod (*Microgadus tomcod*) Collected from the Hudson River Estuary, New York

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The Hudson River estuary contains elevated levels of polychlorinated biphenyls (PCBs) throughout its course. HORN et al. (1979) estimated that almost 300 metric tons of residual PCBs are present in the sediments, a level of contamination exceeding other aquatic ecosystems in the United States (US EPA 1976). General Electric's capacitor manufacturing facilities in the upper river at Fort Edward and Hudson Falls, New York, were the major sources of PCBs (HETLING et al. 1979). Discharges of PCBs from these facilities were sharply curtailed in 1976 and terminated in June 1977. PCB concentrations in the Hudson estuary recently ranged from 4 to 6 µg/g in the sediments and 0.0001 to 0.0011 mg/l in the water column.

PCB contamination in the estuary resulted in the closure of the striped bass and American eel fisheries in early 1976. PCB levels in the edible flesh of several fish species are now regularly monitored (HETLING et al. 1979, ARMSTRONG & SLOAN 1980). These surveys are concerned with human health protection. PCB effects on the health of the exposed fish populations have not been investigated.

A recent study of adult Atlantic tomcod (Microgadus tomcod) collected during the winter spawning season observed grossly visible abnormalities in livers characterized by enlargement and discoloration (SMITH et al. 1979). Histopathological examinations suggested a 25 percent frequency of hepatocellular carcinoma in the 1977-78 spawning population. The cause(s) of the hepatomas was unknown; but based on preliminary tests which revealed PCB residues in liver tissues, SMITH et al. (1979) speculated that PCBs may play a role.

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We present here the results of PCB residue measurements in liver, gonadal and body tissues of adult Atlantic tomcod collected from the same spawning population studied by SMITH et al. (1979). SPAGNOLI & SKINNER (1977) and HORN et al. (1979) presented data on PCB levels in edible body tissues of Atlantic tomcod collected in the Hudson estuary, but PCB levels in liver and gonadal tissues have not been reported. We also examined the available data for evidence that PCBs were involved in hepatoma induction.

MATERIALS AND METHODS

Test Animals

The histopathological studies of SMITH et al. (1979) and our tissue contaminant studies were incidental to a larger project investigating the impact of power plant operations on Atlantic tomcod and other fish populations. Adults were collected during the winter spawning season (mid-January through mid-February 1978) on the spawning grounds in box traps (0.9m x 0.9m x 1.8m) set along the shore of the estuary. Random samples of fresh specimens were regularly returned to the laboratory for routine age, growth, maturity and fecundity measurements.

Three adults from each of the three categories of gross abnormal liver appearance defined by SMITH et al. (1979) as hemorrhagic, small pustules, tumors, were randomly selected for PCB analysis from samples collected for routine age and growth measurements. Three adults whose livers were normal in gross appearance were also randomly selected for PCB analysis. The liver, gonadal and body (remaining) tissues from eight males and four females ranging from 142 to 213 mm (total length) were dissected, separated, individually labelled and frozen at $-5^{\circ}C$ in containers washed with methanol and rinsed with hexane.

Chemical Analyses

Tissues were analyzed in accordance with THOMPSON (1977). Frozen tissues were thawed, macerated with a glass rod, and the fatty portions separated by repetitive extractions with petroleum ether. Halogenated organics were then separated by acetonitrile partitioning. The resulting extracts were concentrated by evaporation in a Kuderna-Danish (K.D.) apparatus, eluted through a Florisil column with 6% ethyl ether in petroleum ether, and concentrated for analysis by another K.D. evaporation.

A portion of the extract was perchlorinated using the method described by ARMOUR (1973). Analysis of this extract for decachlorobiphenyl was used to quantify total PCBs in the sample. Analyses of all extracts were performed on a Perkin-Elmer Model 910 gas chromatograph equipped with electron capture detectors. A packed column of 5% OV-210 on Supercoport was used for initial identification and for quantification of the decachlorobiphenyl. Another packed column consisting of 1.5% OV-17 and 1.95% OV-210 on Supercoport was used to aid

identification of different Aroclors. Recoveries were determined from Aroclor standards which were added as spikes and taken through all of the separation and concentration steps. Results were corrected for both solvent blanks and for recoveries. Concentrations are reported in µg/g wet weight of tissue.

RESULTS AND DISCUSSION

PCBs, identified as a mixture of Aroclors 1016 and 1254, were detected in all tissues (Table 1). All of the major gas chromatograph peaks could be attributed to these Aroclors; therefore, there was no evidence of other halogenated organic compounds in the 6% ethyl ether Florosil fraction. The small sample size (n = 12) and wide variation in the data limited statistical analysis, however, some potentially important trends were apparent.

Concentrations of PCBs in body tissues measured in our study (mean 0.17 µg/g, range 0.01 to 0.67 µg/g) are lower than other reports on Atlantic tomcod collected in the Hudson estuary. HORN et al. (1979) reported a mean concentration of 0.96 µg/g total PCBs (range 0.44 to 4.48 µg/g) from edible flesh. SPAGNOLI & SKINNER (1977) reported mean concentrations of 6.5 µg/g and 7.7 µg/g, respectively, for Aroclors 1242/1016 and 1254 in whole carcasses, with concentrations of 1.5 µg/g and 1.3 µg/g respectively in edible flesh.

Mean concentrations of total PCBs in gonads and livers were 1.17 µg/g (range 0.08 to 7.35 µg/g) and 37.52 µg/g (range 10.94 to 98.22 µg/g) respectively (Table 1). PCB residues in ovarian tissues were generally lower than in testes. We are unaware of previous measurements of PCB residues in Atlantic tomcod gonads and livers. UTHE et al. (1980) reported concentrations of PCBs in testes and livers of a related species, Atlantic cod (Gadus morhua), ranging from 0.05 to 5.3 µg/g and from 3.5 to 374 µg/g wet weight respectively. These concentrations were observed in groups of sexually maturing males fed 1, 5, 10, 25 or 50 µg of Aroclor 1254 per g of diet three times per week for 92 days.

We noted a trend toward lower levels of PCBs in the tissues of larger Atlantic tomcod (Table 1). However, the only statistically significant ($p < 0.05$) association was between body length and PCB concentration in livers of males and females combined ($r = -0.602$). This association was not significant for either males or females analyzed separately. We did not determine the percent lipid in any of the tissue samples.

Levels of PCB residues among body, liver, and gonadal tissues were generally not significantly associated. The single significant ($p < 0.05$) direct association ($r = +0.811$) between the concentration of PCBs in gonads and livers may have been spurious because it was dependent upon the relatively high concentration in the testes of one male (Table 1). When this

TABLE 1

Age, length, liver appearance category, and total PCB concentration in tissues of twelve Atlantic tomcod collected in the Hudson River estuary during January and February 1978

Liver Category[b] (gross external appearance)	Age (yrs)	Sex	Length (mm TL)	Total PCBs (µg/g)[a] Body[c]	Testis	Ovary	Liver
Normal	1	M	163	0.10	1.64		29.43
Normal	1	M	179	0.02	0.86		20.32
Normal	1	M	150	0.08	0.64		49.18
Hemorrhagic	1	F	165	0.04		0.10	15.09
Hemorrhagic	1	M	150	0.10	0.19		31.50
Hemorrhagic	1	M	152	0.09	0.83		59.07
Small Pustules[d]	1	F	187	0.08		0.08	10.94
Small Pustules	1	M	142	0.67	7.35		98.22
Small Pustules	1	M	168	0.04	0.89		56.35
Tumors[d]	1	F	146	0.05		0.83	36.96
Tumors	1	F	190	0.66		0.12	16.02
Tumors	2	M	213	0.11	0.46		27.12
Mean				0.17	1.61	0.28	37.52

[a] Based on wet weight
[b] After categories described by SMITH et al. (1979)
[c] Homogenate of entire fish after liver and gonad were excised
[d] Categories of livers which contained histological evidence of neoplastic nodules of hepatocytes and hepatocellular carcinoma (SMITH et al. 1979)

M = male
F = female
TL = total length

individual was removed from the analysis, the association was no longer significant.

PCB residues in livers whose external appearance was normal varied by a factor of 2.4 and the mean concentration was not discernably different from mean PCB concentrations in abnormal livers (Table 1). Hence, no association between PCB concentration and type of liver abnormality was discernable. More recent histopathological studies revealed that several Atlantic tomcod livers, collected from the 1980-81 spawning population, which were normal and hemorrhagic in external appearance contained neoplastic nodules (personal communication with C. E. Smith, USFWS Fish Cultural Development Center, Bozeman, Montana). Thus, some or all livers classified as normal and hemorrhagic by SMITH et al. (1976) and analyzed in our study for PCBs (Table 1) may have eventually developed neoplastic nodules and hepatocellular carcinoma. Normal livers may be rare in the Hudson River population of Atlantic tomcod.

In conclusion, this study shows that the tissues of adult Atlantic tomcod collected in the Hudson River estuary contain elevated levels of PCBs, especially in livers. However, any role of PCBs in the induction of hepatomas observed by SMITH et al. (1979) is unclear. The PCB-hepatoma induction hypothesis is plausible and deserves further study because: 1) Hudson River sediments are heavily contaminated with PCBs in areas inhabited by Atlantic tomcod (HORN et al. 1979), 2) Atlantic tomcod are bottom-feeding fishes (SCOTT & CROSSMAN 1973) and therefore probably continually exposed to PCB contaminated sediments, 3) PCBs concentrate in fish livers (HANSEN et al. 1971, 1976; CAMP et al. 1974; ZITKO & HUTZINGER 1976; NARBONNE 1979), 4) PCBs have been associated with hepatocellular carcinoma and other liver abnormalities in wild fish populations (FALKMER et al. 1977; McCAIN et al. 1977; PIERCE et al. 1978, 1980), 5) Liver anomalies have been induced in fish exposed to PCBs under laboratory conditions (JENSEN et al. 1970; JOHANNSON et al. 1972; HANSEN et al. 1974; COUCH 1975; LIPSKY et al. 1978; KLAUNIG et al. 1979; UTHE et al. 1980), and 6) Even though there is no evidence that PCBs are hepatocarcinogenic in teleosts (PIERCE et al. 1978), they are hepatocarcinogenic in some mammals (FISHBEIN 1974). Further studies will be required to identify the causal relationship, if any, between PCBs and hepatocellular carcinoma observed in Atlantic tomcod from the Hudson River estuary.

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