High levels of polychlorinated biphenyls in tissues of Atlantic turtles stranded in the Canary Islands, Spain

J. Orós a,*, O.M. González-Díaz b, P. Monagas a

a Veterinary Faculty, University of Las Palmas de Gran Canaria, Trasmonteras s/n, 35413 Arucas (Las Palmas), Spain
b Department of Chemistry, University of Las Palmas de Gran Canaria, Campus Universitario de Tafira, 35017 Las Palmas de Gran Canaria, Spain

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A B S T R A C T

Polychlorinated biphenyls (PCBs 28, 31, 52, 101, 138, 153, 180, and 209) were measured in tissue samples (liver and fat) from 30 loggerhead turtles Caretta caretta, 1 green turtle Chelonia mydas, and 1 leatherback Dermochelys coriacea stranded on the coasts of the Canary Islands, trying to establish a possible relation between PCB concentrations and the lesions and causes of death. Tissues from these turtles contained higher levels of PCBs than those reported in turtles from other geographical regions. ∑PCB concentrations (1980 ± 5320 ng g⁻¹ wet wt.) in the liver of loggerheads were higher than in the adipose tissue (450 ± 1700 ng g⁻¹ wet wt.). Concentrations of PCB 209 in the liver (1200 ± 3120 ng g⁻¹ wet wt.) of loggerheads and in the liver (530 ng g⁻¹ wet wt.) and adipose tissue (500 ng g⁻¹ wet wt.) of the leatherback were remarkable. Frequencies of detection of PCB 209 in the liver (15.5%) and adipose tissue (31%) were also remarkable. Cachexia was detected in 7 turtles (22%) and septicemia was diagnosed in 10 turtles (31%). Statistically, a positive correlation was detected between ∑PCBs concentration and cachexia. Poor physical condition, cachexia and/or septicemia could explain the high levels of PCBs and tissue distribution. However, no histological lesions exclusively attributed to the acute effects of PCBs were described. The most prevalent histological lesions were ulcerative and purulent oesophagitis, purulent dermatitis, necrotizing enteritis, and granulomatous pneumonia. The bacteria most frequently isolated were Escherichia coli, Staphylococcus sp., and Aeromonas sp. Although immunosuppression as a result of PCBs pollution has been described previously, other factors in this study, such as incidental fishing, nutritional status, and exposure to different microorganisms, make it difficult to establish a clear association between PCB concentrations and causes of death.

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1. Introduction

Two families and seven species of sea turtles are currently recognized (Pritchard, 1997) and included in the red list of the World Conservation Union (IUCN, 2007). The family Dermochelyidae includes only the leatherback (Dermochelys coriacea). The family Cheloniidae includes the green turtle (Chelonia mydas), loggerhead (Caretta caretta), hawksbill (Eretmochelys imbricata), Kemp’s ridley (Lepidochelys kempi), olive ridley (Lepidochelys olivacea), and flatback turtles (Natator depressa). The most common species in the Canary Islands is the loggerhead turtle (Mateo et al., 1997). However, evidence of a decline in the population of turtles in the Canary Islands has been reported (López-Jurado and González, 1983; Blanco and González, 1992).

Diseases and causes of mortality among turtles stranded in the Canary Islands have been previously reported (Orós et al., 2004, 2005). However, data available for baseline levels of contaminants and effects on the turtle populations of the Canary Islands are scarce (Torrent et al., 2004).

Polychlorinated biphenyls (PCBs) have a particular significance because of their undesirable effects on environmental quality and animal health (Ahlborg et al., 1994). PCBs were manufactured from the 1930s to the 1970s for several industrial applications, such as liquid coolants for electrical transformers or as softeners in the production of plastics and as components of hydraulic fluids and lubricating oils. PCBs are able to bioaccumulate through the food chain and their effects have been reported on the immune, endocrine, and reproductive systems of different animal species (Fox, 2001). Although much was reported to date on PCBs concentrations in large predators, few studies have been dedicated to turtles. These studies have been focused on turtles from Long Island (Lake et al., 1994), Virginia (Rybinski et al., 1995), Scotland (Mckenzie et al., 1999), the Hawaiian Islands (Miao et al., 2001), the Baja California Peninsula (Gardner et al., 2003) and North Carolina (Kel ler et al., 2004, 2006). Studies focused on the Mediterranean Sea have a particular significance because of their number (Corsolini et al., 2000; Storelli and Marcotrigiano, 2000; Perugini et al., 2006; Storelli et al., 2007).
The aim of this study was to evaluate the presence and patterns of eight PCB congeners (28, 31, 52, 101, 138, 153, 180, and 209) in tissue samples (liver and fat) from 32 turtles stranded on the coasts of the Canary Islands between August 2002 and November 2005. We also tried to determine a possible relation between the PCB concentrations and the lesions and causes of death using the Spearman’s rho correlation method to calculate the correlation between ∑PCB concentrations in both tissues and physical conditions such as cachexia and septicaemia.

2. Materials and methods

2.1. Turtles

Between August 2002 and November 2005, 32 turtles that got stranded on the coasts of four islands belonging to the Canary Islands (Gran Canaria (n = 25; 78.1%), Tenerife (n = 3; 9.4%), Fuerteventura (n = 2; 6.2%), and El Hierro (n = 2; 6.2%)) were submitted for necropsy to the Veterinary Faculty, University of Las Palmas de Gran Canaria (ULPGC). Some of them had been previously submitted to the Tafira Wildlife Rehabilitation Center (TWRC) for health evaluation, medical management, and possible rehabilitation.

Species identifications were made according to Frick (1996). Turtles belonging to three different species were examined: 30 loggerheads, Caretta caretta (93.7%), 1 green turtle, Chelonia mydas (3.1%), and 1 leatherback, Dermochelys coriacea (3.1%). Species, sex, age group, and biometrics data are shown in Table 1. Age group was determined on the basis of straight carapace length (SCL) (Bjorndal et al., 2001; Seminoff et al., 2004) and sexual maturity (estimated from the appearance of their gonads).

2.2. Pathological and microbiological studies

Necropsies were performed at the Veterinary Faculty (ULPGC) within the first 12 h after death. The gross postmortem examinations were carried out using the procedures previously described (Wolke and George, 1981; Orós and Torrent, 2001). Gross lesions were recorded and tissue samples from all major organs were fixed in 10% neutral buffered formalin, embedded in paraffin, sectioned at 5 μm for light microscopy and stained with haematoxylin and eosin. Special stains used on selected cases included Gram stain for bacteria, Ziehl-Neelsen stain for acid-fast organisms, periodic acid-Schiff stain for protozoa and fungal hyphae, Grocott’s methenamine silver nitrate for fungi, and von Kossa stain for calcium (Bancroft and Stevens, 1996). For the microbiological studies, samples were taken from gross lesions and cultured on a variety of selective and non-selective media, including blood agar (Oxoid), MacConkey agar (Oxoid), Baird Parker agar (Oxoid) for staphylococci, and Sabouraud Dextrose agar (Oxoid) for fungi and yeasts. All cultures were incubated at 25 °C aerobically. Once a pure growth was obtained, bacteria were identified based on the biochemical profile (API 20 E, API 20 NE, and API 20 STAPH, BioMérieux).

2.3. Samples collection for PCB analysis

Tissues samples (liver and coelomic fat) were collected during necropsy. After collection, the samples were wrapped in an aluminium foil and stored at −20 °C until analysis. Prior to analysis, tissue samples were homogenized using a commercial blender.

2.4. PCBs analysis

Polychlorinated biphenyls (IUPAC Nos. 28, 31, 52, 101, 138, 153, 180, and 209) were analysed according to the method described by Tanabe et al. (1994). The validity of analytical methods was confirmed with Standard Reference Materials (CARP-2: ground whole carp, Cyprinus carpio) obtained from the National Research Council of Canada. Precision and accuracy are reported in Table 2.

Briefly, aliquots (4–7 g) of the homogenized samples were ground with anhydrous sodium sulphate in a mortar, and extracted using Soxhlet apparatus for 6 h with 300 mL of diethyl ether:hexane (3:1) solvent mixture. Extracts were concentrated in volume to 10 mL in Kuderna-Danish, and the aliquots (2 mL) were transferred to a glass column packed with 20 g of Florisil and dried by passing through nitrogen gas. Organochlorines adsorbed on Florisil were eluted with 150 mL of 20% hexane-washed water in acetonitrile and transferred to a separatory funnel containing 600 mL of hexane-washed water and 100 mL of hexane. After partitioning, the hexane layer was concentrated, cleaned up with sulphuric acid, and passed through a 12 g Florisil-packed glass column for separation.

Final determination of PCBs was carried out using a Varian 3600 gas chromatograph fitted with an electron capture detector (GC-ECD). In all the analyses a fused-silica capillary column Supelco (length = 30 m, inner diameter 0.53 mm and film thickness 0.50 μm) was used. The column oven temperature was programmed from 60 to 160 °C, held for 10 min, and then increased to 260 °C at a rate of 2 °C/min and held for 20 min. Injector and detector temperatures were set at 260 and 280 °C, respectively. Nitrogen was used as a carrier gas at 63.3 mL/min. The PCB patron used as an internal standard was the PCB-Mix 12, Iso-octane (Lab. Dr. Ehrenstorfer). Quantification interval used for PCBs was from 1 ng g⁻¹ (instrumental detection limit) to 50000 ng g⁻¹ (optimum linear limit). Concentrations of PCBs, means of four measurements, are presented as ng g⁻¹ on a wet weight basis.

2.5. Statistical analysis

Mann–Whitney U test was conducted to determine whether the difference in the levels of PCBs were related to the tissues. Spearman’s rho correlation method was used to calculate the

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Table 1

<table>
<thead>
<tr>
<th>Species</th>
<th>Sex</th>
<th>Age group</th>
<th>Weight (kg)</th>
<th>SCL (cm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Caretta caretta (n = 30)</td>
<td>Female</td>
<td>Pelagic juvenile (n = 12; 40%)</td>
<td>11.5 ± 8.3</td>
<td>41 ± 11.7</td>
</tr>
<tr>
<td></td>
<td>Male</td>
<td>Juvenile (n = 18; 60%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chelonia mydas (n = 1)</td>
<td>Female</td>
<td>Juvenile</td>
<td>21</td>
<td>52</td>
</tr>
<tr>
<td>Dermochelys coriacea (n = 1)</td>
<td>Female</td>
<td>Adult</td>
<td>231.5</td>
<td>nm</td>
</tr>
</tbody>
</table>

SCL: straight carapace length.
n: number of turtles.
nm: not measured.

Table 2

<table>
<thead>
<tr>
<th>PCBs</th>
<th>CARP-2</th>
<th>Certified</th>
<th>Found</th>
</tr>
</thead>
<tbody>
<tr>
<td>28</td>
<td>34 ± 4.0</td>
<td>31 ± 3.1</td>
<td></td>
</tr>
<tr>
<td>52</td>
<td>138 ± 43</td>
<td>119.7 ± 13.9</td>
<td></td>
</tr>
<tr>
<td>101</td>
<td>145 ± 48</td>
<td>150.1 ± 24.1</td>
<td></td>
</tr>
<tr>
<td>138</td>
<td>103 ± 30</td>
<td>99.5 ± 18.7</td>
<td></td>
</tr>
<tr>
<td>153</td>
<td>105 ± 22</td>
<td>116 ± 16.6</td>
<td></td>
</tr>
<tr>
<td>180</td>
<td>53.3 ± 13.0</td>
<td>59.2 ± 10.7</td>
<td></td>
</tr>
<tr>
<td>209</td>
<td>4.6 ± 2.0</td>
<td>4.8 ± 3.6</td>
<td></td>
</tr>
</tbody>
</table>

a The concentrations are given in ng g⁻¹ wet wt.
b Number de replicates is 4.
correlation between $\Sigma$PCB concentrations in both tissues and physical conditions such as cachexia and septicaemia. According to the necropsy reports, values of 0, 1, and 2 were given to turtles with absence of cachexia, mild cachexia, and severe cachexia, respectively. Values of 0, 1, and 2 were also given to turtles with absence of infections, infection in one organ, and severe septicaemia.

3. Results

3.1. Gross pathology, histopathology, and microbiology

The pathological study revealed that 23 turtles (72%) died from lesions associated with human activities such as boat-strike injuries ($n=4$; 12.5%), entanglement in derelict fishing nets ($n=7$; 22%), and ingestion of hooks and monofilament lines ($n=12$; 37%). Only 9 turtles (28%) died from spontaneous diseases, including pulmonary emphysema, ulcerative keratoconjunctivitis, purulent adenitis of the salt glands and purulent cloacitis. Cachexia was detected in 7 turtles (22%). Septicaemia was diagnosed in 10 turtles (31%).

The histological lesions observed in the loggerheads included severe necrotic hepatitis ($n=4$; 13%), severe multifocal granulomatous hepatitis ($n=4$; 13%), severe ulcerative and purulent oesophagitis ($n=10$; 33%), mild purulent gastritis ($n=2$; 6.7%), mild to severe necrotizing enteritis ($n=5$; 16.7%), severe purulent cloacitis ($n=1$; 3%), mild to severe granulomatous pneumonia ($n=5$; 16.7%), severe pulmonary emphysema ($n=1$; 3%), severe necrotic splenitis ($n=1$; 3%), severe ulcerative and purulent keratoconjunctivitis ($n=3$; 10%), severe purulent adenitis of the salt glands ($n=4$; 13%) and severe purulent dermatitis ($n=9$; 30%). Bacteria isolated from the tissue lesions included Escherichia coli ($n=5$; 16.7%), Proteus sp. ($n=3$; 10%), Vibrio alginolyticus ($n=2$; 6.7%), Pseudomonas sp. ($n=2$; 6.7%), Streptococcus sp. ($n=2$; 6.7%), Citrobacter freundii ($n=2$; 6.7%), Aeromonas sp. ($n=3$; 10%), Staphylococcus sp. ($n=4$; 13%) and Klebsiella sp. ($n=2$; 6.7%).

Histological lesions observed in the specimen of C. mydas included severe multifocal granulomatous nephritis associated to Candida guillermondii infection and severe oedema of the lamina propria and intestinal submucosa. Histological lesions observed in the specimen of D. coriacea included severe multifocal necrotic nephritis, severe granulomatous pneumonia and severe necrotizing enteritis, and all these lesions were associated with Serratia marcescens infection.

3.2. PCBs

PCB concentrations measured in the two tissues of the three species of turtles analysed are shown in Table 3.

Loggerheads showed the highest hepatic $\Sigma$PCB (IUPAC Nos. 28, 52, 101, 138, 153, 180) mean concentration ($1980 \pm 5320$ ng g$^{-1}$ wet wt.,) followed by the only specimen of D. coriacea analysed ($445$ ng g$^{-1}$ wet wt.,) $\Sigma$PCB concentration in the liver of the only specimen of C. mydas analysed was lower ($116$ ng g$^{-1}$ wet wt.,) than those detected in loggerheads and leatherback turtles. Loggerheads also showed the highest $\Sigma$PCB mean concentration in the adipose tissue ($450 \pm 1700$ ng g$^{-1}$ wet wt.,).

Loggerheads showed the highest hepatic PCB 209 mean concentration ($1200 \pm 3120$ ng g$^{-1}$ wet wt.,) followed by the only specimen of D. coriacea analysed ($530$ ng g$^{-1}$ wet wt.,). However, the leatherback showed the highest PCB 209 concentration in the adipose tissue ($500$ ng g$^{-1}$ wet wt.,). Six loggerhead turtles had individual PCB concentrations higher than $2000$ ng g$^{-1}$, and seven turtles showed individual PCB concentrations higher than $900$ ng g$^{-1}$. All these turtles showed severe septicaemia and/or severe cachexia.

As revealed by statistical analysis, turtles showed significant higher PCBs 28, 31, 153, PCB 209 and $\Sigma$PCBs concentrations in the liver than in the adipose tissue. However, no significant differences between concentrations in both tissues were detected for PCB 138 and PCB 180.

The congener most frequently detected in the liver was PCB 180 (46.8%), followed by PCB 153 (31.2%), PCBs 28, 31 (25%), PCB 52 (25%), PCB 209 (15.6%), PCB 138 (15.6%), and PCB 101 (12.5%). The congeners most frequently detected in the adipose tissue were PCB 180 (50%) and PCB 153 (50%), followed by PCBs 28, 31 (34.4%), PCB 52 (34.4%), PCB 209 (31.2%), PCB 138 (28.1%), and PCB 101 (18.7%).

PCB profiles in the C. caretta tissues were dominated by the higher chlorinated congeners (Fig. 1). Hexachlorobiphenyls (PCBs 138 and 153) were predominant and accounted for 43.6% of the total PCBs, followed by decachlorobiphenyls (PCB 209) (33%) and heptachlorobiphenyls (PCB 180) (18%). Tetrachlorobiphenyls (PCB 52) accounted for 3% of the total PCBs, and trichlorobiphenyls (PCBs 28 and 31) accounted for 1.3% of the total residue. Concerning individual congeners, the most abundant was PCB 209 (33%) followed by PCB 153 (29%), PCB 180 (18%), and PCB 138 (15%). The isomer pattern of PCBs in C. caretta was different in the liver and the fat. In the liver, the most abundant was PCB 209 (37.6%), followed by PCB 153 (29%), PCB 138 (14%) and PCB 180 (14%). In the fat, the most abundant was PCB 180 (47%), followed by PCB 153 (28.6%), PCB 138 (18%) and PCB 209 (3%).

![Fig. 1. Fingerprints of liver and fat of the three species of sea turtles analysed.](image-url)
Regarding PCB profiles in the only specimen of C. mydas analysed, the most abundant was PCB 180 (73%) followed by PCB 153 (22%) and PCB 52 (3%). Although PCB 180 was the most abundant in both tissues, this congener was the only PCB detected in the liver of this turtle.

Regarding PCB profiles in the only specimen of D. coriacea analysed, the most abundant was PCB 209 (66%) followed by PCB 153 (20%) and PCB 180 (7%). The isomer pattern of PCBs in this turtle was slightly different in the liver and the fat. In the liver, the most abundant was PCB 209 (54%) followed by PCB 153 (26%) and PCB 180 (12%). In the fat, the most abundant was PCB 209 (87%), followed by PCB 153 (10%), and PCB 52 (2%).

Hepatic \( \Sigma \) PCBs concentrations were positively correlated with cachexia \((r=0.250, P=0.04)\). No significant correlation was observed between \( \Sigma \) PCBs concentrations and septicaemia.

4. Discussion

The present study represents the first data of PCB levels for any turtle species from the Canary Islands. The turtle tissues analysed in the current investigation contained high levels of PCBs compared to concentrations reported in turtles collected in other locations around the world (Table 4) (McKim and Johnson, 1983; Lake et al., 1994; Rybitski et al., 1995; Mckenzie et al., 1999; Corsolini et al., 2000; Storelli and Marcorigiano, 2000; Gardner et al., 2003; Keller et al., 2004; Perugini et al., 2006; Storelli et al., 2007).

The high degree of variation of concentrations of PCBs detected in our study may be attributed to the different exposure of individual turtles to pollutants and to the physical condition of the animals. Sea turtles visit different areas with different degree of contamination during migration, resulting in differences in exposure for each animal (Ziswiler, 1986). According to recent studies, loggerhead turtles stranded on the coasts of the Canary Islands come from two different migration routes: from the Western Atlantic arriving to the Canary Islands by the Gulf Stream (Pérez-Jiménez, 1997), and from Cape Verde (Dellinger, 2006). In addition, turtles included in this study varied widely in physical condition, ranging from turtles that had been killed incidentally through human interaction to turtles that showed severe emaciation and septicaemia after prolonged illness.

Six loggerhead turtles had individual PCB concentrations higher than the quantification limit of 2000 ng g\(^{-1}\) established by the Food and Drug Administration (FDA). In addition, seven turtles showed individual PCB concentrations higher than 900 ng g\(^{-1}\). All these turtles showed severe septicaemia and/or severe cachexia. Statistically, a positive correlation was detected between \( \Sigma \) PCBs concentration and cachexia. Although sea turtles are long-living high trophic organisms that would be expected to contain high concentrations of PCBs (Lake et al., 1994), we think the poor physical condition of these animals could be the reason for these high PCB levels. It has been described that in cetaceans many diseases affect metabolic centres and thus the capacity to metabolise or excrete pollutants may be affected, questioning whether PCB concentrations in the tissues of these specimens are representative of normal conditions (Aguilar et al., 1999).

<table>
<thead>
<tr>
<th>Species</th>
<th>Locality</th>
<th>Tissue (n)</th>
<th>PCB 180</th>
<th>PCB 209</th>
<th>PCB 218</th>
<th>PCB 28</th>
<th>PCB 52</th>
<th>PCB 101</th>
<th>PCB 118</th>
<th>PCB 138</th>
<th>PCB 153</th>
<th>PCB 155</th>
<th>PCB 180</th>
<th>Reference</th>
</tr>
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<tbody>
<tr>
<td>L. kempi</td>
<td>Long Island</td>
<td>Liver (22)</td>
<td>512.6</td>
<td>148.9</td>
<td>98.3</td>
<td>114.94</td>
<td>170.04</td>
<td>53.62</td>
<td>Lake et al. (1994)</td>
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<tr>
<td>C. caretta</td>
<td>Virginia and North Carolina</td>
<td>Liver (18)</td>
<td>7.46–514</td>
<td>BDL-59.5</td>
<td>2.04–152</td>
<td>102.7</td>
<td>Rybtski et al. (1995)</td>
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<tr>
<td>L. kempi</td>
<td>Mediterranean Sea</td>
<td>Liver (4)</td>
<td>50–102</td>
<td>&lt;0.5–1.6</td>
<td>&lt;0.5–1.6</td>
<td>&lt;0.5–3.4</td>
<td>3.3–8</td>
<td>12–24</td>
<td>13–29</td>
<td>7.3–18</td>
<td>Mckenzie et al. (1999)</td>
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<tr>
<td>C. caretta</td>
<td>Mediterranean Sea</td>
<td>Liver (4)</td>
<td>77.5–893</td>
<td>&lt;4.8–8.0</td>
<td>8.6–37</td>
<td>8–32</td>
<td>54–71</td>
<td>132–169</td>
<td>229–261</td>
<td>73–154</td>
<td>C. mydas</td>
<td>Mediterranean Sea</td>
<td>Liver (9)</td>
<td>&lt;11–77</td>
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<tr>
<td>C. caretta</td>
<td>Adriatic Sea</td>
<td>Liver (11)</td>
<td>39–261</td>
<td>&lt;2.2–3.7</td>
<td>13.25</td>
<td>5.7–33</td>
<td>&lt;2.2–27</td>
<td>&lt;2.2–28</td>
<td>&lt;2.2–3.2</td>
<td>&lt;2.2–13</td>
<td>C. caretta</td>
<td>Adriatic Sea</td>
<td>Liver (4)</td>
<td>4.5–11.0</td>
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<td>3.22–6.54</td>
<td>0.21–0.76</td>
<td>A,B</td>
<td>BDL-148</td>
<td>Lake et al. (2004)</td>
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<tr>
<td>L. olivacea</td>
<td>Baja California peninsula</td>
<td>Liver (7)</td>
<td>334</td>
<td>BDL-49.5</td>
<td>Gardner et al. (2003)</td>
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<tr>
<td>C. caretta</td>
<td>Adriatic Sea</td>
<td>Liver (11)</td>
<td>459.7</td>
<td>2.9–1472.1</td>
<td>6.85–2974.9</td>
<td>6.85–2974.9</td>
<td>Storelli et al. (2007)</td>
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</tbody>
</table>

BDL: below detection limit.
A Value expressed as ng kg\(^{-1}\) lipid weight.
B Value expressed as ng g\(^{-1}\) lipid weight.
\(a\) Young turtles.
\(b\) Adult turtles.
According to other authors, the pattern of tissue distribution of PCBs reported in turtles and other marine species is as follows: adipose tissue > liver > kidney > muscle (Tanabe et al., 1983; Martineau et al., 1987; Colborn and Clement, 1992; Rybiski et al., 1995; Mckenzie et al., 1999). This is attributed to the highest total lipid content and triglycerides for the adipose tissues and the strong correlations between these and their burden of lipophilic pollutants (Cockcroft et al., 1989). However, we detected higher \( \Sigma \)PCBs concentrations in the liver than in the adipose tissue (Fig. 2). Thirteen turtles showed higher PCB levels in the liver than in the fat. All these turtles showed severe cachexia and/or septicaemia. According to different studies, animals with abundant fat deposits can accumulate and tolerate higher concentrations of toxic chemicals because lipophilic pollutants are stored in the adipose tissue and are less available to target organs and receptors. In contrast, emaciated animals which have mobilised their lipid stores may be more susceptible to toxic effects as a result of remobilisation of the pollutants, resulting in higher concentrations of PCBs in the remaining tissues such as the liver (Bernhoff and Skaare, 1994). Thus, the cachexia observed in our turtles may indicate a previous remobilisation of the fat and the pollutants, and could explain the high concentration of PCBs in the liver. In addition, the septicaemic condition reported in these turtles could be caused by a possible immunosuppression as one of the most distinguished effects of organochlorine compounds.

In our study, PCB profiles in the \( C. \ caretta \) tissues were dominated by the higher chlorinated congeners (Fig. 1). Hexachlorobiphenyls and heptaclorobiphenyls are predominant in turtles and marine mammals (Muir et al., 1988; Rybiski et al., 1995; Colborn and Smolen, 1996; Alam and Brim, 2000; Corsolini et al., 2000; Storrelli and Macorotigiano, 2000; Miao et al., 2001; Perugini et al., 2006; Storrelli et al., 2007). However, Gardner et al. (2003) reported in \( C. \ mydas \) a profile dominated by lower-chlorinated congeners. Differences in PCB patterns in turtles may be attributed to differences in the congener compositions of environmental media among regions, dietary differences or differences in the abilities of the various species and populations to metabolize PCBs (Gardner et al., 2003).

In our study, the concentrations and frequencies of detection of PCB 209 were remarkable. Usually, published reports do not include this congener. Gardner et al. (2003) detected very low concentrations of PCB 209 following a decreasing order in muscle, liver and adipose tissue of turtles. Storrelli et al. (2007) reported in a study on 19 loggerhead turtles that PCB 209 accounted only for 0.1–1.6% of total PCBs.

One of our objectives was to establish a possible relationship between PCBs concentrations and the causes of death of each turtle. It was difficult to assess the impact of organochlorine compounds in the turtles of our study because the most frequent causes of death (72%) were attributed to fishing activity. In addition, few toxicological studies have been published regarding organochlorines in reptiles and no safe concentrations have been established. It is remarkable that almost all turtles with severe septicaemia showed very high levels of PCBs. Immunosupression as result of PCBs pollution has been previously described (Keller et al., 2004, 2006). However, in our study the presence of other factors different to PCBs concentrations, such as incidental fishing, nutritional status, and exposition to different micro-organisms, make it difficult to establish a clear association between PCB concentrations and causes of death. According to the histopathological study no lesions exclusively attributed to acute effects of PCBs were described. All the histological lesions were attributed to bacterial and/or fungal infections mainly associated with gross lesions caused by fishing activity. However, chronic effects of PCBs are much difficult to determine.

While there was an insufficient number of samples to assess differences among species, qualitative evaluation could indicate the potential effects of poor physical condition differences among individuals of \( C. \ caretta \) and subsequent poor metabolic performance of detoxification systems. In addition, the detection of high levels of PCBs in these turtles makes it necessary to continue to monitor the levels of these chemicals in turtles from the Canary Islands to contribute to the protection of these currently endangered species.

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References


