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Polychlorinated Biphenyl Residues in Blubber of Male Atlantic Bottlenose Dolphins *(Tursiops truncatus)* That Stranded and Died at Matagorda Bay

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Prior to 1976, polychlorinated biphenyls (PCBs) were released into the environment in the course of legal manufacture and use. Today, PCB pollution results largely from volatilization and runoff from hazardous waste sites, leakage from aging PCB-containing equipment, and accidental or deliberate dumping (Llados and Faroon 1997). The degree ofpollution by PCBs and related organochlorines (OC) varies by source proximity, with heavily industrialized areas being more contaminated than remote sites (Tanabe and Tatsukawa 1986). These pollutants are not confined to waters proximal to industrial centers. Rather, PCB residues have been detected in the air (Tanabe et al. 1983) water (Hargrave et al. 1988), and biota (Tanabe et al. 1987), and small cetaceans in pristine waters have been found to have parts per million (ppm) levels of PCBs in their blubber (Tanabe et al. 1988), indicating planet-wide dispersion of PCBs. This widespread distribution of PCBs has resulted in their being called "current constituents of living beings in our epoch" (Wassermann et al. 1979).

PCB manufacture was banned in Japan in 1972 and in the U.S. in 1976. Since that time, PCB concentrations have decreased in many areas where they were once quite high. Unfortunately, discontinued production and diminished levels of PCBs at pollution sources have not led to universally decreased levels in the evironment. PCBs are environmentally and metabolically stable, and concentrations of PCBs and other OC in remote sites may remain detectable for years following removal of the contaminant source (Iwata et al. 1994). Bioconcentrated residues of PCBs in storage lipids may be quite difficult to eliminate, as seen by the minimal decrease in PCB concentrations in polar bear tissues over a twenty year period after the ban of their manufacture (Norstrom et al. 1988). The problem is even worse in cetaceans, which, due to their long lives, high fat content, and slow metabolism of hydrocarbons, efficiently bioconcentrate these compounds. Residues of PCBs in tissues of striped dolphins in a remote site off Japan increased for more than a decade following restrictions on manufacture and use of the chemicals (Loganathan and Kannan 1991). Furthermore, the environmental release of PCBs has not stopped. In 1988, Tanabe estimated that sixty-five percent of all the PCBs ever produced remained in dumps, landfills, or storage, or was still in use in aging equipment, and in 1989 it was estimated that between 11 and 17 tons of total PCBs were deposited in the North Sea each year from existing reservoirs. While the disposal and/or destruction of PCBs and other OC are now regulated, there continues to be an enormous potential for harm to biological organisms from the existing reservoir of pollutants.

During the winter of 1990 a number of Atlantic bottlenose dolphins stranded and died in the Matagorda Bay area of the Texas gulf coast. This event was originally studied in a combined effort by NOAA, the National Marine Fisheries Service, and the Southeast Fisheries Science Center (Hansen 1992). In this paper, the authors present data from a continuation of those studies showing the levels and TEQ values of a number of PCB congeners extracted from the blubber of bottlenose dolphins, *T. truncatus.*

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MATERIALS AND METHODS

PCBs were determined at the TAMU Geochemical and Environmental Research Group (GERG) laboratory. Tissue samples were excised to include the entire thickness of blubber plus the skin and some underlying muscle, wrapped in foil and frozen until analyzed. Aliquots of 0.5 to 0.81 g were weighed, minced, and macerated x3 in high purity dichloromethane (DCM) with a Tissumizer for 3 min. Extracts were filtered through sodium sulfate and concentrated in a Kuderna-Danish (KD) concentrator to 1 mL in GC-MS grade hexane. A 100 µL aliquot was dried for lipid determination. Chromatography of the extracts through a column containing copper beads, 5% deactivated silica gel (60-200 mesh) and 1% deactivated alumina with pentane followed by 1:1 pentane:DCM as the mobile phases removed any sulfur and separated aliphatic hydrocarbons and polar interferences from the analytes. Eluates were reduced to 1 mL in hexane using a KD concentrator. Lipids were separated from the analytes in DCM using a Thermo Separation Products AS100 HPLC equipped with a Phenomenex Phenogel 10 µm 100Å column, 7 mL/min, and a Waters 440 absorbance detector. Fractions were concentrated, aliquots were diluted and analyzed on a Hewlett Packard model 5890 gas chromatograph with an HP 7630 autosampler, a 30 m, 0.25 internal diameter J&W DB-5 fused silica column, and an HP G1223A electron capture detector equipped with a 15 mCu nickel-63 source. The 2 µL injection was splitless, and the injection port temperature was 275°C. Helium was the carrier gas. The 99 min run used a 5°C/min ramp from 100°C to 140°C a 1.5°C/min ramp to 250°C and a 10°C/min ramp to 300°C with 1 min holds at each level and a 10 min hold at the end. Coplanar congeners 77, 81, 126, and 169 were analyzed separately. Blubber samples were weighed and extracted as above. The extract was eluted through a 5 g charcoal column, first with 40 mL of 90/10 DCM/hexane to remove unwanted PCBs, then with 30 mL toluene to elute coplanar PCBs. The second fraction was concentrated to 100 µL and run on the GC/ECD in a 32 min run using a 6°C/min ramp from 120°C to 300°C with one min holds at the beginning and end.

Controls for GC methods included procedural blanks, internal standards, calibration standards, a matrix spike sample, and Standard Reference Material (SRM) 1945 (National Institute of Standards and Technology, Gaithersburg, MD). Internal standards for samples consisted of 99.6 µg 4,4-dibromooctafluorobiphenyl, 102.7 µg PCB 103, and 108.6 µg PCB 198 added before extraction and 105.3 µg tetrachloro-*m*-xylene added after extraction in the first method. In the analysis for coplanar PCBs, 2.1 ng of polybrominated biphenyl congener 77 was added after extraction. Calibration standards included a mixture of 22 of the PCB congeners at 200 ng/ml each and three dilutions of this mixture, 80 ng/ml, 20 ng/ml, and 5 ng/ml. Each mixture also contained the internal standards. Sample extracts were typically diluted and rerun if the analyte concentrations exceeded the range of the calibration standards. Recoveries were calculated using a matrix spike sample, a duplicate of a dolphin sample plus internal standards except for the addition of 40 ng of all ofthe calibrated analytes. SRM 1945 is a preparation of pilot whale blubber containing certified concentrations of 15 chlorinated pesticides and 27 PCB congeners. All glassware was combusted in a 400°C oven. Solvents were verified to be free of analytes by GC/ECD analysis after 200-fold concentration.

RESULTS AND DISCUSSION

Blubber samples from the ten best preserved male *T. truncatus* of 26 animals that stranded at Matagorda Bay were evaluated by GC/ECD, yielding 87 PCB analytes (Table 1). Of these, 19 congeners co-eluted, giving 68 separate values. Total PCBs per animal ranged between 13.7 ppm and 41.4 ppm. Analysis of residues showed a weak correlation (R=0.45) between estimated age and total PCBs. However, two animals with excessive PCB residues, 28 ppm at age 4 and 41 ppm at age 17, appeared to skew the data. The eight other male dolphins exhibited residue levels that appeared to correlate well (R=0.86) with age of the animal estimated by length and girth measurements (data not given).

Table 1. A GC/ECD analysis of PCB congeners in blubber samples from Atlantic bottlenose dolphins (*Tursiops truncatus*). Blubber from dolphins that stranded at Matagorda Bay was evaluated for PCB congeners reported as ng/g lipid weight (ppb).

Dolphin ID	PO 101	PO 106	PO 94	PO 99	PO 110	PO 117	PO 100	PO 98	PO 109	PO 104	Mean
PCB 15	82.2	18.7	50.1	70.5	24.9	16.0	32.9	28.1	12.3	43.0	37.9
PCB 18/17	47.5	27.8	47.4	24.6	23.9	24.6	23.8	19.9	18.3	23.3	28.1
PCB 22/51	3.7	1.8	ND	4.9	ND	5.4	6.8	ND	1.8	ND	2.4
PCB 25	ND	ND	ND	ND	57.8	ND	ND	ND	ND	ND	5.8
PCB 26	1175.1	1928.0	600.9	2216.6	1151.5	2196.4	3563.0	2657.9	1481.4	2289.4	1926.0
PCB 28	32.1	29.8	36.8	ND	30.3	16.1	ND	7.7	28.1	ND	18.1
PCB 31	ND	ND	ND	ND	ND	ND	24.7	ND	ND	24.4	4.9
PCB 40	9.5	ND	47.4	ND	ND	3.3	ND	ND	ND	ND	6.0
PCB 44	89.1	92.7	123.8	104.1	56.1	58.8	42.7	81.0	64.4	94.7	80.7
PCB 45	12.0	6.5	31.4	14.0	8.1	7.1	7.1	12.6	7.5	7.1	11.3
PCB 47/75	172.2	262.6	401.8	261.0	146.3	317.3	304.8	282.1	135.7	263.2	254.7
PCB 48	60.3	ND	ND	ND	41.1	ND	ND	52.0	26.3	ND	18.0
PCB 49	127.5	52.9	155.6	61.5	77.4	57.1	55.0	86.1	39.3	61.1	77.4
PCB 52	367.0	444.9	522.6	487.6	359.8	653.8	646.7	636.2	362.5	495.9	497.7
PCB 56/60	ND	111.6	98.5	64.5	36.2	87.4	209.4	96.0	45.3	70.9	82.0
PCB 66	33.4	62.7	65.7	22.4	48.6	ND	ND	ND	45.4	ND	27.8
PCB 67	80.2	151.3	187.5	63.5	61.7	44.4	65.8	56.1	43.5	68.6	82.2
PCB 69	13.2	7.1	ND	ND	3.8	5.7	ND	9.0	4.4	7.2	5.0
PCB 70	1.2	31.3	71.3	28.4	19.6	38.4	88.2	56.8	14.4	47.9	39.8
PCB 74/61	141.8	200.4	289.8	77.3	124.1	82.1	80.3	110.8	105.7	129.8	134.2
PCB 83	32.1	ND	39.2	ND	12.1	15.6	ND	29.0	11.3	12.4	15.2
PCB 87/115	115.8	69.5	110.7	53.1	55.6	48.1	37.6	74.2	41.9	55.4	66.2
PCB 91/55	263.3	343.5	443.6	332.4	235.6	235.3	416.6	351.1	232.1	327.0	318.0
PCB 92	208.6	359.8	586.9	357.4	285.4	299.1	465.0	513.6	309.3	332.7	371.8
PCB 95/80	267.4	360.5	673.6	384.7	289.6	454.4	670.1	605.1	284.3	501.5	449.1
PCB 97	46.9	16.5	101.2	9.5	11.1	9.8	10.1	15.7	7.7	13.3	24.2
PCB 99	902.8	1283.9	1571.4	1417.2	766.0	1592.2	2560.0	2145.1	1060.8	1421.6	1472.1
PCB 101/90	826.6	749.5	1221.0	633.8	589.4	658.0	790.9	943.7	624.3	678.5	771.6
PCB 105	197.8	205.4	308.8	ND	143.5	187.0	205.8	200.7	180.0	194.4	182.3
PCB 110	107.5	102.2	166.8	ND	80.5	ND	ND	ND	88.5	ND	54.6
PCB 118	770.7	624.6	1052.3	445.8	494.8	412.4	384.2	660.9	547.6	524.6	591.8
PCB 119	93.8	126.7	134.5	93.5	64.7	89.5	138.9	131.3	73.1	89.0	103.5
PCB 128	323.6	384.6	528.4	459.8	243.8	605.3	744.8	724.7	289.9	512.8	481.8

Dolphin ID	PO 101	PO 106	PO 94	PO 99	PO 110	PO 117	PO 100	PO 98	PO 109	PO 104	Mean
PCB 129	3.9	15.7	11.4	35.5	3.8	23.7	29.0	25.3	5.0	12.2	16.5
PCB 130	378.0	565.5	399.5	691.5	377.1	662.9	1008.8	795.8	505.4	768.6	615.3
PCB 138 /160	2068.7	2210.9	2433.7	3742.0	1858.1	3614.4	5659.2	4761.7	1454.0	2956.9	3076.0
PCB 146	381.6	370.8	890.8	562.1	333.2	422.3	832.6	848.7	419.4	572.3	563.4
PCB 149/123	452.5	453.2	1050.7	666.4	312.2	682.6	1090.8	957.6	358.6	665.0	669.0
PCB 151	225.5	369.8	546.8	621.3	263.2	743.2	1032.8	749.3	340.5	584.1	547.6
PCB 153/132	2554.6	2748.5	5192.9	5002.8	1977.3	6088.0	8543.0	6658.5	2506.0	4677.2	4594.9
PCB 158	194.0	236.3	371.8	494.6	148.9	491.8	738.9	722.6	180.7	459.5	403.9
PCB 167	125.3	140.4	168.6	117.9	80.6	85.6	ND	137.9	92.1	ND	94.8
PCB 170/190	356.2	450.6	688.2	682.5	269.2	585.0	1097.4	927.0	351.1	631.5	603.9
PCB 171/202	ND	ND	ND	86.5	ND	68.7	97.8	132.1	155.0	101.0	64.1
PCB 172	ND	69.2	ND	ND	ND	ND	ND	ND	56.4	ND	12.6
PCB 174	96.4	123.8	215.0	177.3	84.4	166.8	306.4	264.8	102.0	194.8	173.2
PCB 175	ND	73.0	64.8	111.5	ND	97.0	156.1	ND	ND	82.6	58.5
PCB 176/137	104.0	200.2	283.3	ND	132.2	113.9	ND	162.1	174.2	ND	117.0
PCB 177	110.0	147.7	323.7	240.8	107.6	212.2	375.0	294.7	139.7	ND	195.1
PCB 178	113.8	126.9	247.7	234.5	90.6	172.7	333.7	272.0	121.0	202.0	191.5
PCB 180	363.5	453.6	1587.6	1429.2	551.6	1329.1	2458.5	2154.8	770.6	1449.0	1254.7
PCB 183	ND	ND	478.2	ND	ND	ND	ND	ND	847.4	ND	132.6
PCB 185	64.9	88.3	76.9	76.7	51.6	63.7	119.0	99.7	60.4	82.0	78.3
PCB 187	641.5	721.6	1576.5	1240.9	485.5	1108.9	2112.5	1756.9	695.7	1158.1	1149.8
PCB 189	21.9	28.1	38.2	38.2	ND	28.1	58.1	53.1	19.3	34.8	32.0
PCB 191	ND	ND	37.1	ND	ND	ND	ND	ND	ND	ND	3.7
PCB 193	80.3	109.2	157.1	143.2	75.8	126.0	234.6	205.7	106.5	146.3	138.5
PCB 194	195.4	218.6	314.1	343.8	114.6	238.3	517.8	482.8	183.3	340.7	295.0
PCB 195/208	194.5	207.6	182.9	295.3	115.5	168.5	373.6	328.1	188.5	279.1	233.3
PCB 197	37.4	47.3	21.0	64.9	25.6	40.5	89.1	71.7	36.3	60.0	49.4
PCB 199	218.0	252.6	371.0	414.8	147.9	284.8	614.4	536.7	209.9	383.4	343.4
PCB 200	18.9	16.2	35.3	27.6	14.1	13.2	52.9	39.6	18.0	31.8	26.8
PCB 201/157/173	109.9	123.8	ND	176.3	84.5	131.4	249.3	228.1	87.7	172.9	136.4
PCB 203/196	246.6	301.0	352.2	445.2	151.3	299.2	656.4	594.9	232.6	425.5	370.5
PCB 205	56.4	79.7	55.2	82.8	28.9	54.4	107.5	100.4	42.6	78.9	68.7
PCB 206	129.7	131.5	123.8	188.2	72.0	91.8	211.4	224.6	118.5	181.4	147.3
PCB 207	91.1	91.7	45.3	143.5	49.1	74.5	156.0	148.6	87.8	130.9	101.9
PCB 209	337.3	334.5	190.3	529.2	179.1	326.4	484.1	654.1	352.3	586.8	397.4
Dolphin ID	PO 101	PO 106	PO 94	PO 99	PO 110	PO 117	PO 100	PO 98	PO 109	PO 104	Mean

For each of the ten animals the combined congeners 153/132 and 138/160 made up slightly less than half of the total for the ten most prevalent PCB congeners. When congeners 26 and 180 were added to that list, the six congeners comprised about 70% of the total for the ten most prevalent PCB residues in each animal (Fig. 1). This was expected, as 26, 153, 138 and 180 are the most common congeners in the biota. To assess the potential toxicity of PCB mixtures found in these animals, toxic equivalencies (TEQs) were calculated for each congener, where possible, by multiplying toxic equivalency factors (TEFs; determined using rodents) for each congener times the congener concentration. These determine the toxicity of each congener relative to toxicity of the same concentration of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD). The contribution of mono- and di-ortho coplanar PCB congeners to the total TEQ of each animal showed that PCBs in these animals have a cumulative TEQ between 0.35 and 0.7 ppb of TCDD (Fig. 2). However, when analyses of the non-ortho coplanar PCBs were completed on five animals, the data showed that some of the non-ortho congeners made a large contribution to the total TEQ even though the residue levels were very small compared with total PCBs (Fig. 3). The non-ortho PCB congeners 77, 126 and 169 were responsible for significant increases in total TEQ, particularly congener 126. PCB residues of dolphin PO98 went from a TEQ of 0.70 without non-orthos to 2.53 when non-ortho PCBs were included in the calculation. Dolphin PO1 10, a 15 year old male with a PCB TEQ of 0.36 without the non-ortho congeners, exhibited a TEQ almost twice that value, 0.64, when the non-orthos were considered. While three animals' PCB values approximately doubled, two others, P098 and P0100, showed a three- to four-fold increase in the TEQ when non-ortho congeners were considered. Although toxic chemical effects may differ dramatically between species, it is possible that TEQ values from data determined on rodents may be extrapolated to considerations of PCB toxic effects in marine mammals. The expense of determining non-ortho PCBs precluded determining these values for all of the animals.

Environmental exposure of animals to organic pollutants, many of which may alter endocrine function (Roman et al. 1998), is associated with onset of a number of adverse physiological responses. Of these, the majority are a function of the agonist/antagonist actions of chemicals on receptor-mediated gene expression, and include reproductive abnormalities and decreased fertility of malt offspring (You et al. 1998; Roman et al. 1998), abnormalities of somatic development (Abbott et al. 1994), and decreased immune system function (Hardin et al. 1992). Exposure of pregnant females to OCs such as the PCBs and related chemicals may cause prenatal mortality in a variety of animals (Olson and McGarrigle 1992). Developmental abnormalities may occur at lower OC concentrations than those causing prenatal mortality (Mably et al. 1992). The LD_{50} of TCDD in developing rainbow trout fry, 40 pg/kg egg weight, is approximately 25-fold lower than the LD_{50} for juvenile trout (Walker and Peterson 1991). In a separate study, embryos from trout eggs into which TCDD was introduced at 385 pg/g of egg weight exhibited a series of craniofacial abnormalities and vascular defects directly correlated with TCDD levels (Homung et al. 1999). Prenatal exposure of rat pups to very low levels of TCDD from a single dose to the pregnant female resulted in body burdens of 18.1 pg/g on gestational day 16 and caused reduced fertility in subsequent generations (Hurst et al. 1998). In this study of fetal rats, overt effects of TCDD on pregnant females were not detected even though maternal blood levels were comparable to fetal levels. Similar data on PCB toxicity in developing mammals are not as extensive, but available TEQ values allow toxicity of some PCB congeners to be expressed as a function of comparable TCDD toxicity.

Reddy et al. (1998) reported that blood and blubber levels of PCBs in Atlantic bottlenose dolphins were correlated. The data of Hurst et al. (1998) indicated that maternal blood levels of OC in rats were approximately equivalent to fetal levels. Assuming that terrestrial mammal data can be extrapolated to dolphins, the data of Reddy and Hurst would suggest that maternal blubber levels of OC are likely to be correlated with maternal blood levels of OC, and that maternal OC blood levels may be correlated with fetal OC blood levels. Thus, it is possible

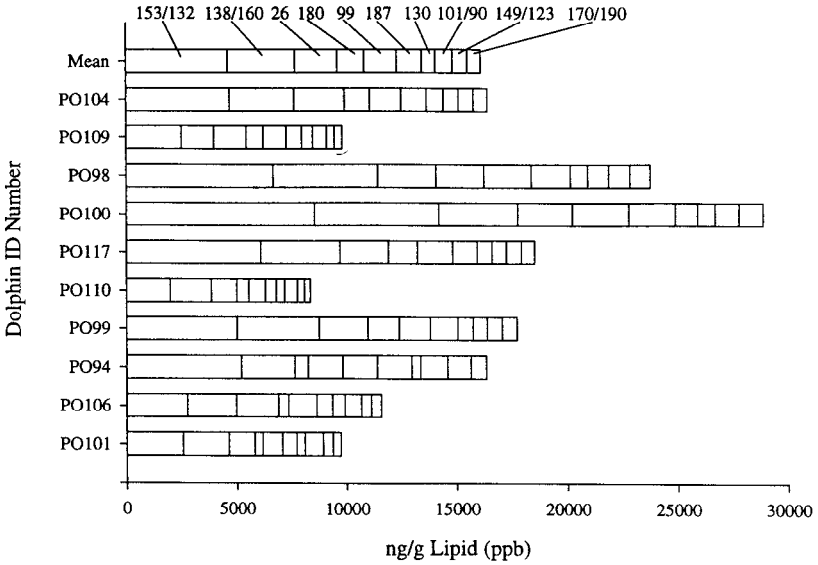

Figure 1. A compilation of the ten PCB congeners most prevalent in the blubber samples from Matagorda Bay animals.

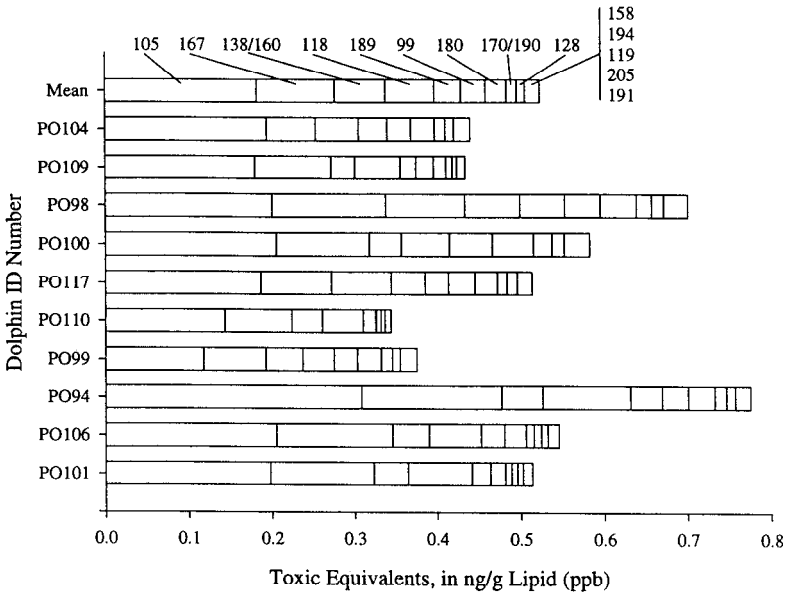

Figure 2. The total TEQs of PCBs per animal showing the contribution of each congener, mono- and di-ortho only, to the total TEQ given in ng/g blubber lipid.

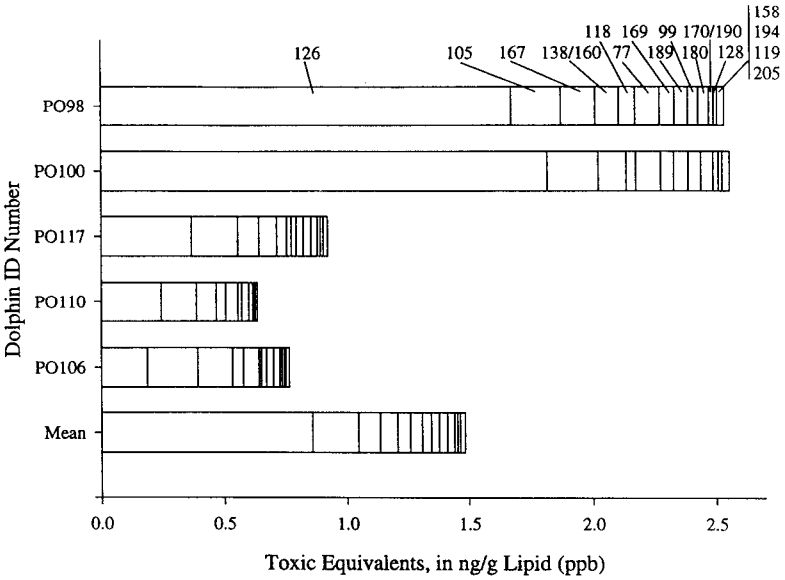

Figure 3. The total TEQs for five animals on which data were obtained to show mono-, di- and non-ortho congeners. TEQ data are given in ng/g blubber lipid for each animal.

that maternal blubber or blood levels of OC may be useful to approximate those found in embryonic tissues. This proposal would be extremely difficult to evaluate were it not for data comparing PCB residues in maternal blubber and pre-nursing dolphin calves. Pre-natal and nursing transfer of lipid soluble chemicals, including PCBs, from females to offspring has been previously reported (Tanabe et al. 1982) and data from healthy female dolphins and their stillborn, never nursed, calves suggest that offspring tissues contained PCB residues lower than, but dependent on, PCBs in maternal tissues *(S. Ridgway and M. Reddy,* ***unpublished data).*** These data combine to suggest that it might be possible to predict the toxic effects of PCBs on fetal development in dolphins by calculating TEQ values based on accumulated PCB residues in blubber. These analyses were completed on male dolphins because the PCB residues from female animals would be dependent on pregnancies and the duration of lactation. The values reported for males would be expected to be greater than those for adult females, but similar to those of females that had never calved.

The variety of animal responses to endocrine disruptive and AhR-interactive xenobiotics is thought to be related to the differential capacity of chemicals to bind cellular receptors in different species; however, these responses have not been defined for the vast majority of animals. Toxic equivalency factors (TEFs) are available for many of the more physiologically active PCB congeners known to bind the Ah receptor. The concentration of each congener times its TEF is expressed as a TEQ value, a measure of probable toxicity in laboratory animals relative to the effects of a specific concentration of TCDD given as a single dose. TEQ values do not consider synergisms or antagonisms between chemicals in complex mixtures and account only for AhR-mediated toxicity, thus the values may be difficult at best to correlate with toxic effects exhibited by animals chronically exposed to low levels of chemicals in complex mixtures. Further, the TEFs from which one calculates TEQs are based on doses administered to specific species, and may not reflect the toxicity of body burdens of chemicals bioaccumulated by a different species. The relationships of TEF and TEQ values

to the chronic effects exhibited by the majority of animals exposed to low levels of chemicals as complex mixtures have not been established. None-the-less, the calculation of TEQ values provides essentially the only mechanism by which one can approximate the cumulative effects of toxic chemicals *in vivo.*

In this study we determined the residue levels of a variety of PCBs, including non-, mono- and di-ortho coplanar congeners in blubber from male dolphins that stranded and died. Tissue levels of total PCBs ranged from 13.7 to 41.4 µg/g blubber lipid. While this is much less than the 90-1400 µg/g reported by Corsolini et al. (1995) for bottlenose and Risso's dolphins in the Mediterrean, it is still a high level of PCBs. Corsolini estimated TEQ values of 18.8 ng/g lipid for bottlenose dolphins from Italian coastal waters, a value they attributed largely to mono-ortho PCBs. This was about 6-fold higher than the highest TEQ for the Matagorda Bay animals. The highest TEQ of Matagorda Bay dolphins based on mono- and di-ortho PCBs was 0.81 ng/g of blubber lipid. When the non-ortho congeners were evaluated, a maximum total of 2.6 ng/g of blubber lipid was found, for a PCB total of 3.41 ng/g of blubber lipid. While less than the TEQ values estimated for Mediterrean dolphins, this is substantial considering that 0.018 ng/g of body weight was associated with developmental abnormalities and reduced fertility in fetal rats (Hurst et al. 1998). While there were significantly lower tissue residues of non-ortho congeners compared with mono- and di-ortho forms, the non-ortho PCBs made the greatest contribution to potential toxicity in the animals. The TEQ values reported here for PCBs bioaccumulated in male animals suggest that some of the Texas Gulf Coast T. *truncatus population* that stranded and died may have had blubber levels of PCBs that, had they occurred in pregnant females, would be consistent with the onset of fetal developmental anomalies, diminished immunological function, and/or decreased reproductive efficiency in terrestrial mammals.

A confounding factor in this study was effects of the weather. A hard freeze had occurred about two weeks prior to the mass stranding, resulting in the death of a major dolphin food species. Miller (1992) proposed that the absence of food fish in the stomachs and the emaciated conditions of stranded animals suggested that starvation and cold were significant contributing factors in their deaths. Korytko et al. (1999) showed that dogs fed 25 ppm Aroclor 1248 for 10 weeks exhibited the presence of new PCB congeners in the serum after 24 hr of fasting, that the congeners were present in serum as long as the animals were fasted, and that fasting resulted in a 293% increase in serum PCB concentrations. During the period of starvation described by Miller, dolphins would have metabolized stored fat reserves, and the subsequent release of lipid-soluble chemicals would be expected to increase circulating serum PCB levels, causing the pharmacologically effective PCB concentrations to be increased. There is no mechanism by which one can evaluate the potentially altered circulating level of PCBs in these stranded animals, nor can one speculate on the gene-expression disrupting effects of PCB levels transiently elevated by an extended period of food deprivation. There remains, however, the fact that blubber levels of PCBs in some of these animals were at concentrations expected to be consistent with the initiation of toxic effects.

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