The Biology of Sea Turtles

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11 Exposure to and Effects of Persistent Organic Pollutants

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11.1 BACKGROUND ON PERSISTENT ORGANIC POLLUTANTS

Man-made chemicals have enhanced our quality of life for centuries through improvements in healthcare, industrial efficiency, food production, and consequently increased economic profits. Other benefits include improved fire safety and simplification of daily activities (e.g., stain and stick repellant chemicals make cleaning household spills easier). However, some chemicals have consequences, especially those that persist for long periods as contaminants in the environment, preferentially accumulate in animal tissues (termed bioaccumulative), and have known toxicities. Chemical contaminants with these characteristics that are also organic in structure (consist of a carbon backbone) have been termed persistent organic pollutants (POPs).

Two historical events in the last century heightened awareness of the negative effects of POPs, which subsequently lead to environmental legislation. In 1962, Rachel Carson described the harmful effects of environment pollutants in wildlife in her book, "Silent Spring" (Carson, 1962). Her vivid imagery of a world devoid of bird song because of highly toxic organochlorine insecticides, like dichlorodiphenyltrichloroethane (DDT), prompted the United States and some other countries to ban certain POPs in the 1970s and 1980s. The second event took place in 2001, when an international treaty known as the United Nations Stockholm Convention on Persistent Organic Pollutants was signed. The Stockholm Convention originally named twelve chemicals (or chemical classes) as POPs, which are considered to be too persistent, too bioaccumulative,

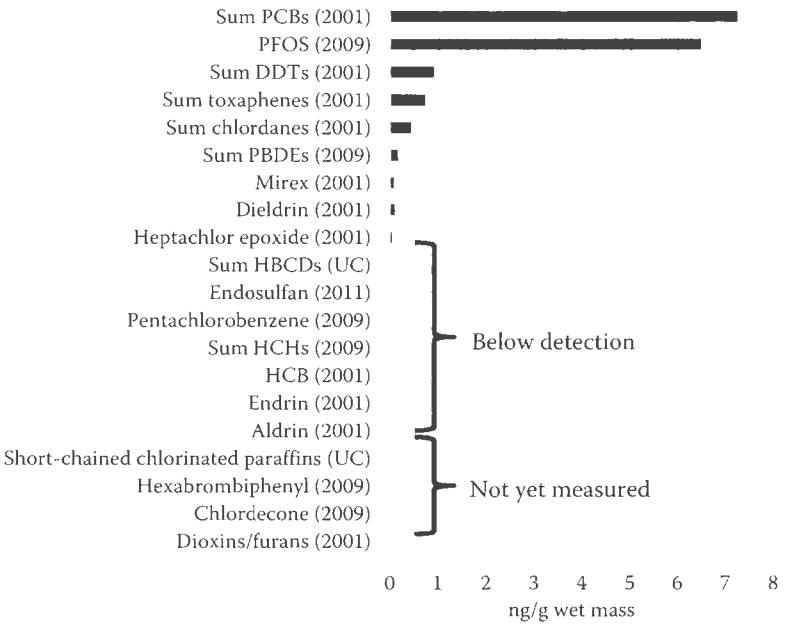

FIGURE 11.1 Relative concentrations of persistent organic pollutants (POPs) measured in loggerhead sea turtle blood components along the east coast of the United States. Arithmetic means were converted from Keller et al. (2004a, 2005a) and taken from Ragland et al. (2011) and O'Connell et al. (2010). The year the chemical class was listed on the Stockholm Convention is shown in parentheses along the y-axis; UC = under consideration for the Stockholm Convention. Of the chemicals indicated as "not yet measured," none have been measured in sea turtles except for dioxins and furans in green sea turtle blood from Australia (Hermanussen et al., 2006). The high ranking of toxaphenes may be misleading, because toxaphene data are available only from adult males that likely have higher concentrations relative to juveniles that were used for most other POP classes shown here. Chemical abbreviations are PCBs = polychlorinated biphenyls; PFOS = perfluorooctane sulfonate; DDTs = 4,4′-dichlorodiphenyltrichloroethane-related compounds; PBDEs = polybrominated diphenyl ethers; HBCDs = hexabromocyclododecanes; HCHs = hexachlorohexanes; and HCB = hexachlorobenzene.

and too toxic for continued widespread use; many of these chemicals were named by Carson four decades earlier. Figure 11.1 shows the so-called Dirty Dozen compounds listed in 2001 as well as recent additions to the Stockholm Convention's list of chemicals to eliminate or restrict from production and use or to reduce unintentional releases. Abbreviations for these compound classes are also provided in Figure 11.1.

More than 22,000 chemicals are listed on selected United States and Canadian chemical registries, which do not account for all chemicals in use or production. A recent assessment of their chemical structure and properties demonstrated that about 610 of them might be persistent and bioaccumulative, two of the three characteristics of POPs (Howard and Muir, 2010). The majority of these have not yet been discussed by the Stockholm Convention. Furthermore, only 47 of these chemicals are routinely monitored in the environment (Howard and Muir, 2010) and only 16 have been reported in the sea turtle literature (Figure 11.1).

11.2 INTRODUCTION TO EXPOSURE AND EFFECTS

Understanding the exposure and effects of POPs on sea turtles is important because societies care about conserving their populations. Moreover, POPs have contributed to population declines of several wildlife species (Fox, 2001; Guillette et al., 1994). For example, an American alligator

(*Alligator mississippiensis*) population inhabiting Lake Apopka in Florida declined by 85% within 3 years of a spill of a pesticide called Dicofol containing DDT (Guillette et al., 1994), and egg shell thinning caused by widespread DDT use contributed to sharp population declines of birds in the 1950s and 1960s (Fox, 2001). Adding to evidence of cause and effect, the populations began to recover after DDT was banned. While no studies have directly investigated the effects of environmental pollutants on sea turtles at the population level, many scientists suspect that contaminants contribute to health problems, disease prevalence, altered embryonic growth, mortality or reduced reproductive success of sea turtles (Aguirre et al., 1994; Herbst and Klein, 1995; Keller et al., 2004c; van de Merwe et al., 2010b). While a handful of studies have provided correlative evidence that chemical pollutants may affect the health, survival, or reproduction of sea turtles, much more research and a weight of evidence approach is needed to better understand the toxic effects and more importantly to provide resource managers information to determine mortality risk due to this threat. In fact, a committee of 35 sea turtle researchers named chemical pollution as a top global priority for future sea turtle research (Hamann et al., 2010). Furthermore, the National Oceanic and Atmospheric Administration convened a "*Sea Turtles and Contaminants Workshop*" in May 2010 to address the uncertainty surrounding this threat; the workshop report has been delayed in part because of the Deepwater Horizon Oil incident, which coincidentally occurred 2 weeks before the workshop.

While POPs are the topic of this chapter, it is important to note that POPs are only one class of environmental chemical pollutants. To put POPs into perspective, other contaminant classes include metals like mercury, lead, copper, cadmium, and zinc; organometallics, such as methylmercury and tributyltin; petroleum products of several types; oil dispersants; polycyclic aromatic hydrocarbons (PAHs); plastics; plasticizers; surfactants; current-use pesticides like organophosphates and carbamates; excess nitrogen loading from fertilizer; sewage and urban runoff; nanoparticles like fullerenes and quantum dots; pharmaceuticals including antibiotics, hormones, and antidepressants to name only a few; and even naturally produced toxic chemicals from harmful algal blooms. A few of these chemical classes have been assessed in sea turtles, particularly metals (see Pugh and Becker [2001] for a comprehensive bibliography prior to 2001 or more recent tissue-specific or species-specific reviews [Aguirre et al., 2006; D'Ilio et al., 2011; Guirlet et al., 2008; Perrault et al., 2011]). Additionally, antibiotic resistance has been detected in bacterial swabs from sea turtles, suggesting that environmental exposure to antibiotics is changing the microbial communities that sea turtles confront (Al-Bahry et al., 2009; Foti et al., 2009). Several studies have also investigated harmful algal toxins in sea turtles (Arthur et al., 2008; Harris et al., 2011; Pierce and Henry, 2008; Takahashi et al., 2008; Walsh et al., 2010). Many more studies are needed to understand the effects of not only POPs but all chemical classes in sea turtles.

11.3 SEA TURTLE EXPOSURE TO POPS

11.3.1 Global Account of Studies

Currently, the number of peer-reviewed papers attempting to measure POP concentrations in sea turtles is only 51 (Table 11.1; Figure 11.2). This is a meager number considering that there are seven species inhabiting wide ranges of habitat types globally from remote oceanic realms to highly contaminated urban harbors. In addition, most of these studies focus on one or two POP classes of chemicals and are spread over more than four decades (Table 11.1). For perspective, this number represents only 6% of the POP literature covering three categories of marine megafauna (sea turtles, seabirds, and marine mammals). Fortunately, the interest in this field has been increasing exponentially over four decades (Figure 11.2).

The first two published measurements of POP concentrations in sea turtle samples occurred in 1974. Interestingly, one came from an unlikely remote location, Ascension Island in the central South Atlantic Ocean, where Thompson (Thompson et al., 1974) sampled green turtle (*Chelonia mydas*)

TABLE 11.1
List of Published Studies on Persistent Organic Pollutant Concentrations in Sea Turtle Tissues

Published Study	Species	Tissue	Location	Years Sampled	Compound Class Measured
Hillestad et al. (1974)	Cc	Egg	South Carolina-Georgia	NR	OCPs, metals, radionuclide
Thompson et al. (1974)	Cm	Egg yolk	Ascension Island	1972	PCBs, OCPs
Clark and Krynitsky (1980)	Cc, Cm	Egg contents	Merritt Island, Florida	1976	PCBs, OCPs
McKim and Johnson (1983)	Cc, Cm	Liver, muscle	East Florida, USA	NR	PCBs, 4,4′-DDE
Clark and Krynitsky (1985)	Cc	Egg contents	Merritt Island, Florida	1979	OCPs
Davenport et al. (1990)	Dc	Blubber	Wales, United Kingdom	1988	PCBs, OCPs, metals
Aguirre et al. (1994)	Cm, Cc	Liver, adipose, kidney, egg shells, hatchling tissues	Hawaii	NR	PCBs, OCPs, OPs, carbamates, metals
Lake et al. (1994)	Cc, Lk	Body fat, liver	Long Island, New York	1980–1989	PCBs, OCPs
Rybitski et al. (1995)	Cc, Lk	Fat, liver, muscle, kidney	Virginia-North Carolina	1991–1992	PCBs, OCPs
Cobb and Wood (1997)	Cc	Egg contents, CAM	Cape Island, South Carolina	1993	PCBs
Godley et al. (1998)	Dc	Adipose	United Kingdom	1993–1996	PCBs, OCPs, PAHs, metals
Podreka et al. (1998)	Cm	Egg contents except shell membranes	Heron Island, Queensland, Australia	1995	4,4′-DDE
Mckenzie et al. (1999)	Cc, Dc, Cm	Egg, liver, adipose, hatchling	Cyprus, Greece, and Scotland	1993–1995	PCBs, OCPs
Alam and Brim (2000)	Cc	Egg contents, all stages	Northwest Florida	1992	PCBs, OCPs, PAHs, metals
Storelli and Marcotrigiano (2000)	Cc	Liver, kidney, muscle, heart, lung	Southern Adriatic Sea and Ionian Sea	1990–1991	PCBs, OCPs
Corsolini et al. (2000)	Cc	Adipose, liver, muscle	Northeast Italy, Adriatic Sea	1993	PCBs
Vetter et al. (2001)	Cm	Fat	Australia	1998	PCBs, OCPs, unknown Br compounds
Miao et al. (2001)	Cm	Liver, adipose	Oahu, Hawaii	1992–1993	PCBs
Gardner et al. (2003)	Cm, Lo, Cc	Adipose, liver, muscle, kidney	Baja California Peninsula	NR	PCBs, OCPs

Keller et al. (2004b)	Cc	Plasma, whole blood, RBC	Core Sound, North Carolina	1998–2001	PCBs, OCPs
Keller et al. (2004a)	Cc, Lk, Dc, Cm	Adipose, blood	North Carolina and Massachusetts	1999–2001	PCBs, OCPs
Keller et al. (2004c)	Cc	Adipose, blood	Core Sound, North Carolina	2000–2001	PCBs, OCPs
Keller et al. (2005b)	Cc, Lk	Plasma	North Carolina-Florida	2003	PFCs
Keller et al. (2005a)	Cc	Plasma	South Carolina-Florida	2003	PCBs, PBDEs
Deem et al. (2006)	Dc	Plasma	Gabon, Africa	2001–2002	PCBs, OCPs, metals
Perugini et al. (2006)	Cc	Liver, muscle, fat	Southern and central Adriatic Sea	2003–2004	PCBs, OCPs
Hermanussen et al. (2006)	Cm	Whole blood	Moreton Bay, Australia	2000	Dioxins, furans
Alava et al. (2006)	Cc	Egg yolk	Boca Raton and Sarasota, Florida	2002	PCBs, OCPs
Storelli et al. (2007)	Cc	Liver, kidney, lung, muscle	Southern Adriatic Sea and Ionian Sea	1999–2001	PCBs, DDTs
Innis et al. (2008)	Lk	Plasma, liver, kidney, fat, brain	Cape Cod, Massachusetts	2005	OCPs, metals
Hermanussen et al. (2008)	Cm, Ei, Nd	Muscle, liver, adipose, blood, plasma	Queensland, Australia	2004–2006	PBDEs
Monagas et al. (2008)	Cc	Fat, liver	Canary Islands	2003–2004	2,4′-DDTs
Orós et al. (2009)	Cc, Cm, Dc	Liver, fat	Canary Islands	2002–2005	PCBs
van de Merwe et al. (2009b)	Cm	Egg yolk + albumen	Heron Island, Queensland, Australia	1998	PCBs, OCPs, PBDEs
van de Merwe et al. (2009a)	Cm	Egg yolk + albumen	Malaysian markets	2006	PCBs, OCPs, PBDEs
Deem et al. (2009)	Cc	plasma	Georgia-Florida	2000–2004	PCBs, OCPs, metals
Richardson et al. (2010)	Cc, Cm, Lo	Liver	Baja California Peninsula	2001–2003	PCBs
Swarthout et al. (2010)	Lk, Cm	Whole blood	Gulf of Mexico and South Carolina-Florida	2001–2002	PCBs, OCPs, PBDEs
van de Merwe et al. (2010a)	Cm	Liver, muscle, kidney, whole blood	Queensland, Australia (rehab)	2006–2007	PCBs, OCPs, PBDEs, metals
van de Merwe et al. (2010b)	Cm	Maternal blood, egg contents, hatchling blood	Peninsular Malaysia	2004	PCBs, OCPs, PBDEs

(continued)

TABLE 11.1 (continued)
List of Published Studies on Persistent Organic Pollutant Concentrations in Sea Turtle Tissues

Published Study	Species	Tissue	Location	Years Sampled	Compound Class Measured
Guirlet et al. (2010)	Dc	Maternal blood, egg contents	French Guiana	2006	PCBs, OCPs
O'Connell et al. (2010)	Cc	Plasma or serum	Maryland-Florida	2000–2008	PFCs
Lazar et al. (2011)	Cc	Yellow fat	Eastern Adriatic Sea	2001–2002	PCBs, OCPs
Ragland et al. (2011)	Cc	Plasma	Cape Canaveral, Florida	2006–2007	PCBs, OCPs, PBDEs, HBCDs, pentachlorobenzene
Stewart et al. (2011)	Dc	Maternal blood, egg contents, fat, blubber	North Carolina, South Carolina, Florida	1999–2003	PCBs, OCPs, PBDEs
Alava et al. (2011)	Cc	Egg yolk	North Carolina and Florida	2002	PCBs, OCPs, PBDEs
Malarvannan et al. (2011)	Cm, Ei, Cc	Liver	Japan	1998–2006	PCBs, OCPs, PBDEs
Labrada-Martagon et al. (2011)	Cm	Plasma	Baja California Peninsula	2005 and 2007	OCPs, metals
Harris et al. (2011)	Dc	Serum	Central California and St. Croix, USVI	2005–2007	PCBs, OCPs, metals
Komoroske et al. (2011)	Cm	Plasma	San Diego Bay	2007–2009	OCPs, PBDEs, metals
Keller et al. (2012)	Cm, Ei, Dc, Cc, Lk	Plasma	North Carolina and eastern Florida	2006–2007	PFCs

Loggerhead (Cc), green (Cm), leatherback (Dc), Kemp's ridley (Lk), olive ridley (Lo), hawksbill (Ei), flatback (Nd) sea turtles, chorioallantoic membrane (CAM), red blood cells (RBCs), not reported (NR), organochlorine pesticides (OCPs), polychlorinated biphenyls (PCBs), organophosphate pesticides (OPs), polycyclic aromatic hydrocarbons (PAHs), perfluorinated contaminants (PFCs), polybrominated diphenylethers (PBDEs), hexabromocyclododecanes (HBCDs).

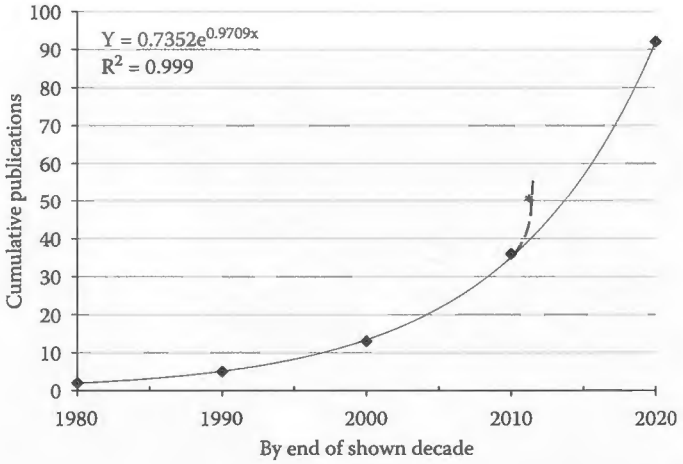

FIGURE 11.2 Cumulative number of peer-reviewed publications on persistent organic pollutant concentrations in sea turtles by decade. The exponential curve was fit through the data from the 1970s to 2010 to predict the number of publications by 2020. The asterisk is the current number of publications and the dotted line shows how the field's growth may exceed the predicted exponential trend.

eggs for PCBs and DDTs. This site has not been sampled since. The other study analyzed loggerhead sea turtle (*Caretta caretta*) eggs from South Carolina and Georgia (Hillestad et al., 1974), which has become one of the top sampled regions and species for POPs exposure assessment (Figure 11.3). This focus is not because the Southeastern United States is the most contaminated region or that the loggerhead is the species of most concern, but because the highest density of sea turtle biologists inhabits this region where loggerhead turtles are abundant.

Spatially, the 51 available studies span the globe providing POP baseline concentrations in sea turtles from many regions, but major data gaps still exist (Figure 11.3). While the list of regions sampled suggests wide coverage (e.g., from Hawaii to the Mediterranean Sea to Australia), the distribution shown in Figure 11.3 can be misleading. The compiled studies represent different tissue types sampled from different age classes during different decades, and they report on different POP classes or used different methods, which cannot be compared. For example, the loggerheads sampled from Florida Bay represent one study on juvenile turtle plasma for a newer contaminant class, the perfluorinated contaminants (PFCs) (O'Connell et al., 2010). Our knowledge is devoid of other POP classes, such as PCBs or pesticide concentrations, in any sea turtle species or age from this location (but a study is underway). Worst yet, the data from several of the pie charts shown in Figure 11.3 have limited usefulness because of low sample sizes or use of analytical methods that were not sensitive enough for the species or tissue chosen. For example, 18 green turtles have been analyzed for POPs from Hawaii (Aguirre et al., 1994; Miao et al., 2001), but only one of these studies used methods that could detect the target compounds; so, known baseline concentrations are available on only three turtles from this region. Additionally, plasma or serum samples from 53 loggerheads from South Carolina and Georgia, 18 Kemp's ridleys (*Lepidochelys kempii*) from Massachusetts, and 33 leatherbacks (*Dermochelys coriacea*) from California, St. Croix, and Gabon were wasted because of high detection limits of the chosen method (Deem et al., 2006, 2009; Harris et al., 2011; Innis et al., 2008). These examples teach a lesson; that it is important to choose analytical methods that will detect the contaminant range expected from the sampled species and tissue type to avoid wasting time, money, and valuable samples. Additionally, it is very important for analytical laboratories to use quality assurance and quality control practices, including analyzing field and procedural blanks and certified reference materials, using high quality authentic chemical standards plus internal standards, and participating in inter-laboratory comparison exercises.

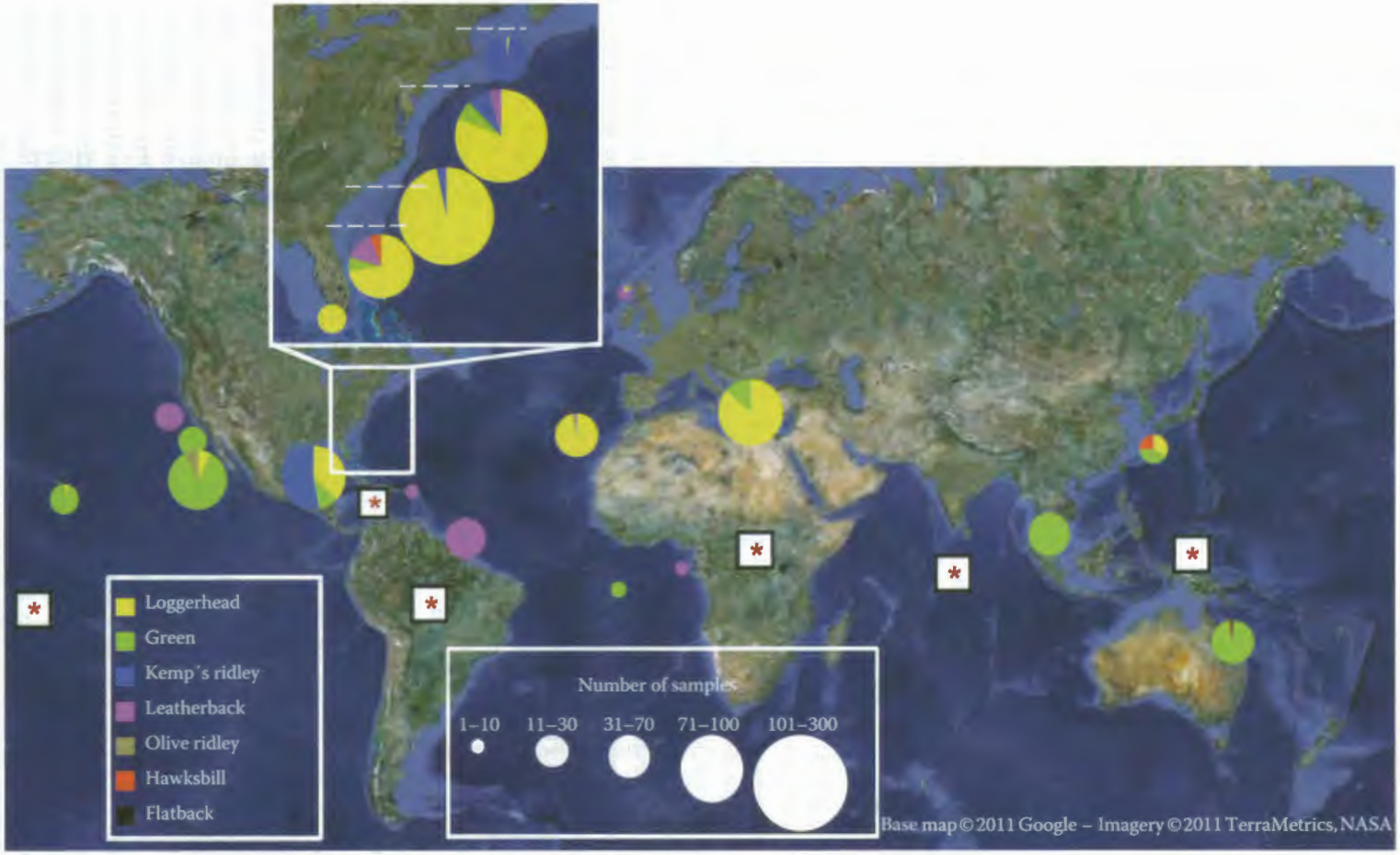

FIGURE 11.3 Global map of studies examining persistent organic pollutant (POP) concentrations in sea turtle tissues from 1974 to 2012. The number of samples included in studies assessing each species by region is shown by different sized pie-charts. The proportion of samples by species is shown inside each pie-chart. Red asterisks signify regions that have major data gaps. Dashed horizontal lines indicate latitudinal delineations of regions along the east coast of North America.

Low sample size is an issue easily deciphered from Figure 11.3. This problem is especially important for the less studied species, like the hawksbill sea turtle (*Eretmochelys imbricata*). Only three studies have reported POPs in hawksbills, all using small sample sizes: only three liver samples were analyzed from Japanese waters, a single blood sample from Australia, and five plasma samples from Florida (Hermanussen et al., 2008; Keller et al., 2012; Malarvannan et al., 2011). Worst yet is the flatback turtle (*Natator depressus*) with only a single turtle ever sampled for any POP (Hermanussen et al., 2008). Small sample sizes are likely the result of limited funding, opportunistic field sampling, and/or difficulty in accessing these endangered or threatened species, but studies with small sample sizes like these cannot provide an accurate baseline concentration for the species or location.

Despite these problems, Figure 11.3 accurately portrays that the loggerhead sea turtle, followed by the green turtle, has been the most frequently analyzed species. The east coast of the United States greatly outnumbers other regions, but the recent additions of locations, such as French Guiana, Malaysia, Baja California, and Japan (Gardner, 2003; Guirlet et al., 2010; Labrada-Martagon et al., 2011; Malarvannan et al., 2011; van de Merwe et al., 2009a, 2010b; Richardson et al., 2010) are beginning to provide a more descriptive understanding of baseline POP concentrations in sea turtles across the globe. However, very large spatial data gaps still exist, notably the South Pacific Ocean, Caribbean Sea, both coasts of South America, Africa, the Indian Ocean, and Southeast Asia (Figure 11.3).

Because of the different confounding factors, detection problems, and sample size limitations, a robust meta-analysis for assessing geographic hotspots or temporal trends is not possible even after four decades of work. More research is needed to gain a better picture of temporal and spatial trends and to simply understand baseline concentrations in vast regions globally and in species of certain age classes not yet assessed. With the predicted exponential growth of this field, surely more species and locations will be better assessed with time.

11.3.2 Reported Sea Turtle POP Concentrations

Since the last review of chemical pollutants in all sea turtle tissues in 2001 (Pugh and Becker, 2001), a larger diversity of tissues, species, and locations has been added to the literature. Tables 11.2 through 11.5 show measured POP concentrations from selected sea turtle studies that analyzed blood components, adipose, liver, and egg contents, respectively. The white space within each table is another indication of data gaps.

It is important to acknowledge that not all studies or all concentrations are shown in Tables 11.2 through 11.5. Selection criteria excluded studies or portions of data from studies that could not detect POPs (Aguirre et al., 1994; Deem et al., 2006; Harris et al., 2011; Innis et al., 2008), chose compounds that were not typically found in sea turtles or other wildlife (Monagas et al., 2008), reported their data in another publication with a larger sample size (Alava et al., 2006; Cobb and Wood, 1997; Keller et al., 2004a,b, 2006b), or presented data in units that could not be converted into the units shown (Davenport et al., 1990; Miao et al., 2001; Vetter et al., 2001). Also excluded are portions of data reported for other less studied tissues, like muscle, heart, lung, or kidney.

The units used for reporting POP concentrations also caused difficulty in creating comprehensive tables. POP tissue concentrations are most commonly reported as either wet mass, wet volume, or lipid normalized (e.g., ng/g wet tissue mass, ng/mL plasma, or ng/g lipid in tissue, respectively), and rarely in dry mass (e.g., ng/g dry tissue mass). Conversions are required for direct comparison of POP concentrations reported with different units, but often conversions must be done with estimates of water content, density, or lipid content, which can lead to inaccuracies in the POP concentrations. Normalizing POP concentrations to the actual lipid content of each sample is often argued to be the best choice for most POPs (not PFCs) in most tissues (not in blood) for comparisons. Unfortunately, the most commonly reported unit for sea turtle fat and liver is on a wet mass basis. Reporting both wet mass and lipid-normalized concentrations is suggested so that future comparisons can select which values to use.

TABLE 11.2
Persistent Organic Pollutant Levels in Blood Matrices of Sea Turtles from Selected Studies

Species	Stage/ Sex	Location	Year	Tissue	N	ΣPCBs	4,4′-DDE	ΣChlordanes	Dieldrin	Mirex	Σ2,3,7,8-PCDD/F	ΣPBDEs	PFOS	Reference
Lk	JMF	South Carolina-Florida	2001–2002	WB	3	6.610 (12.200)[a] NA (2.400–24.700)	0.773 (1.800)[a] NA (0.188–3.530)	1.140 (1.850)[a] NA (0.353–3.870)	0.500 (0.409)[a] NA (0.206–1.020)	0.00917 (NA)[a] NA (<0.0006–0.00952)		0.105 (0.141)[a] NA (0.038–0.307)		Swarthout et al. (2010)
Lk	JMF	Cape Cod, Massachusetts	1999	WB	8	4.33 (4.52)[b] 2.42 (0.304–11.4)[b]	0.894 (0.731)[b] 0.783 (0.186–2.38)[b]	0.417 (0.406)[b] 0.333 (0.057–1.29)[b]	0.114 (0.0834)[b] 0.0741 (<0.013–0.235)[b]	0.0460 (0.0646)[b] 0.0195 (<0.013–0.192)[b]				Keller et al. (2004a)
Lk	JMF	Louisiana-Texas, Gulf of Mexico	2001–2002	WB	46	3.190 (3.620)[a] NA (0.227–21.590)	0.472 (0.633)[a] NA (0.0439–4.110)	0.163 (0.249)[a] NA (0.0109–1.190)	0.199 (0.119)[a] NA (<0.0748–0.609)	0.0100 (0.0042)[a] NA (<0.0006–0.019)		0.146 (0.273)[a] NA (0.0195–1.450)		Swarthout et al. (2010)
Lk	JMF	South Carolina-Florida	2003	P	6								39.4 (17.1) 41.9 (13.8–60.2)	Keller et al. (2005b)
Lk	JMF	Core Sound, North Carolina	2006	P	10								15.7 (9.86) 10.8 (6.85–35.0)	Keller et al. (2012)
Cc	AM	Transients; migrated north to SC-NJ	2006–2007	P	9	13.1 (17.2) 7.27 (NA)	1.557 (2.656) 0.701 (NA)	0.335 (NA)[c] 0.241 (NA)[c]		0.0783 (0.0625) 0.0528 (NA)		0.0876 (0.0965) 0.043 (NA)		Ragland et al. (2011)
Cc	JMF	Core Sound, North Carolina	2000–2001	WB	48	7.23 (6.86)[b] 5.56 (0.157–31.1)[b]	0.830 (0.887)[b] 0.584 (<0.030–4.94)[b]	0.293 (0.260)[b] 0.207 (<0.013–1.28)[b]	0.0874 (0.181)[b] 0.477 (<0.013–1.24)[b]	0.0633 (0.0893)[b] 0.0202 (<0.013–0.384)[b]				Keller et al. (2004c)
Cc	AM	Residents; stayed at Cape Canaveral, Florida	2006–2007	P	11	5.38 (2.25) 5.03 (NA)	0.0917 (0.0323) 0.106 (NA)	0.0615 (NA)[c]		0.0230 (0.0161) 0.0144 (NA)		NA 0.0221 (NA)		Ragland et al. (2011)

Cc	JMF	South Carolina-Florida	2003	P	29	3.38 (3.43)[d] 2.34 (0.210–13.4)[d]	0.467 (0.644)[d] 0.242 (<0.0286–3.05)[d]			0.0277 (0.0260)[d] 0.0161 (<0.006–0.0937)[d]	0.152 (0.182)[d] 0.0780 (<0.0177–0.698)[d]		Keller et al. (2005a)
Cc	AF, JMF	Georgia-Florida	2000–2004	P	53	<20	<20	<20	<20				Deem et al. (2009)
Cc	JMF, AM	North Carolina-Florida	2003	P	73							11.0 (17.2) 5.45 (1.40–96.8)	Keller et al. (2005b)
Cc	JMF	Chesapeake Bay, Maryland	2005–2006	P or S	14							9.34 (11.4) 3.54 (NA)	O'Connell et al. (2010)
Cc	JMF	Core Sound, North Carolina	2006	P	15							6.47 (7.52) 3.13 (0.305–26.5)	Keller et al. (2012); O'Connell et al. (2010)
Cc	JMF	Charleston, South Carolina	2005–2006	P	9							3.86 (2.92) 2.87 (NA)	O'Connell et al. (2010)
Cc	JMF	Florida Bay	2005–2006	P	11							3.67 (1.03) 3.801 (NA)	O'Connell et al. (2010)
Cc	JMF	Cape Canaveral, Florida	2005–2006	P	10							1.44 (0.94) 1.24 (NA)	O'Connell et al. (2010)
Dc	AF	Juno Beach, Florida	2003	WB	6	2.50 (2.27) 1.62 (0.162–6.54)	0.424 (0.235) 0.317 (0.211–0.865)	0.328 (0.158) 0.281 (0.081–0.507)	0.140 (0.100) 0.097 (0.040–0.328)	<LOD <LOD (<0.009–<0.062)	0.198 (0.190) 0.155 (<0.050–0.510)		Stewart et al. (2011)
Dc	AF	French Guiana	2006	WB	≤44	1.26 (0.71) NA (0.86–4.04)							Guirlet et al. (2010)
Dc	JMF	Juno Beach, Florida	2007	P	7							3.95 (2.51) 4.42 (0.884–7.83)	Keller et al. (2012)
Cm	JAMF	San Diego Bay, California	2007–2009	P	20		0.736 (0.097)[e] 0.750 (<LOD–1.56)	0.775 (NA)[e] 0.846 (NA)[e]			<0.200 (<0.200–0.760)[f]		Komoroske et al. (2011)
Cm	JAMF	Punta Abreojos, Baja California	2005 and 2007	P	39		<LOD[g]	0.230227 (0.001–1.31)[g]	<LOD				Labrada-Martagon et al. (2011)

(continued)

TABLE 11.2 (continued)
Persistent Organic Pollutant Levels in Blood Matrices of Sea Turtles from Selected Studies

Species	Stage/ Sex	Location	Year	Tissue	N	ΣPCBs	4,4′-DDE	ΣChlordanes	Dieldrin	Mirex	Σ2,3,7,8-PCDD/F	ΣPBDEs	PFOS	Reference
Cm	JAMF	Bahía Magdalena, Baja California	2005 and 2007		13		<LOD[g]	0.313725 (0.001–0.84)[g]	<LOD					Labrada-Martagon et al. (2011)
Cm	HMF	Peninsular Malaysia	2004	WB	11 pools	0.8508 (0.1052)[e] NA (0.5594–1.4566)				0.1324 (0.0340)[e] NA (<LOD–0.3406)		0.0830 (0.0144)[e] NA (0.0238–0.1732)		van de Merwe et al. (2010b)
Cm	JMF	Queensland, Australia (rehab)	2006–2007	WB	16	0.6839 (0.1528)[e] NA (0.1983–1.6948)	<0.035		<0.035	0.0428 (0.0162)[e] NA (<0.035–0.1157)		0.0793 (0.0108)[e] NA (0.0424–0.1208)		van de Merwe et al. (2010a)
Cm	AF	Peninsular Malaysia	2004	WB	11	0.5789 (0.0856)[e] NA (0.3164–1.2065)				0.161 (0.0430)[e] NA (<LOD–0.4763)		0.1208 (0.0141)[e] NA (0.0575–0.2243)		van de Merwe et al. (2010b)
Cm	JMF	Texas, Gulf of Mexico	2001–2002	WB	9	0.331 (0.701)[a] NA (0.117–2.330)	0.0664 (0.110)[a] NA (<0.0101–0.348)	0.0164 (0.0257)[a] NA (<0.0022–0.0643)	0.0960 (NA)[a] NA (<0.0748–0.096)	0.0111 (0.0114)[a] NA (<0.0006–0.0377)		0.0806 (0.217)[a] NA (0.0244–0.623)		Swarthout et al. (2010)
Cm	JAMF	Queensland, Australia	2004–2006	P	1 pool of 7							0.00900		Hermanussen et al. (2008)
Cm	JAMF	Queensland, Australia	2004–2006	WB	1 pool of 7							0.00444		Hermanussen et al. (2008)
Cm	MF	Polluted site, Moreton Bay, Australia	NR	WB	7						0.910 (NA)[h] NA (0.510–1.400)[h]			Hermanussen et al. (2006)
Cm	MF	Variable site, Moreton Bay, Australia	NR	WB	6						0.580 (NA)[h] NA (0.160–1.900)[h]			Hermanussen et al. (2006)

Cm	MF	Background site, Moreton Bay, Australia	NR	WB	16	0.090 (NA)[h] NA (0.030–0.200)[h]			Hermanussen et al. (2006)
Cm	JMF	Core Sound, North Carolina	2006	P	10			2.41 (1.12) 2.37 (0.871–3.87)	Keller et al. (2012)
Ei	JF	Queensland, Australia	2004–2006	WB	1		0.01300		Hermanussen et al. (2008)
Ei	JMF	Juno Beach, Florida	2006	P	5			11.9 (6.27) 11.9 (5.45–21.2)	Keller et al. (2012)
Nd	AF	Queensland, Australia	2004–2006	WB	1		0.00609		Hermanussen et al. (2008)

Mean (SD) and/or median (range) in ng/g wet mass, unless otherwise stated. Ranked generally from highest to lowest by species and location.

Kemp's ridley (Lk), loggerhead (Cc), leatherback (Dc), green (Cm), hawksbill (Ei), flatback (Nd) sea turtles, juvenile (J), adult (A), male (M), female (F), hatchling (H), whole blood (WB), plasma (P), serum (S), polychlorinated biphenyls (PCBs), polychlorinated dibenzo-*p*-dioxin/furan (PCDD/F), perfluorooctane sulfonate (PFOS), not available (NA), not reported (NR), below detection (<LOD).

[a] Geometric mean (standard deviation).

[b] Original data corrected by multiplying values in ng/g wet mass by 1.3 because of immiscible solvent used for internal standards (Keller et al., 2009); summary statistics recalculated using Helsel (2005) recommendations.

[c] Sum of means or medians of detected compounds.

[d] Summary statistics recalculated using original data in ng/g wet mass and Helsel (2005) recommendations.

[e] Arithmetic mean (standard error).

[f] Values include only PBDE 47.

[g] 4,4′-DDE was not detected, but 2,4′-DDD was. Total chlordane values represent only *trans*-chlordane and *cis*-chlordane; other chlordanes were <LOD.

[h] Units are ng/g lipid, no lipid content was provided for a conversion.

TABLE 11.3
Persistent Organic Pollutant Levels in Fat of Sea Turtles from Selected Studies

Species	Stage/ Sex	Location	Years	Tissue	N	ΣPCBs	4,4′-DDE	ΣChlordanes	ΣHCHs	HCB	Dieldrin	Mirex	Heptachlor Epoxide	ΣPBDEs	% Lipid	Reference
Lk	JMF	Long Island, New York	1985	Body fat	7	1250 (985)	386 (250)									Lake et al. (1994)
Lk	J	Virginia-North Carolina	1991	Fat	3	660 (333) 794 (281–904)	194 (98.2) 194 (95.7–292)									Rybitski et al. (1995)
Lk	JMF	Cape Cod, Massachusetts	1999	Yellow fat	9	701 (893)[a] 368 (49.0–2920)[a]	99.6 (76.4)[a] 75.2 (19.2–238)[a]	103 (95.3)[a] 58.5 (12.7–305)[a]	52.5 (73.3)[a] 10.9 (<1–205)[a]	13.6 (9.8)[a] 12.2 (<1–30.7)[a]	21.3 (13.7)[a] 22.5 (5.32–43.8)[a]	3.83 (3.09)[a] 2.20 (<1–11.1)[a]	16.7 (23.0)[a] 7.52 (0.998–75.3)[a]		65.8 (10.6) 70.7 (57.7–74.8)	Keller et al. (2004a)
Lk	JMF	Cape Cod, Massachusetts	1999	Brown fat	10	525 (545)[a] 317 (16.6–1680)[a]	91.0 (72.0)[a] 75.7 (6.00–219)[a]	84.1 (70.7)[a] 52.0 (5.92–206)[a]	60.8 (62.9)[a] 47.9 (<1–170)[a]	10.5 (9.09)[a] 4.95 (<1–25.5)[a]	18.3 (13.3)[a] 14.3 (1.20–40.4)[a]	3.96 (2.78)[a] 2.54 (<1–10.9)[a]	22.1 (28.4)[a] 13.3 (<1–91.5)[a]		62.0 (23.9) 72.8 (0.521–80.3)	Keller et al. (2004a)
Lk	JMF	Long Island, New York	1989	Body fat	6	476 (273)	232 (157)									Lake et al. (1994)
Lk	J	Cape Cod, Massachusetts	2005	Fat	3		54 (51–209)	<10[b]	<10		<10 (<10–10)		<10			Innis et al. (2008)
Cc	JAMF	Cyprus and Greece	1994–1995	Adipose	3	840 (60.0) 853 (775–893)	509 (173) 446 (376–705)	19.7 (11.6) 14 (12–33)			5.4 (4.2) 6.2 (<1.8–9.2)					Mckenzie et al. (1999)
Cc	NR	NR (probably U.S. East coast)	1986	Body fat	NR (maybe 1)	647	300									Lake et al. (1994)
Cc	JAMF	Virginia-North Carolina	1991–1992	Fat	20	551 (473) 365 (55.4–1730)	195 (266) 99.0 (2.86–1210)									Rybitski et al. (1995)

Cc	JAMF	Eastern Adriatic Sea	2001–2002	Yellow fat	27	474 (547) 312 (177–2934)	92.4 (53.0) 81.0 (19.1–282)			1.9 (1.3) 1.8 (<LOD–5.9)					47.9 (13.6) 51.1 (25.9–68.2)	Lazar et al. (2011)
Cc	JMF	Southern and central Adriatic Sea	2003–2004	Fat	9	459.7 (NR) NA (2.9–1472.1)	280.9 (NR) NA (1.5–621)									Perugini et al. (2006)
Cc	JMF	Canary Islands	2002–2005	Fat	30	450 (1700) NA (<LOD–9800)										Orós et al. (2009)
Cc	JMF	Northeast Italy, Adriatic Sea	1993	Adipose	4	334 (179) 136–563										Corsolini et al. (2000)
Cc	JMF	Core Sound, North Carolina	2000–2001	Fat	44	256 (269)[a] 171 (7.99–1360)[a]	64.9 (64.3)[a] 43.2 (<0.09–273)[a]	26.9 (21.3)[a] 20.3 (<0.339–87.8)[a]	2.15 (7.15)[a] 0.0655 (<1–43.5)[a]	1.13 (2.38)[a] 0.233 (<1–12.6)[a]	5.04 (3.90)[a] 3.54 (<0.09–16.7)[a]	4.52 (4.06)[a] 3.34 (<0.09–18.8)[a]	3.11 (2.23)[a] 2.25 (<1–10.6)[a]		26.3 (20.6) 26.1 (4.68–42.6)	Keller et al. (2004a)
Cc	NR	Baja California	NR	Adipose	1	<3		<3		<3	<3					Gardner et al. (2003)
Dc	AMF, JF	South Carolina-North Carolina	1999–2003	Blubber	7	193 (384) 66.9 (1.52–1061)	41.5 (64.4) 22.7 (4.80–185)	52.2 (93.6) 23.3 (3.11–263)	<LOD	0.700 (0.335) 0.657 (0.323–1.11)	8.39 (10.3) 4.67 (2.92–31.6)	<LOD (<0.352–7.60)	1.73 (2.79) 0.170 (<0.349–7.80)	25.7 (41.1) 9.99 (<2.94–116)	52.7 (16.9) 58.5 (14.9–63.2)	Stewart et al. (2011)
Dc	AM	Scotland and Wales	1993–1996	Adipose	3	178 (47–230)	57 (10–68)			2 (<1–3)	13 (<1–13)				50 (41–74)	Godley et al. (1998)
Dc	AM	Scotland	1993–1995	Adipose	2	NA (47–178)	NA (10–57)	NA (12–22)			NA (13–19)					Mckenzie et al. (1999)
Dc	AMF, JF	South Carolina-North Carolina	1999–2003	Fat	7	90.1 (65.9) 75.1 (4.87–188)	19.7 (10.9) 20.0 (5.14–35.7)	22.4 (14.4) 19.6 (3.14–47.3)	<LOD	0.628 (0.363) 0.743 (0.121–1.18)	4.41 (1.92) 4.71 (2.16–7.92)	0.379 (0.404) 0.164 (<0.046–0.944)	0.929 (0.716) 0.430 (<0.225–2.29)	15.4 (6.67) 13.2 (<1.67–26.0)	48.1 (23.9) 60.6 (0.697–67.9)	Stewart et al. (2011)
Dc	AF	Canary Islands	2002–2005	Fat	1	77										Orós et al. (2009)
Cm	JF	Canary Islands	2002–2005	Fat	1	144										Orós et al. (2009)
Cm	JMF	Cyprus	1995	Adipose	3	109 (39–261)	6 (2.4–19)				1.5 (<1.9–3.5)					Mckenzie et al. (1999)

(continued)

TABLE 11.3 (continued)
Persistent Organic Pollutant Levels in Fat of Sea Turtles from Selected Studies

Species	Stage/ Sex	Location	Years	Tissue	N	ΣPCBs	4,4′-DDE	ΣChlordanes	ΣHCHs	HCB	Dieldrin	Mirex	Heptachlor Epoxide	ΣPBDEs	% Lipid	Reference
Cm	JMF	Baja California	NR	Adipose	7	NA (<3–49.5)		NA (<3–65.1)		<3	<3					Gardner et al. (2003)
Cm	AF	Queensland, Australia	2004–2006	Adipose	1									0.25740	78.00	Hermanussen et al. (2008)
Lo	NR	Baja California	NR	Adipose	1	18.4		8.1		<3	<3					Gardner et al. (2003)

Mean (SD) and/or median (range) in ng/g wet mass, unless otherwise stated. Ranked generally from highest to lowest by species and location.

Kemp's ridley (Lk), loggerhead (Cc), leatherback (Dc), green (Cm), olive ridley (Lo) sea turtles, juvenile (J), adult (A), male (M), female (F), polychlorinated biphenyls (PCBs), hexachlorocyclohexanes (HCHs), hexachlorobenzene (HCB), polybrominated diphenylethers (PBDEs), not available (NA), not reported (NR), below detection (<LOD).

[a] Summary statistics recalculated using original data in ng/g wet mass and Helsel (2005) recommendations.

[b] *Trans*- and *cis*-chlordane were the only chlordanes measured.

TABLE 11.4
Persistent Organic Pollutant Levels in Liver of Sea Turtles from Selected Studies

Species	Stage/ Sex	Location	Years	N	ΣPCBs	4,4′-DDE	ΣChlordanes	ΣHCHs	HCB	Dieldrin	Mirex	Heptachlor Epoxide	ΣPBDEs	% Lipid	Reference
Lk	JMF	Long Island, New York	1985	8	738 (737)	253 (162)									Lake et al. (1994)
Lk	J	Virginia-North Carolina	1991	3	360 (158–608)	55.5 (54.2–56.8)									Rybitski et al. (1995)
Lk	JMF	Long Island, New York	1989	6	272 (126)	137 (85.3)									Lake et al. (1994)
Lk	J	Cape Cod, Massachusetts	2005	3		14 (<10–15)	<10[a]	<10		<10		<10			Innis et al. (2008)
Cc	JMF	Canary Islands	2002–2005	30	1980 (5320) NA (<LOD–34900)										Orós et al. (2009)
Cc	NR	NR (likely east coast U.S.)	1986	1	360[b]	110[b]									Lake et al. (1994)
Cc	J	Scotland	1995	1	159	149	NR			NR					Mckenzie et al. (1999)
Cc	JAMF	Virginia-North Carolina	1991–1992	18	145 (158) 99.7 (7.46–514)	47.5 (104) 18.9 (<2–458)									Rybitski et al. (1995)
Cc	JMF	Northeast Italy, Adriatic Sea	1993	4	119 (60) 69–205										Corsolini et al. (2000)
Cc	NR	NR (likely east coast U.S.)	1988	1	110[b]	50[b]									Lake et al. (1994)
Cc	JMF AM	Cyprus and Greece	1994–1995	4	84.0 (24.3) 92.0 (50–102)	60.5 (13.7) 58.5 (49–76)	3.0 (1.8) 2.1 (1.8–5)			1.8 (1.3) 2.5 (0.3–2.7)					Mckenzie et al. (1999)
Cc	JMF	Southern and central Adriatic Sea	2003–2004	11	83 (NR) NA (7.6–247.3)	40.6 (NR) NA (3.6–217.3)									Perugini et al. (2006)
Cc	JMF	Southern Adriatic Sea and Ionian Sea	1990–1991	5	77.2[c]	6.31[c]			0.616[c]					1.54 (0.59) NA (0.90–2.42)	Storelli and Marcotrigiano (2000)

(continued)

TABLE 11.4 (continued)
Persistent Organic Pollutant Levels in Liver of Sea Turtles from Selected Studies

Species	Stage/ Sex	Location	Years	N	ΣPCBs	4,4′-DDE	ΣChlordanes	ΣHCHs	HCB	Dieldrin	Mirex	Heptachlor Epoxide	ΣPBDEs	% Lipid	Reference
Cc	JMF	East Florida	NR	8	64.4 (61.1) 40.5 (8.0–182)	21.5 (32.8) 8.5 (2–100)									McKim and Johnson (1983)
Cc	AF	Southern Adriatic Sea and Ionian Sea	1990–1991	6	55.7[c]	65.2[c]			6.36[c]					15.91 (9.64)	Storelli and Marcotrigiano (2000)
Cc	JMF	Southern Adriatic Sea and Ionian Sea	1999–2001	19	52.32 (74.99) NA (6.85–297.49)									6.5 (5.7) NA (0.5–23.3)	Storelli et al. (2007)
Cc	NR	Baja California	NR	1	41.0		<3		<3	<3					Gardner et al. (2003)
Cc	JMF	Baja California	2001 2003	4	19.3 (5.80)										Richardson et al. (2010)
Cc	JMF	Ishigaki Island and Kochi, Japan	1998 2006	4	2.10 (1.21) 1.71 (1.185–3.8)	2.46 (0.94)[d] 2.16 (1.7–2.37)[d]	0.711 (0.506) 0.553 (0.292 1.444)	0.257 (0.172) 0.183 (0.150–0.513)	0.0901 (0.0383) 0.0869 (0.05688-0.1159)				0.269 (0.391) 0.081 (0.060–0.855)	10.5 (5.7) 8.2 (6.5–19)	Malarvannan et al. (2011)
Lo	NR	Baja California	NR	1	58.1		45.3		3.5	<3					Gardner et al. (2003)
Lo	JMF	Baja California	2001–2003	4	15.2 (6.17)										Richardson et al. (2010)
Dc	AF	Canary Islands	2002–2005	1	445										Orós et al. (2009)
Dc	AM	Scotland	1993–1995	2	NA (3.1–3.7)	NA (1.7–6.5)	NA (2.3 2.3)			NA (2.5–3.1)					Mckenzie et al. (1999)
Cm	JF	Canary Islands	2002–2005	1	116										Orós et al. (2009)
Cm	JMF	East Florida	NR	4	65.3 (15.7) 69 (43–80)	3.4 (4.5) 1.5 (<1–10)									McKim and Johnson (1983)
Cm	JMF	Cyprus	1995–1996	9	33.6 (22.2) 34 (<1.1–7.7)	4.89 (6.40) 2.7 (<1.0–2.1)	1.07 (1.35) 0.45 (<0.4–3.7)			1.43 (1.07) 1.5 (<0.4–3)					Mckenzie et al. (1999) Mckenzie et al. (1999)
Cm	JMF	Baja California	NR	7	NA (<3 44.7)		NA (<3–10.4)		NA (<3 18.6)	<3					Gardner et al. (2003)

Cm	JMF	Baja California	2001--2003	3	10.5 (4.92)										Richardson et al. (2010)
Cm	JMF	Queensland, Australia (rehab)	2006–2007	16	1.1527 (0.3092)[e] NA (0.4111–3.0823)	0.0355 (0.0137)[e] NA (<0.010–0.1144)	0.138[f]	0.131[g]	0.0404 (0.0120)[e] NA (<0.010–0.1200)	0.3871 (0.0716)[e] NA (0.0766–0.7770)	0.07351 (0.03528)[e] NA (0.00592–0.35739)	0.1692 (0.0403)[e] NA (0.0218–0.4109)	0.1201 (0.0213)[e] NA (0.0544–0.2360)	2.37 (0.44)[e] NA (1.15–4.84)	van de Merwe et al. (2010a)
Cm	AF	Queensland, Australia	2004–2006	1									0.08080	5.05	Hermanussen et al. (2008)
Cm	JMF	Ishigaki Island, Japan	2003–2005	5	0.257 (0.375) 0.0975 (0.0403–0.924)	0.0809 (0.113)[d] 0.0225 (<0.003–0.104)[d]	0.372 (0.716) 0.026 (0.015–1.65)	0.123 (0.094) 0.0840 (0.045–0.26)	0.0439 (0.0152) 0.0468 (0.03–0.0645)				0.150 (0.260) 0.029 (0.0084–0.613)	11.8 (3.5) 13.0 (7.7–15.0)	Malarvannan et al. (2011)
Ei	JMF	Ishigaki Island, Japan	2002–2004	3	7.34 (5.59) 6.12 (2.46–13.44)	3.43 (4.84)[d] 0.72 (0.56–9.02)[d]	1.12 (0.41) 1.312 (0.648–1.4)	0.160 (0.148) 0.133 (0.02706–0.3192)	0.0245 (0.0267) 0.0134 (0.00504–0.0549)				1.85 (2.77) 0.386 (0.115–5.04)	5.8 (2.3) 5.6 (3.6–8.2)	Malarvannan et al. (2011)

Mean (SD) and/or median (range) in ng/g wet mass, unless otherwise stated. Ranked generally from highest to lowest by species and location.

Kemp's ridley (Lk), loggerhead (Cc), leatherback (Dc), green (Cm), olive ridley (Lo) sea turtles, juvenile (J), adult (A), male (M), female (F), polychlorinated biphenyls (PCBs), hexachlorocyclohexanes (HCHs), hexachlorobenzene (HCB), polybrominated diphenylethers (PBDEs), not available (NA), not reported (NR), below detection (<LOD).

[a] Summary statistics recalculated using original data in ng/g wet mass and Helsel (2005) recommendations.

[b] *Trans*- and *cis*-chlordane were the only chlordanes measured.

TABLE 11.5
Persistent Organic Pollutant Levels in Eggs and Hatchlings of Sea Turtles from Selected Studies

Species	Location	Years	Tissue	N Nests (Eggs/ Nest)	Live Eggs Sacrificed?	ΣPCBs	ΣDDTs	ΣChlordanes	ΣHCHs	HCB	Dieldrin	Mirex	Heptachlor Epoxide	ΣToxaphenes	ΣPBDEs	% Lipid	Reference
Cc	Northwest Florida, Gulf of Mexico	1992	Egg contents	20 (4–12)	No	240–3720	<LOD–178[a]	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD				Alam and Brim (2000)
Cc	Cape Lookout, North Carolina	2002	Egg yolk	9 (2–7)	No	117 (118)[b] 70.8 (2.52–273)	53.2 (57.1)[b] 61.9 (0.369–164)	28.6 (33.1)[b] 27.8 (0.285–97.2)	0.239 (0.314)[b] 0.0707 (<0.049–0.989)		2.35 (2.73)[b] 0.623 (<0.180–6.94)	0.789 (0.679)[b] 0.708 (0.0334–2.24)	4.37 (5.34)[b] 2.76 (<0.048–16.2)	0.240 (0.244)[b] 0.162 (0.0177–0.677)	1.04 (1.09)[b] 0.522 (0.0392–2.77)	7.68 (0.33)[b] 7.41 (6.51–9.51)	Alava et al. (2011)
Cc	South Carolina-Georgia	NR	Egg	>3 (NR)	NR		NA (58–305)				NA (<LOD–56.4)						Hillestad et al. (1974)
Cc	Cyprus	1995	Whole egg	1 (1)	No	89	155	1.8			0.6						Mckenzie et al. (1999)
Cc	Merritt Island, eastern Florida	1979	Egg contents	1 (56)	Yes		99 (56–150)[c]										Clark and Krynitsky (1985)
Cc	Merritt Island, eastern Florida	1976	Egg contents	9 (20+)	Yes and no	78 (32–201)[d]	47 (18–200)[c,d]	NA (<LOD–26)[c]			NA (<LOD–28)	NA (<LOD–5)	NA (<LOD–6)				Clark and Krynitsky (1980)
Cc	Cyprus	1995	Hatchling	≤4 (1+)	No	34.0 (22–71)	36.5 (5.3–113)	3.0 (0.9–7.9)			1.3 (<0.2–9.2)					7.7 (4.8–8.6)	Mckenzie et al. (1999)

Cc	eastern Florida	2002	Egg yolk	24 (1–10)	No	18.4 (29.0)[b] 10.1 (0.449–113)	10.6 (23.7)[b] 2.95 (0.0493–108)	8.46 (12.8)[b] 3.13 (0.046–55.5)	0.0956 (0.165)[b] 0.0423 (<0.049–0.620)		0.734 (0.797)[b] 0.447 (<0.180–3.28)	0.505 (1.31)[b] 0.124 (<0.049–6.21)	1.45 (1.96)[b] 0.705 (<0.048–8.84)	0.131 (0.148)[b] 0.0499 (0.006–0.546)	0.220 (0.195)[b] 0.175 (<0.019–0.657)	7.68 (0.54)[b] 7.40 (4.53–13.1)	Alava et al. (2011)
Cc	Sarasota, Florida, Gulf of Mexico	2002	Egg yolk	11 (2–8)	No	3.14 (5.20)[b] 0.842 (0.158–16.5)	2.23 (2.60)[b] 1.05 (0.168–7.95)	1.94 (3.38)[b] 0.618 (<0.049–11.4)	0.0282 (0.0159)[b] 0.0241 (<0.049–0.0624)		0.457 (0.490)[b] 0.321 (0.204–1.88)	0.0926 (0.185)[b] 0.00939 (<0.049–0.603)	0.477 (0.575)[b] 0.256 (<0.048–1.81)	0.0291 (0.0227)[b] 0.0172 (<0.005–0.0678)	0.0936 (0.085?)[b] 0.0586 (<0.017–0.245)	8.65 (0.82)[b] 8.42 (2.6–12.7)	Alava et al. (2011)
Dc	Juno Beach, Florida	2003	Yolk + albumen	8 (1–6)	No	8.45 (7.59) 4.15 (0.441–19.9)	1.87 (1.02) 1.53 (0.683–3.49)	2.28 (1.71) 1.36 (0.562–5.39)	<LOD	0.225 (0.076) 0.207 (0.150–0.368)	0.535 (0.347) 0.450 (0.132–1.16)	<0.084 (<0.083 to <0.084)	0.219 (0.091) 0.183 (0.096–0.362)	0.074 (0.029) 0.061 (0.048–0.121)	0.845 (0.630) 0.689 (0.121–1.64)	5.00 (0.380) 4.89 (4.67–5.69)	Stewart et al. (2011)
Dc	French Guiana	2006	Yolk + albumen	46 (1)	Yes	6.98 (5.02) NA (1.18–23.62)	1.44 (1.26) NA (0.08–5.82)		0.41 (0.26) NA (0.08–1.0)							12.9 (5.1) NA (3.8–25.5)	Guirlet et al. (2010)
Cm	Ascension Island	1972	Egg yolk	4 (10)	Yes	76 (64) 45 (20–220)											Thompson et al. (1974)
Cm	Cyprus	1995	whole egg	1 (1)	No	6.1	4.3	<0.3			<0.3						Mckenzie et al. (1999)
Cm	Merritt Island, eastern Florida	1976	Egg contents	2 (5+)	Yes and no		NA (<LOD–47)[f]										Clark and Krynitsky (1980)
Cm	Heron Island, Queensland, Australia	1995	Egg contents	4 (1–5)	Yes		1.7 (0.2)[g] 1.6 (1.5–2.0)[c]										Podreka et al. (1998)
Cm	Cyprus	1995	Hatchling	3 (1)	No	1.1 (<0.4–13)	0.2 (<0.4–5.8)	<LOD			<LOD					7.53 (0.55)	Mckenzie et al. (1999)
Cm	Peninsular Malaysia	2004	Yolk + albumen	11 (3)	Yes	0.5536 (0.0546)[b] NA (0.3928–0.8394)		0.0183 (0.0009)[b,i] NA (0.0138–0.0223)[b]	0.1723 (0.0074)[b,i] NA (0.1378–0.2078)			0.0094 (0.0011)[b] NA (<LOD–0.0128)			0.1293 (0.0081)[b] NA (0.0617–0.1638)	8.9 (0.2)[b] NA (6.8–10.9)	van de Merwe et al. (2010b)

(continued)

TABLE 11.5 (continued)
Persistent Organic Pollutant Levels in Eggs and Hatchlings of Sea Turtles from Selected Studies

Species	Location	Years	Tissue	N Nests (Eggs/ Nest)	Live Eggs Sacrificed?	ΣPCBs	ΣDDTs	ΣChlordanes	ΣHCHs	HCB	Dieldrin	Mirex	Heptachlor Epoxide	ΣToxaphenes	ΣPBDEs	% Lipid	Reference
Cm	Peninsular Malaysian markets	2006	Yolk + albumen	Perhaps 55 (1)	Yes	0.4705 (0.0833)[b] NA (0.1466–3.6915)	0.0835 (0.0183)[b] NA (<LOD–0.7019)	0.0575 (0.0094)[b] NA (0.0247–0.5142)	0.0688 (0.0087)[b] NA (0.0132–0.2301)						0.0214 (0.0066)[b] NA (<LOD–0.3527)	9.33 (0.14)[b] NA (7.3–13.54)	van de Merwe et al. (2009a)
Cm	Heron Island, Australia	1998	Yolk + albumen	10 reps of 1 pool of 15 eggs from 3-4 nests	Yes	0.25616[j]		0.04319[k]		0.01294 (0.00073)[b]	0.4225 (0.0039)[b]		0.01974 (0.00026)[b]		0.30956[l]		van de Merwe et al. (2009b)

Mean (SD) and/or median (range) in ng/g wet mass, unless otherwise stated. Ranked generally from highest to lowest by species and location.

Loggerhead (Cc), leatherback (Dc), green (Cm) sea turtles, polychlorinated biphenyls (PCBs), sum of dichlorodiphenyltrichloroethane-related compounds (ΣDDTs), hexachlorocyclohexanes (HCHs), hexachlorobenzene (HCB), polybrominated diphenylethers (PBDEs), not available (NA), not reported (NR), below detection (<LOD).

[a] Value represents only 4,4′-DDD and was estimated from ng/g dry weight using a conversion factor calculated from the PCB concentrations that were reported as both wet mass and dry mass (0.223 g dry mass/g wet mass).

[b] Arithmetic mean (standard error).

[c] Geometric mean (range) of only 4,4′-DDE.

[d] Geometric mean (range).

[e] Maximum value estimated by summing *trans*-nonachlor and oxychlordane maximums.

[f] Maximum value estimated by summing 4,4′-DDE and 4,4′-DDT maximums.

[g] Values represent only 4,4′-DDE.

[h] Values represent only *trans*-chlordane, the only chlordane detected.

[i] Values represent only γ-HCH, the only HCH isomer detected.

[j] Value represents sum of means of 16 individual PCB congeners reported.

[k] Value represents sum of means of *cis*- and *trans*-chlordane and *cis*-nonachlor.

[l] Value represents sum of means of seven individual PBDE congeners reported.

Another complication is that most POP data are not normally distributed, so the best estimates of central tendency are medians or geometric means, rather than arithmetic means, but few studies provide these. Thus, Tables 11.2 through 11.5 show concentrations reported only in wet mass units (or estimated using conversions), and the tables show both the arithmetic mean as well as a preferred estimate of central tendency (median or geometric mean) when available.

11.3.2.1 Comparison of Different POP Classes

The predominant POP classes are shown in Tables 11.2 through 11.5. Chemicals were excluded if they were of very low concentrations, seldom measured, or could be represented in totals of a class of POP (represented here with the symbol Σ). Regardless of the tissue, the different classes of POPs generally ranked from highest to lowest concentration in sea turtles in the following order: ΣPCBs, ΣDDTs, other organochlorine pesticides (Σchlordanes, Σtoxaphenes, mirex, dieldrin), and ΣPBDEs (Figure 11.1; Tables 11.2 through 11.5). HCB and ΣHCHs are often very low in concentrations, when detected. In blood, however, PFOS can rival or exceed ΣPCB concentrations. No study has measured every single POP listed on the Stockholm Convention in the same turtle, but Figure 11.1 is the best across-study comparison for the largest number of POP classes. In loggerhead blood from the southeastern region of the United States, ΣPCBs are usually the predominant POP class in concentration, followed closely by the single compound of PFOS and then three classes of OCPs, ΣPBDEs, and a few more OCPs. Several compound classes were below detection in loggerhead blood from the southeastern coast of the United States, but this pattern may be different in other species, tissues, or regions. Four POP classes have not been measured in this population of loggerheads (dioxins/furans, chlordecone, the paraffins, and hexabromobiphenyls) nor in any other sea turtle, except for dioxins/furans, which have been detected in green turtles from Australia (Hermanussen et al., 2006).

Many of these POP concentrations are reported as totals or sums, which create additional confounding complexity when comparing results from different studies. For example, there are 209 possible PCB congeners (or configurations of chlorine atoms around the biphenyl structure), and usually the highly recalcitrant bioaccumulative congener, PCB 153, dominates the PCB pattern. Because each laboratory measures and sums a different suite and number of PCB congeners, it is not always accurate to compare summed or total PCB concentrations. Thus, comparing only one PCB congener, like PCB 153, may make a better comparison. However, individual congener concentrations have not been reported in most sea turtle studies, leaving only ΣPCB concentrations to be compared here. The same problem exists for the six DDT-related compounds (of which 4,4′-DDE, the final and very persistent metabolite of the DDT pesticide formulation, nearly always predominates), the several chlordane compounds (*trans*-nonachlor and oxychlordane predominate but detections of *cis*- and *trans*-chlordane, and *cis*-nonachlor are also common), the large number of toxaphenes (usually Parlars 26 and 50 are the highest), and the 209 possible PBDE congeners (PBDE 47 most always predominates in wildlife tissues).

The POP patterns measured in sea turtles generally match those measured in other wildlife with a few exceptions in localized areas. Surprisingly, 4,4′-DDE was not the predominant DDT in sea turtles from Baja California, where 2,4′-DDD and 4,4′-DDD were predominant (Gardner, 2003; Labrada-Martagon et al., 2011) or in loggerhead eggs from northwestern Florida, where 4,4′-DDD was predominant (Alam and Brim, 2000). These patterns are difficult to explain and should be validated. Additionally, an interesting trend in PBDE patterns is emerging in the sea turtle literature. Globally, PBDE 47 is the PBDE congener in highest concentration in most wildlife (Hites, 2004). However, some sea turtles have higher proportions of PBDEs 99, 100, 153, and/or 154 instead. This deviation from the expected pattern is found exclusively in turtles (sea turtles, diamondback terrapins, and freshwater turtles) inhabiting the eastern United States between 35°N and 45°N latitude (Basile et al., 2011; Carlson, 2006; Moss et al., 2009; Ragland et al., 2011). Other wildlife or fish within this region (Chen et al., 2010; Hale et al., 2006), and freshwater and sea turtles just outside of this region accumulate the expected pattern (Keller et al., 2005a; Ragland et al., 2011; de Solla et al., 2007; Stewart et al., 2011; Swarthout et al., 2010). Thus, these findings are not specific to one sea

turtle species, rather to turtles in general within this region. Some sea turtles in regions far from the United States also have PBDE patterns that deviate from the expected pattern (Hermanussen et al., 2008; van de Merwe et al., 2010b). This is interesting because it suggests that sea turtles are good bioindicator species of release of unusual environmental contaminants at least at the regional scale.

11.3.2.2 Biological Factors Influencing POP Concentrations

It is crucial to examine Tables 11.2 through 11.5 with the understanding that POP concentrations are driven not only by localized uses and releases of the chemicals within certain watersheds but also by environmental factors on scales ranging from local to global (e.g., water currents, air movements, temperature, precipitation, salinity, and organic matter content) and biological factors. Here, biological factors (lipid content, body condition, trophic status, age, and sex) influencing POP concentrations and accumulation in sea turtles will be discussed.

11.3.2.2.1 Lipid Content Influences Tissue Differences

Lipid content of various organs drives the distribution of POPs throughout the body. Most POPs, except for PFCs, distribute into tissues with the highest lipid content; therefore, fat, adipose, or blubber tissues (30%–80% lipid) followed by liver (5%–15% lipid) have higher lipid and thus higher POP concentrations than other bodily tissues. These are often the tissues chosen for POP assessment, but they can only be obtained from a dead animal or with invasive surgical procedures. Eggs, containing 5%–10% lipid, are also a choice sample. Earlier studies focused on these tissues, while more recent studies have switched to blood sampling (Table 11.1).

Because blood has very low lipid content (<1%), POP levels in blood are much lower than concentrations in fat, liver, or eggs (Tables 11.2 through 11.5). Blood analysis, therefore, requires highly sensitive methods for detection. This issue has been a major disadvantage for several unsuccessful studies whose detection limits for individual compounds were 10 or 20 ng/g wet mass (Deem et al., 2006; Harris et al., 2011; Innis et al., 2008), which would not allow detection of the maximum concentration in sea turtle blood analyzed to date (Table 11.2). Because of the low lipid level in blood, POPs likely also bind to plasma proteins, which is one argument against normalizing blood POP concentrations to solely lipid content. Additionally, blood concentrations expressed more simply on a per wet mass or volume basis are more relevant when assessing toxicity as it is easier to compare sea turtle levels to those in laboratory-exposed animals. However, lipid normalization is recommended when comparing concentrations in blood to other more lipid-rich tissues from the same animal. This was done in studies demonstrating that POP concentrations in blood were significantly correlated to those in fat or other internal tissues from the same loggerhead, Kemp's ridley, or green sea turtles (Keller et al., 2004a; van de Merwe et al., 2010a). These results indicated that blood, a less invasive sample, reasonably represents the concentration stored in more routinely analyzed fatty tissues. POPs have been measured in several blood components of sea turtles, including plasma/serum, whole blood, and red blood cells (RBCs). Keller et al. (2004b) and Carlson (2006) showed that POPs preferentially distribute into the plasma rather than the RBC fraction, and linear regressions were provided to convert concentrations measured in one matrix to another.

Early studies analyzed internal organs from stranded animals or eggs for POPs; however, because blood has been proven to be a reasonable tissue for POP measurements, many recent projects are choosing this nonlethal, simple tissue sampling methodology. Additional advantages of using blood over tissues from stranded animals are that a less biased population of live animals can be sampled and that more simultaneous health and biological data can be collected from the same animals. For these reasons, six of the seven sea turtle species have been monitored for blood POP concentrations (Table 11.2); one more species than liver and fat. Blood studies also contain more POP classes than the traditional fat and liver studies, adding PBDEs, PFOS, toxaphenes, and dioxins/furans to the sea turtle literature.

PFOS has been measured exclusively in blood samples from sea turtles, and it holds the record for the highest concentration of a single compound among all POPs measured in sea turtle blood

(Keller et al., 2005b, 2012; O'Connell et al., 2010). This compound, along with other PFCs, differs from the other POP classes because it associates with proteins instead of lipids. Thus, PFCs preferentially distribute into plasma and liver of animals rather than fat, which makes these tissues the target for analysis. An important point to emphasize regarding blood is that POP concentrations in this tissue are being circulated and perfusing internal organs that could be targets for toxicological effects, rather than being stored away in fatty depots.

Eggs are one of the best sampling compartments for measuring POP exposure and have been used extensively to monitor temporal and geographical trends of POPs in birds from Antarctica to the Arctic (Braune et al., 2007; Corsolini et al., 2011). POP concentrations in eggs represent the contamination of the maternal foraging grounds (Alava et al., 2011), because the compounds are transferred into the egg during yolk production, which occurs months before females migrate to nesting beaches often far distances from their foraging grounds. Evidence of maternal transfer comes from the significant correlations between maternal blood POP concentrations and those in her eggs for green and leatherback sea turtles (Guirlet et al., 2010; van de Merwe et al., 2010b; Stewart et al., 2011). Guirlet et al. (2010) also showed that POP concentrations declined with subsequent clutches within a season from the same mother, and the decline was related to a coincident decline in lipid content of the eggs. Leatherback turtles that took 3 years off between nesting also appeared to make eggs with higher POP concentrations than those that only took 2 years (Guirlet et al., 2010). Often people are curious if POPs can transfer across the shell during incubation from contaminated sand. Basile (2010) demonstrated that negligible amounts of POPs transfer from highly contaminated sediments into diamondback terrapin (*Malaclemys terrapin*) eggs and that indeed >98% of the POP concentration within an egg was maternally transferred.

Eggs from the same sea turtle nest have very similar concentrations (Alava et al., 2006; van de Merwe et al., 2010b), whereas clutches from different females show great variability (Table 11.5). Several studies have used unhatched sea turtle eggs after live hatchlings emerge, which makes this egg sampling technique nonlethal (Table 11.5) (Alam and Brim, 2000; Alava et al., 2006, 2011; Mckenzie et al., 1999; Stewart et al., 2011). When using unhatched eggs, it is important to consider the effects of embryonic development on egg POP concentrations. Alava et al. (2006) showed that if only the yolk is measured for POPs, the concentrations within the yolk increase through development mainly because the yolk sac becomes smaller as water is taken up and lipids are concentrated during embryonic growth. Thus, the yolk sac taken from a late stage embryo will not have the same concentration as the yolk taken from an egg without development. Standardized sampling protocols using unhatched eggs should either homogenize the entire egg contents or collect subsamples (e.g., yolk) from only one stage of development. A recent study has shown that unhatched loggerhead eggs have the same concentration as freshly laid eggs from the same nest (Keller, unpublished data).

Since egg sampling is so simple, it is surprising how few studies have used this meaningful packet from sea turtles. Only loggerheads, leatherbacks, and greens have been examined for egg POP concentrations (Table 11.5). Moreover, loggerhead eggs have been sampled from only the southeastern United States aside from one egg from the Mediterranean Sea. Exposure of other nesting loggerhead subpopulations is completely unknown. Ironically, no green sea turtle egg has been analyzed from the southeastern United States since 1976, and the majority of the available egg concentration data for this species now come from Australia and Malaysia.

11.3.2.2.2 Body Condition Influences POP Concentrations

POP levels are known to fluctuate in all tissues during significant weight changes, but most drastically in blood. POPs (except PFCs) are normally found in fatty tissues because they preferentially bind to lipids. During weight loss, these POPs along with associated lipids will move out of fat depots into blood (Chevrier et al., 2000; Debier et al., 2006; Hall et al., 2008). The body utilizes the lipids for energy, but the POPs remain circulating in the blood stream, because these compounds are difficult to metabolize or eliminate even in healthy animals. Thus, the higher circulating POP levels are more available to enter and cause toxic effects to vulnerable target

organs (liver, kidney, brain, gonads, etc.) until foraging resumes and fat stores are replenished. Because POP concentrations can be dramatically affected by body condition, this factor must be considered when interpreting POP concentrations (Ross, 2004). In apparently healthy loggerhead sea turtles and bottlenose dolphins (*Tursiops truncatus*), blood POP concentrations increase as fatty tissue lipid content decreases (Keller et al., 2004a; Yordy et al., 2010b). Likewise, loggerhead turtles from North Carolina with poorer body condition had significantly higher dieldrin blood concentrations (Keller et al., 2004c). Similar correlations were seen between body condition and ΣPCBs in livers from loggerhead turtles that stranded in the Canary Islands (Orós et al., 2009) and plasma HCHs in green turtles from Baja California (Labrada-Martagon et al., 2011). Monagas et al. (2008) grouped dead stranded loggerhead turtles into emaciated and normal body condition to look for differences in fat and liver concentrations of 2,4′-DDTs. Within the emaciated group, liver concentrations were higher than fat concentrations (both on wet mass basis), suggesting that the pesticides had mobilized from fat into other less lipid-rich and more protein-rich tissues. Furthermore, several extremely emaciated and sick loggerhead sea turtles (debilitated syndrome) were found to have nine times higher blood POP concentrations and much lower lipid content in their fat depots compared to their healthy counterparts along the southeastern coast of the United States (Keller et al., 2006a).

11.3.2.2.3 Trophic Status Influences Species Differences

Species differences are apparent and consistent across all tissues (Tables 11.2 through 11.5) with blood and liver samples allowing the most comparisons among species (Tables 11.2 and 11.4). Broad comparisons across all studies support the general conclusion that has been stated in many past sea turtle studies that POP concentrations are highest in Kemp's ridley sea turtles, followed by loggerhead, leatherback, and finally green sea turtles. This ranking follows closely with their trophic status, which is an expected relationship because POPs are known to biomagnify up food webs. Thus, the turtle species with the highest trophic status will accumulate the highest POP concentrations. The diets of the different sea turtle species were reviewed by Bjorndal (1997), and Chapter 9 summarizes dietary analyses including C and N isotopic signatures that characterize trophic and regional feeding. Kemp's ridley sea turtles feed mostly on crabs, while loggerheads are omnivorous carnivores choosing a variety of marine invertebrates, including crustaceans and mollusks. Leatherbacks eat almost exclusively gelatinous zooplankton, and green turtles are primarily herbivores. Future studies should pair contaminant and stable isotope analysis to validate that these species differences are primarily caused by differences in trophic status. Additionally, dietary shifts are known in sea turtles, especially for the green turtle where younger pelagic turtles are omnivores and older neritic stages are herbivores (Bjorndal, 1997). These shifts could influence POP concentrations.

Tissues from olive ridley (*Lepidochelys olivacea*), hawksbill, and flatback sea turtles have been measured so infrequently that it is unwise to make any strong conclusions as to their POP exposure level relative to the other species. However, one olive ridley along Baja California had POP fat and liver concentrations higher than a loggerhead from this region, potentially placing it on a similar exposure level as the related Kemp's ridley sea turtle (Tables 11.3 and 11.4) (Gardner, 2003). In contrast, a more recent paper on levels in liver places the olive ridley nearly equivalent or slightly below the loggerhead sea turtle (Table 11.4) (Richardson et al., 2010). Surprisingly the spongivorous hawksbill sea turtle may turn out to be the most exposed species, as demonstrated by higher PCB concentrations in liver than loggerhead and green sea turtles within Japanese waters (Table 11.4) (Malarvannan et al., 2011). Similarly, one hawksbill blood sample had higher PBDE concentrations than green sea turtles and one flatback turtle from Australia (Hermanussen et al., 2008). These findings from very small sample sizes were supported by recent PFC measurements, in which hawksbill plasma was found to have the highest concentrations of PFOS compared to four other species along the southeastern U.S. coast (Table 11.2) (Keller et al., 2012). With only one sample from flatback sea turtles, too few values are available to determine their placement within the sea turtle ranks.

Along the lines of prey choices, sea turtles are known to ingest plastic trash, probably mistaking it for food. While this causes a mortality risk to sea turtles from entanglement or gastrointestinal blockage, a new concern with this interaction has emerged (Moore, 2008). Plastic polymers are indeed persistent pollutants containing several organic and toxic chemicals, but they also adsorb and concentrate POPs and other hydrophobic contaminants from the water. Ingestion of these plastics could be an additional source of exposure to POPs and other pollutants for sea turtles. This new hypothesis deserves some research attention.

11.3.2.2.4 Sex and Age Class Influences on POP Concentrations

POPs are known to accumulate through the life of animals, increasing with age for males and increasing until reproductive maturity for females, which is especially well documented in marine mammals (Yordy et al., 2010a). Females are known to offload a significant portion of the POP burden into their offspring and this offloading, or maternal transfer, is known for both oviparous and live-bearing reproductive strategies. For sea turtles, all classes of POPs are expected to transfer from mother to egg during yolk deposition into ovarian follicles. This occurs while the female is acquiring and storing energy resources on her foraging grounds and developing her next season's follicles. Thus, the concentrations in eggs represent the contamination of her distant foraging grounds, not from the nesting beach where she ultimately lays her eggs. Thus as a female ages, she is able to offload her POP burden to her eggs, thereby decreasing her tissue concentrations. Males do not have a similar offloading mechanism, so they continually accumulate POPs. It is expected that adult female sea turtles would have lower POP concentrations than adult males, but only one study has ever measured a large enough sample size of adult males to make any comparison (Ragland et al., 2011). Ragland et al. (2011) found that adult male loggerhead plasma had the highest POP concentrations compared to any other age class, sex, or species of sea turtle studied (Table 11.2). During the juvenile and subadult stages, males and females are not expected to have different concentrations as supported by studies on PCBs, OCPs, and PFCs (Keller et al., 2004a, 2005b; Komoroske et al., 2011; Lazar et al., 2011; Malarvannan et al., 2011).

Some studies have examined whether POP concentrations change through age by analyzing sea turtles that span a range of carapace lengths. Unfortunately, live sea turtles cannot be aged, and turtle length, the only proxy for age, has its flaws because the relationship between age and length is quite variable. Nevertheless, observed relationships between length and POP concentrations are inconsistent. Some studies show expected increases with loggerhead turtle length for concentrations of PFCs (Keller et al., 2005b), PCB congeners 52 and 114 (Lazar et al., 2011), and several OCPs and ΣPCBs (Ragland et al., 2011). Other studies show no relationship between turtle length and POP concentrations (Komoroske et al., 2011; Labrada-Martagon et al., 2011; O'Connell et al., 2010; Swarthout et al., 2010), while others show negative correlations for loggerheads (Keller et al., 2004a) but mostly for green turtles (Malarvannan et al., 2011; Mckenzie et al., 1999; Richardson et al., 2010). The latter studies suggest growth dilution instead of accumulation with age, and this is highly likely in green turtles after they shift from being omnivorous as young juveniles to a more herbivorous diet later in the juvenile stage (Bjorndal, 1997). These ontogenetic shifts and the fact that tissue POP concentrations are a culmination of POPs accumulated through all past life stages make interpretation of POPs through age difficult. No study has strategically examined the POP burden of each life history stage of a subpopulation at major locations along its migratory routes.

Few studies have measured POPs in hatchling sea turtles (Aguirre et al., 1994; Mckenzie et al., 1999; van de Merwe et al., 2010b). One large and potentially crucial data gap is the concentrations of POPs in the blood of hatchlings during and after the frenzy period when they have metabolically utilized all the yolk resources. That flush of lipids and its associated POPs during this critical developmental stage in such a small animal could plausibly result in acute toxicity or nonlethal effects that lead to mortality indirectly. Van de Merwe et al. (2010b) measured blood POP concentrations in hatchlings and also in eggs and maternal blood. The hatchling blood samples were collected quickly after emergence from the nest, just before the frenzy period, and had 1.5 times higher mean

concentrations of PCBs than the adult females (Table 11.2). During the rapid use of the absorbed yolk sac in the following few days, the blood concentration of these hatchlings would likely increase dramatically. Thus, it remains to be shown if hatchlings struggling to swim offshore may also face some of the highest POP concentrations among the sea turtle age classes.

11.3.2.3 Nonbiological Factors Influencing POP Concentrations

POPs are known to change across space and time. These nonbiological factors could be examined with a meta-analysis approach if enough quality data were available without major confounding factors mentioned previously. In the next sections an attempt is made to examine spatial and time trends across available studies in Tables 11.2 through 11.5 as well as to highlight a few recent studies that have examined these nonbiological factors directly within a controlled sampling regimen with statistical hypothesis testing.

11.3.2.3.1 Spatial Trends

A global spatial delineation is becoming apparent with newer studies on fat, liver, and eggs (Tables 11.3 through 11.5) showing that loggerhead and green sea turtles in the North Atlantic and Mediterranean Sea have higher exposure to PCBs and OCPs than those sampled along Baja California, Japan, Malaysia, and Australia. More sampling is needed in these and other regions with larger sample sizes along with standardized collection years and analytical methods to make global comparisons more robust.

Several recent studies have directly sought to assess spatial differences in sea turtle POP exposure. These studies have determined that even highly migratory sea turtles can be used as indicator species of marine contamination on a regional scale (Alava et al., 2011; Hermanussen et al., 2006; Keller et al., 2005b; O'Connell et al., 2010; Ragland et al., 2011; Swarthout et al., 2010). These studies selected samples from the same species, age class, and years of collection and analyzed samples in a single laboratory with standardized methods to eliminate confounding factors in order to compare only across locations. The results show significant differences among sampling locations. Hermanussen et al. (2006) linked dioxin concentrations in green sea turtles from Australia to measured concentrations in the abiotic environment (sediments) that the turtles were inhabiting. Most other studies focused on loggerhead sea turtles sampled within the United States, and taken together, they conclude that higher POP concentrations are detected in loggerheads inhabiting regions further north along the U.S. Atlantic coastline (Figure 11.4). These findings suggest that subpopulations utilizing areas of higher contamination (e.g., along the mid-Atlantic and northeastern regions of the United States) accumulate higher concentrations and could be at higher risk for toxic effects. One specific location that deserves more attention is near Brunswick, Georgia. This coastal city hosts three U.S. EPA Superfund sites and its estuary and coastal habitats are contaminated with a unique and highly chlorinated PCB mixture, mercury, and toxaphenes (Balmer et al., 2011; Maruya and Lee, 1998). Preliminary data show that loggerhead turtles captured offshore of Brunswick have a pattern of PCBs primarily composed of higher chlorination groups indicative of exposure from this Superfund site (Keller et al., unpublished data). This is one known hot spot for a mixture of POPs that should be examined for sea turtle exposure and toxic effects.

Among the spatial studies, two U.S. studies have attempted to relate POP concentrations to known migratory pathways of sea turtles. Loggerhead turtles nesting in North Carolina had higher concentrations of PCBs, OCPs, and PBDEs, represented in their eggs, than loggerheads nesting on the east or west coast of Florida (Alava et al., 2011). Using previously published satellite tracks of females nesting in these three regions, Alava et al. (2011) showed that North Carolina nesters forage in areas further north than those from Florida. Likewise, Ragland et al. (2011) measured POP concentrations in plasma of adult male loggerheads that were satellite tagged during the mating season off of Cape Canaveral, Florida. After the mating season, two major migratory pathways were evident. The residents, those males that stayed near Cape Canaveral into and through the

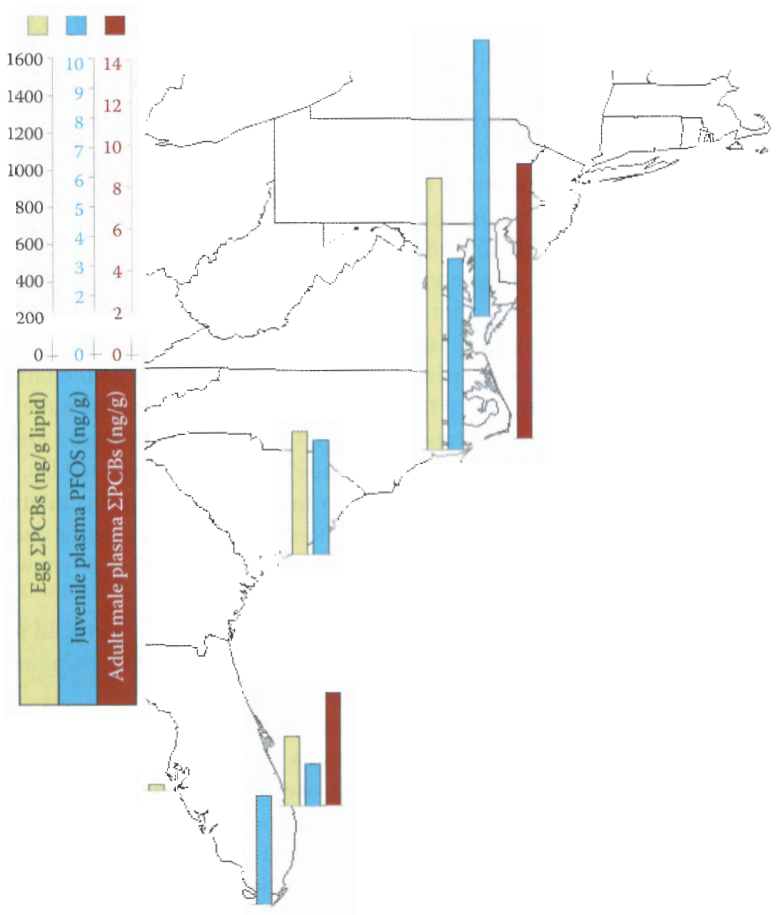

FIGURE 11.4 Spatial comparison of average persistent organic pollutant (POP) concentrations in loggerhead sea turtle tissues along the coast of the United States. Data were taken from Alava et al. (2011) and Keller (unpublished data) (yellow), O'Connell et al. (2010) (blue), and Ragland et al. (2011) (red).

winter, had lower POP concentrations than the transients that chose to forage in northern regions off of South Carolina to New Jersey. Their choice of foraging habitat influenced their POP exposure, and these satellite tracking studies provide critical information about previously unknown foraging habitat selection.

11.3.2.3.2 Temporal Trends

Temporal trend studies are very important for monitoring changes in POP concentrations after the onset of manufacture or restriction in use of a compound. POP concentrations have been shown to change through time in the environment, but trends are often species and location dependent (Tuerk et al., 2005). Using Tables 11.2 through 11.5, it is very difficult to extract time trends in sea turtle tissues. Blood sampling has only occurred in the last decade, so no across-study comparisons are available (Table 11.2). Kemp's ridley fat and liver collected from stranded animals in the northeastern United States generally show a decline in PCBs and OCPs from 1985 to 2005 (Tables 11.3 and 11.4) (Innis et al., 2008; Keller et al., 2004a; Lake et al., 1994; Rybitski et al., 1995). Likewise, PCB and 4,4′-DDE concentrations seem to have declined in loggerhead fat from Virginia-North Carolina from 1991–1992 to 2000–2001 (Table 11.3) (Keller et al., 2004a; Rybitski et al., 1995) and in loggerhead eggs from eastern Florida between 1976 and 2002 (Table 11.5) (Alava et al., 2011; Clark and Krynitsky, 1980).

Aside from these general comparisons, specimen banks, or well-maintained archives of samples collected over several years and stored appropriately, are excellent resources of samples for assessing temporal trends of environmental pollutants. Two studies have made use of informal banks of sea turtle plasma to directly test temporal trends in POP concentrations (Carlson, 2006; O'Connell et al., 2010). Concentrations of PCBs, OCPs, and PBDEs were assessed in five juvenile loggerhead plasma samples per year from Core Sound, North Carolina, from 1998 to 2006 (Carlson, 2006). Respective mean concentrations by year in pg/g wet mass were: 2340, 6130, 2950, 2730, 1440, 8710, 3030, 1150, and 9580 for ΣPCBs; 251, 159, 182, 262, 117, 663, 171, 117, and 312 for 4,4′-DDE; and 156, 42.0, 65.7, 63.7, 10.5, 139, 90.6, 14.8, and 62.4 for ΣPBDE (data not shown in Table 11.2). No significant temporal trends were observed for any of these POPs. While declining trends might be optimistically anticipated for PCBs and DDTs, which were banned from use in the United States in the 1970s, the lack of trend is not too surprising. In other U.S. locations, like the Great Lakes, PCB concentrations in fish exponentially declined from the 1970s until 1990, but then the decline slowed (Stow et al., 2004). Thus, this nine-year sampling design for sea turtles centered around 2000 may be too late or too short to detect a trend. During this study's sampling of sea turtles, PBDE use was ongoing in the United States until certain states began to restrict certain formulations in the late 2000s; thus, the stable PBDE concentrations indicate no significant increase or decrease into this sea turtle habitat during this time period. Future samples would be expected to show lower concentrations of all three POP classes.

Another study measured PFC concentrations in 10 or more juvenile loggerhead plasma samples per year from offshore of Charleston, South Carolina, from 2000 to 2008 (O'Connell et al., 2010). Respective median PFOS concentrations by year in ng/g wet mass were 9.43, 7.11, 7.24, 6.35, 2.10, 1.26, 2.99, 1.91, and 3.07 (data not shown in Table 11.2). These data indicated a significant decline in PFOS over this time period, and more importantly, a successful and positive environmental response within sea turtle habitat quickly after PFOS use was discontinued around 2001. These temporal trend studies are good examples of using a specimen bank, and sea turtle contaminant and health research will benefit from a longer-term, formal specimen banking program, similar to the Marine Turtle Molecular Research Collection at the Southwest Fisheries Science Center or the Marine Environmental Specimen Bank at the National Institute of Standards and Technology (Pugh et al., 2008; Serra-Valente et al., 2010). The latter of which is developing a sea turtle specimen bank project called the Biological and Environmental Monitoring and Archival of Sea Turtle tissues (BEMAST).

11.3.3 Sea Turtle POP Exposure Compared to Other Wildlife and as Food for Humans

In comparison to other marine/coastal wildlife, sea turtle POP concentrations are often orders of magnitude lower than those in sharks, alligators, seabirds, and marine mammals from similar locations (Bazan, 2011; Blus et al., 1974; Harper et al., 1999; Heinz et al., 1991; Houde et al., 2006; Yordy et al., 2010b). This ranking is seen in blood as well as eggs for PCBs and pesticides (Figures 11.5 and 11.6). For PFCs, few data are available from plasma of marine wildlife from the southeast U.S. coast for comparison, except for bottlenose dolphins (Houde et al., 2005). Dolphins from Charleston, South Carolina, in 2003 had a geometric mean PFOS concentration of 1171 ng/g wet mass in their plasma (Houde et al., 2005), compared to 6.35 ng/g wet mass, the median in loggerhead turtle plasma from the same location and year (O'Connell et al., 2010). These differences are due mainly to different trophic levels. Most of these other species are fish eaters, except alligators, which eat a variety of vertebrates, placing their trophic status higher than any sea turtle species. One exception to this common ranking is that dioxin/furan toxic equivalencies (TEQs or summed concentrations of dioxins and furans that are weighted based on the toxicity of the individual compounds) in green turtles in more polluted sites within Moreton Bay, Australia were found to be higher than TEQs measured in some marine mammals from locations often considered to be

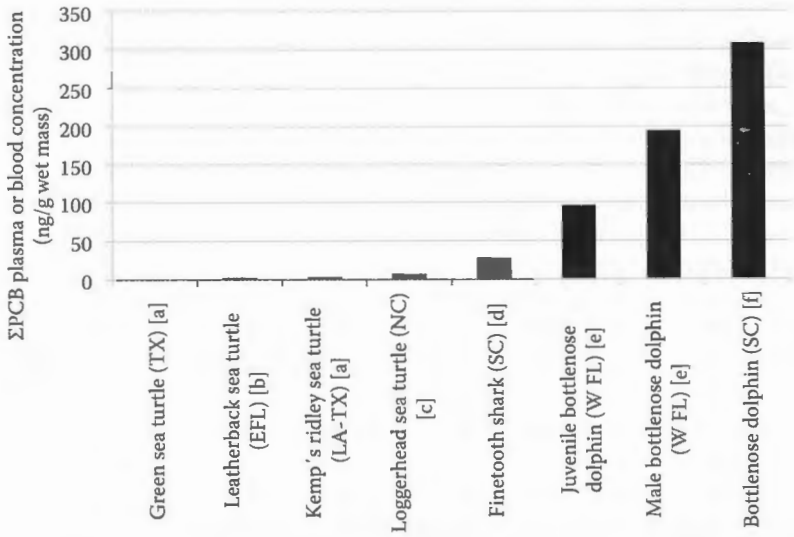

FIGURE 11.5 Comparison of average blood or plasma concentrations of total PCBs among sea turtle species and other coastal marine organisms. Location abbreviations are TX=Texas, E FL=eastern Florida, LA=Louisiana, NC=North Carolina, SC=South Carolina, W FL=western Florida. Data from [a] Swarthout et al. (2010), [b] Stewart et al. (2011), [c] Keller et al. (2004a), [d] Bazan (2011), [e] Yordy et al. (2010b), and [f] Houde et al. (2006).

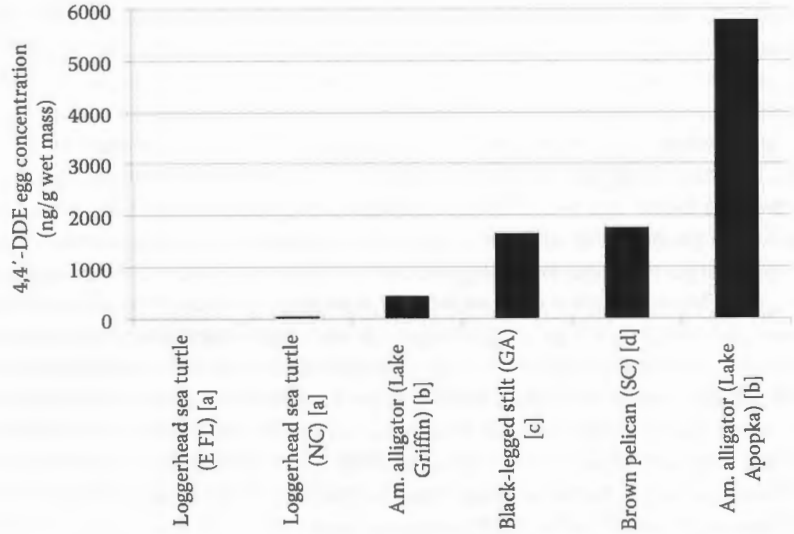

FIGURE 11.6 Comparison of average egg concentrations of 4,4′-DDE among sea turtle species and other organisms from the Southeastern United States. Location abbreviations are E FL=eastern Florida, NC=North Carolina, GA=Georgia, SC=South Carolina. Data from [a] Alava et al. (2011), [b] Heinz et al. (1991), [c] Harper et al. (1999), and [d] Blus et al. (1974).

much more polluted, like the Mediterranean Sea (Hermanussen et al., 2006). This study emphasizes the need to examine sea turtle POP exposure in more urbanized bays and harbors.

Two papers have addressed POP concentrations in sea turtle tissues as an issue for human consumption (Aguirre et al., 2006; van de Merwe et al., 2009a). Green turtle eggs from Malaysian markets had concentrations of coplanar PCBs that exceed the acceptable daily intake limits established

by the World Health Organization (WHO) by threefold or more (van de Merwe et al., 2009a). The coplanar PCBs (PCBs 77, 126, and 169) are the PCB congeners most like dioxin and contribute the most to TEQs, but they are difficult to measure with accuracy. Often other PCB congeners interfere and lead to overestimation of the concentrations, but these authors showed in a separate paper that their analytical methods only overestimated these congeners by approximately 35% in a fish tissue Standard Reference Material (van de Merwe et al., 2009b). Therefore, even after correcting for this overestimation, the minimum egg would still have concentrations higher than the WHO limit. Aguirre et al. (2006) reviewed the literature on environmental contaminants in sea turtle meat (muscle) and eggs and found that these sea turtle tissues have OCPs and PCBs within the range of or exceed safe exposure limits set by the WHO and the U.S. Agency for Toxic Substances and Disease Registry. They note that sea turtle products contain lower levels than some other seafood choices, but developmental effects of POPs are a concern even at these levels, especially for nursing mothers and children. These studies emphasize that POP accumulation in sea turtles is a concern, despite the fact that sea turtles generally have lower POP concentrations than other higher trophic level marine species.

11.4 TESTING THE TOXICITY OF POPS IN SEA TURTLES

While other marine species have higher POP concentrations than sea turtles, and many of their populations are doing very well despite their exposure, the sensitivity of sea turtles to toxic effects of POPs is not well understood. Testing for the toxicity, or determining the adverse biological effects of exposure to a chemical pollutant, can be done using numerous experimental designs that range in their ability to definitively document toxic effects. The most definitive tests use direct laboratory exposure of the species in question to a known and administered dose of a chemical. These are rare in the sea turtle literature because of their conservation status and the ethical/legal issues of sacrificing or intentionally harming endangered sea turtles. Only one study has used this approach for testing sea turtles' sensitivity to POP effects. Podreka et al. (1998) applied 4,4′-DDE to green sea turtle eggshells, incubated the eggs at temperatures that are known to produce males, and found that it did not cause embryonic sex reversal. They applied 4,4′-DDE concentrations of 3,300–66,500 ng to the shell, resulting in egg concentrations up to 543 ng/g, which exceeded the maximum ever measured in any sea turtle egg (see Table 11.5). Thus, they concluded that lower concentrations should not present a problem for sea turtle sexual determination, as well as other measured endpoints, including incubation time, hatching success, incidence of body deformities, and hatchling length and weight. This conclusion may have been premature because Willingham (2004) showed that lower doses of 4,4′-DDE (7 ng applied) caused more sex reversal in red-eared slider turtles (*Trachemys scripta*) than higher doses (28 ng). This suggests that this endocrine disruptive effect follows a hormetic dose–response curve (upside down U shape), which is very common in toxicology (Calabrese and Blain, 2011) and that Podreka et al. (1998) may have used a dose too high to observe an effect in green turtles. Podreka et al. (1998) also did not assess other toxic endpoints that might be more sensitive. Future studies should focus on the developing immune system, other parts of the endocrine system like thyroid hormones, or the nervous system, all of which disrupted at such an early developmental stage could cause lasting and profound effects on future survival or reproduction (Willingham and Crews, 2000).

Another study used the direct laboratory exposure approach to assess the effects of crude oil, which is not a POP but is worth mentioning here. Lutcavage et al. (1995) exposed juvenile loggerhead sea turtles to a layer of crude oil on the surface of the water in their tanks and demonstrated that the oil caused skin necrosis, dermal hemorrhage, and extensive inflammatory cells infiltrating the epidermis, as well as salt gland dysfunction and hematological changes. Aside from these direct exposures, other studies use less definitive approaches.

Another possibly more acceptable approach is to expose captive sea turtles to wild-caught prey items that come from a more contaminated area. This approach has not been used for sea turtles,

but was modeled by a chronic study on POPs in which captive harbor seals (*Phoca vitulina*) were fed fish caught for human consumption from two different ocean basins, one more heavily contaminated than the other (Ross et al., 1996). The seals fed the more contaminated fish exhibited suppression of several immune functions, including natural killer cell activity, lymphocyte proliferation, delayed type hypersensitivity, and antibody responses. Differences in nutritional content and other non-POP contaminant concentrations in the two batches of fish created difficulties in linking immunosuppression with solely POP exposure.

11.4.1 Surrogate Species Toxicity Testing

Examining the effects of POPs on surrogate species is commonly used to test the toxicity of chemicals to set exposure limits for humans, who like sea turtles cannot usually be directly exposed to known harmful chemicals. Laboratory mammals, like mice or rats, are exposed to the chemical using the route of exposure expected for humans and then examined for toxic endpoints ranging from the most severe/least sensitive (death) to the more subtle/most sensitive (e.g., neurological, immune, endocrine, or developmental toxicity). Because each species, as well as different sexes or races, have different sensitivities to a chemical, a safety factor of 100 or more is commonly used to divide the concentration just below the lowest concentration that causes the toxic effect in a surrogate species (the no adverse effect level or NOAEL) to calculate a safe margin of exposure, or a safe exposure limit, for the species of concern (human or sea turtle). This safety factor is quite arbitrary and some may argue is not safe enough. One review provides reasons against a safety factor of 100 (McConnell, 1985); the toxic concentration of dioxin on six commonly used laboratory mammal species ranged four orders of magnitude; which would require a safety factor of 1000 to account for the species difference between the guinea pig and the hamster (McConnell, 1985), and that is among only mammals. It is important to mention that turtles are considered to be highly sensitive to the effects of POPs (Bishop and Gendron, 1998; Sheehan et al., 1999). Surrogate species proposed for predicting toxic effects in sea turtles include the diamondback terrapin because they are aquatic turtles that use the same unique organ, a salt gland, for tolerating ingestion of saltwater and can be reared in captivity with success (Keller et al., 2006c). The red-eared slider, although a freshwater turtle, is another good option because of their commercial availability and their extensive use in reptilian toxicology research (Willingham and Crews, 2000). The surrogate species approach has been used intentionally only once to determine POP toxicity to sea turtles. This study exposed mice to PFOS at concentrations known to be in plasma of three species of interest (sea turtles, dolphins, and humans) (Peden-Adams et al., 2008). Male mice exhibited immune suppression (T-cell dependent IgM antibody response) at concentrations found in all three species. This lack of a margin of safety suggests that if sea turtles are as sensitive as mice, then they too might have suppressed immune systems because of their environmental exposure to PFOS. Margins of safety were assessed for five sea turtle species to investigate their risk of toxic effects based on their PFOS exposure and known effects in laboratory species on the liver, neurodevelopment, endocrine, and immune systems (Keller et al., 2012). Several margins of safety were <100, including all species for immune suppression, indicating possible risk. Aside from these studies, a wealth of literature exists on the toxic effects of POPs on red-eared sliders and other reptiles that could be used for a risk assessment for sea turtles. The collective conclusions from these studies are that reptiles are indeed quite sensitive to the effects of POPs and a multitude of toxic effects have been documented ranging from altered growth rates, sex reversal, and altered hormone concentrations (Sheehan et al., 1999; Willingham and Crews, 2000).

11.4.2 In Vitro Toxicity Testing

In vitro study designs are yet another laboratory approach that allow for direct exposure of the target organism's cells to a pollutant, but without using the whole animal. Because only one or a

few cell or tissue types are present in these experiments, it is questionable whether the results are representative of what would occur in the whole animal. Aside from this disadvantage, in vitro approaches provide the ability to answer mechanistic questions at the tissue, cellular, or subcellular level. At least three studies have used an in vitro approach to assess POP effects on sea turtles (Ikonomopoulou et al., 2009; Keller and McClellan-Green, 2004; Keller et al., 2006b). Keller et al. (2006b) exposed loggerhead peripheral blood leukocytes in culture to PCBs and 4,4′-DDE at concentrations that included known blood concentrations in this species. The lymphocytes in that cell mixture were tested for their ability to be stimulated by mitogens and begin proliferation, which is an early step in immune reactions. They found that both POPs enhanced lymphocyte proliferation relative to the nonexposed control cells at approximately 10 ng/mL total PCBs and 1 ng/mL 4,4′-DDE as the lowest observed adverse effect level (LOAEL). These doses were strikingly similar to the blood concentrations in wild-caught chronically exposed loggerhead turtles with the maximal lymphocyte proliferation response and supported correlative findings also reported in that study. Keller and McClellan-Green (2004) assessed the effects of OCPs on cytochrome P450 aromatase (an enzyme that converts testosterone to estradiol and is thus important for reproductive development and function) in an in vitro system using a green turtle testis cell line. Atrazine is a pesticide not categorized as a POP but has endocrine-disrupting activity like many POPs. It significantly induced aromatase activity in the cells. 4,4′-DDE inhibited activity but only at the highest tested concentration (31,000 ng/mL), which was cytotoxic to the cells and more than two orders of magnitude higher than concentrations found in the fat of sea turtles. Ikonomopoulou et al. (2009) assessed the ability of 4,4′-DDT, 4,4′-DDE, and dieldrin to affect the binding affinity of sex-steroid binding proteins in the plasma of nesting green turtles to testosterone and estradiol. In the two different assays performed, dieldrin and 4,4′-DDT affected the ability of binding proteins to associate with the steroids, but sometimes in unexpected ways. These effects occurred at concentrations of 1000 ng/mL or more, which is three orders of magnitude higher than maximum concentrations measured in sea turtle blood samples (Table 11.2).

11.4.3 Comparative or Correlative Toxicity Field Studies

Alternative approaches not requiring laboratory exposures, including comparative or correlative field studies, are the most common toxicity studies for sea turtles. These approaches take advantage of exposures in the wild and either (1) compare the exposure level of two or more groups that have different health statuses, (2) compare the health of two or more groups that have different levels of exposure, or (3) use correlative relationships to compare individuals within the same population that have varying levels of exposure or health status. The number of sea turtle studies using this type of approach is still too few to make concrete conclusions about whether the health of sea turtles in the wild are being affected by POP exposure, but they provide early evidence to assess whether a cause-and-effect relationship exists (as part of a weight of evidence approach) (Fox, 1991). The first study to attempt this approach with sea turtles was performed by Aguirre et al. (1994) in which tissues from 10 green turtles with fibropapillomatosis from Hawaii were analyzed for POPs with the objective to compare them to 2 green turtles without tumors. Unfortunately, the limits of detection were too high to detect any POPs and no comparison could be made. Sixteen years later, no study has yet answered the question of whether POPs or other environmental contaminants contribute to the incidence or severity of this debilitating disease. This speculation is plausible, especially since POPs affect the immune system and could make it more difficult for turtle immune cells to detect and destroy viruses or tumor cells.

A decade later, Keller et al. (2004c, 2006b) examined approximately 48 wild-caught juvenile loggerhead sea turtles from Core Sound, North Carolina, for PCB and OCP concentrations in blood and fat biopsies, while also measuring a suite of health parameters. Most turtles appeared healthy based on external exams, plasma chemistry panels, and hematology. However, concentrations of several POPs, depending upon the compound and tissue, significantly correlated with many health

parameters. Significant positive correlations were observed for total white blood cell counts, the heterophil:lymphocyte ratio (Keller et al., 2004c), and the ability of B- and T-lymphocytes to proliferate after mitogen stimulation (Keller et al., 2006b); negative correlations were observed for plasma lysozyme activity (Keller et al., 2006c). These findings suggest that POPs could be altering the number of immune cells in the peripheral blood and affecting the way those cells respond to non-self antigens. The positive correlation with the heterophil:lymphocyte ratio indicates that turtles with higher POP exposure may respond physiologically as if they are experiencing more stress, and the negative correlations with lysozyme suggests that more highly exposed turtles are producing less antibacterial enzymes, possibly leaving them more susceptible to infections. Aside from immunology, significant positive correlations were seen between POP concentrations and blood urea nitrogen (BUN), aspartate aminotransferase (AST), and plasma sodium concentrations (Keller et al., 2004c). Significant negative correlations were observed for body condition, plasma glucose, albumin, albumin:globulin ratio, alkaline phosphatase (ALP) activity, gamma glutamyl transferase (GGT) activity, magnesium, and markers of anemia (hemoglobin, hematocrit and red blood cell counts) (Keller et al., 2004c). The authors concluded from these correlations that exposure to environmental PCB and OCP concentrations may affect a wide variety of biological functions in loggerhead sea turtles, including immunity, organ health, and homeostasis of proteins, carbohydrates, and ions. What is most striking about these correlations is that they were seen in turtles that appeared mostly healthy and were foraging in a fairly pristine location, supporting prior conclusions that turtles may be exquisitely sensitive to POPs (Bishop and Gendron, 1998; Sheehan et al., 1999).

Three recent studies have repeated this type of study while focusing on sea turtle populations that are more highly exposed. Correlative studies have been performed for juvenile Kemp's ridley sea turtles from the Gulf of Mexico (Swarthout et al., 2010), adult male loggerheads captured near Cape Canaveral, Florida (Ragland et al., 2011), and green turtles from San Diego Bay, California (Komoroske et al., 2011). In all three, blood POP concentrations were similar or higher than the loggerhead study from North Carolina (Table 11.2). In approximately 20 Kemp's ridley turtles, significant negative correlations were seen between ΣDDT blood concentrations and T-lymphocyte proliferation, suggesting immunosuppression in the more exposed turtles (Swarthout et al., 2010). This correlation is in the opposite direction as was seen in loggerhead sea turtles, indicating that more research is needed to understand how POPs modulate the sea turtle immune system. Significant positive correlations were noted between BUN and Σchlordane concentrations (similar to the finding in loggerhead turtles by Keller et al. [2004c]) and between ΣDDTs and plasma potassium concentrations. Significant negative correlations were observed between ΣPCBs and creatine phosphokinase (CPK) activity, which is an unexpected direction similar to the correlations seen for ALP and GGT in loggerheads, and between dieldrin and testosterone concentrations in female turtles. Some of these correlations were similar to the previous loggerhead study, while others differed. Differences could be due to the smaller sample size of this Kemp's ridley study or caused by species or location differences. In the 19 adult male loggerheads assessed by Ragland et al. (2011), no significant correlations were observed between plasma ΣPCBs, ΣDDTs, Σchlordanes, or ΣPBDEs and any hematological or plasma chemistry value. It is difficult to determine why no correlations were observed as these turtles were the most exposed group of sea turtle ever monitored by blood sampling, but the sample size could have limited the power to detect relationships. In addition, nothing is known about normal ranges of these health indicators in adult male sea turtles, let alone how those values might change during the mating season, which is when these males were sampled. Understanding the toxic effects of POPs on male reproductive success is important especially if climate change results in warmer nest temperatures, skewing the sex ratio toward fewer males. Temperature is the most important factor for sex determination in developing sea turtles, but hormones and hormone disruptors like POPs also play a role. In 20 green turtles from San Diego Bay, γ-HCH, *trans*-chlordane, and 4,4′-DDE plasma concentrations correlated with a few health parameters (γ-HCH negatively

with eosinophils and total protein and positively with ALP activity and albumin:globulin ratio; *trans*-chlordane negatively with heterophils; and 4,4′-DDE positively with hematocrit, albumin and uric acid). None of these correlations are similar in endpoint or direction as compared to the findings of Keller et al. (2004c), and could indicate differences in sensitivity among sea turtle species. Again, it is important to note that correlative relationships cannot prove cause and effect, and the lack of consistency among studies could be due to confounding factors that affect health endpoints or POP concentrations. Alternatively, some relationships could be due to statistical chance; most significant correlation coefficients were above 0.4, but the full range (0.18–0.79) included some weak correlations.

Peden-Adams et al. (2005) presented correlations between loggerhead health parameters and concentrations of plasma PFOS and other PFCs. According to them PFOS significantly and positively correlated with T-lymphocyte proliferation, AST activity, and concentrations of plasma globulin, glucose, potassium, total protein, and BUN. Likewise, PFOS significantly and negatively correlated with plasma lysozyme activity. These correlations suggest PFOS may be modulating the immune system, damaging liver or other tissues, and altering other plasma chemistries. Since these specific effects have been documented in laboratory animals exposed to PFOS, it is plausible that environmental exposure to PFOS may be causing these effects in loggerhead turtles. Future studies should focus on other sea turtles that have even higher PFOS exposure, like Kemp's ridleys and hawksbills (Table 11.2).

Another correlative study measured PCB concentrations in liver and fat of stranded loggerhead turtles in relation to body condition and bacterial infections (Orós et al., 2009). Liver concentrations were significantly higher in turtles with poorer body condition, as discussed in a previous section regarding lipid mobilization. The PCB concentrations were not significantly correlated to septicemia (defined by the authors as multiple bacterial infections found throughout the body).

Similar to the study on green turtle FP and POPs, a study addressed whether POP exposure could contribute to another sea turtle disease or syndrome. Loggerhead debilitated syndrome, identified as turtles with extreme emaciation, lethargy, and a heavy barnacle cover on skin, was on the rise in the early 2000s along the southeast coast of the United States. These debilitated turtles suffer from a range of secondary bacterial and parasitic infections (Norton et al., 2005). Turtles in this condition were measured for fat and blood POP concentrations and compared to apparently health loggerhead turtles (Keller et al., 2006a, 2007). PCB and OCP concentrations on a lipid-normalized basis in fat and on a wet mass basis in blood were approximately seven to nine times higher in these turtles than the healthy turtle group. The suggestion that POPs contributed to the onset of this syndrome was discounted after considering lipid mobilization of POPs. As debilitated turtles regained fat stores during rehabilitation, their plasma POP concentrations decreased to concentrations typically observed in healthy turtles, likely sequestering the POPs back into fat stores (Keller et al., 2007). This finding suggests that these turtles had average POP concentrations before they became ill. During recovery, plasma chemistries and hematology coincidentally improved as plasma POP concentrations decreased, so it is possible that higher POPs in blood due to mobilization from fat during weight loss contributed to the progression of this syndrome. Robust statistical analyses of these data are ongoing.

One correlative study has examined POP exposure and possible effects on hatchling green sea turtles (van de Merwe et al., 2010b). The authors found that among seven nest success parameters, only hatchling body condition (mass/SCL) was negatively related to ΣPOP egg concentrations. Hatching success, emergence success, percentage of abnormal hatchlings, as well as hatchling mass, length, and abnormality index were not correlated to POP concentrations. If the observed correlation is causative, meaning exposure to POPs in the egg are causing hatchlings to have lower body condition, then the more highly exposed hatchlings could be less fit during the swim out to sea and more susceptible to predation because of their thinner body condition. Much more research like this study is needed to address very possible lethal and sublethal effects of POPs on the sensitive early life stages of sea turtles.

11.4.4 Toxicological Biomarker Studies

Within only the last 5 years, studies have begun to look at traditional toxicological biomarkers in sea turtles, primarily enzymes that combat oxidative stress and a protein that indicates endocrine disruption (Casini et al., 2010; Labrada-Martagon et al., 2011; Richardson et al., 2009, 2010; Valdivia et al., 2007; Zaccaroni et al., 2010). Richardson et al. (2009) confirmed the presence and measured the activity of glutathione *S*-transferase (GST) enzyme in livers of four species (loggerhead, green, olive ridley, and hawksbill sea turtles). GST is an enzyme that is commonly upregulated in animal tissues when antioxidant defenses are needed, such as during oxidative stress when an animal is attempting to detoxify or biotransform a chemical pollutant. Hawksbill turtles had the highest GST activity, which is intriguing since this species feeds on sponges that create toxic natural chemicals and has been shown preliminarily to be the highest exposed sea turtle species to POPs (Hermanussen et al., 2008; Keller et al., 2012; Malarvannan et al., 2011). A follow-up study measured PCB concentrations in these animals and looked for correlations between the PCBs and activity of GST as well as expression of cytochrome P450 enzymes (Richardson et al., 2010). Many cytochrome P450 enzymes are commonly upregulated when an organism is exposed to certain POPs, like dioxins and PCBs, in order to attempt the first phase of metabolism and ultimately to excrete the compounds. While Richardson et al. (2010) was able to confirm the expression of CYP2K and CYP3A in three species of sea turtles, they could not detect CYP1A, which is the most commonly upregulated cytochrome P450 enzyme following exposure to PCBs and dioxins. GST activity and cytochrome P450 enzyme expression did not correlate significantly with PCB concentrations. It is important to note that these studies were meant primarily to characterize and develop the methods to measure these biomarkers, and sample sizes were small so statistical power was probably low to detect relationships.

A second set of studies to address traditional toxicological biomarkers focused on oxidative stress in green turtles from Baja California (Labrada-Martagon et al., 2011; Valdivia et al., 2007). Valdivia et al. (2007) tested methods to measure oxidative stress in livers, particularly superoxide radical production, lipid peroxidation, and activities of several antioxidant enzymes. Labrada-Martagon et al. (2011) expanded this work by comparing OCP concentrations in plasma of green turtles from two sites as well as lipid peroxidation and antioxidant enzyme activity levels in the red blood cells from the same animals. Turtles from the Punta Abreojos site, where a larger sample size of 35 was collected, showed several significant correlations. Elevated lipid peroxidation is an indicator of oxidative stress, in which reactive oxygen species are being created at destructive levels. Exposure to chemicals, as well as other environmental stressors like UV or heat, can cause oxidative stress and increase lipid peroxidation. Lipid peroxidation was negatively correlated with β-HCH concentrations, which is the opposite from the expected direction and difficult to interpret, but positively correlated with aldrin and ΣDDT concentrations. GST activity levels in the red blood cells were positively correlated to all three HCH isomers, ΣHCHs, and Σheptachlor concentrations measured in the plasma. Likewise, catalase (CAT), glutathione peroxidase (GPx), and superoxide dismutase (SOD) enzymes are commonly upregulated in tissues during oxidative stress. Few of these enzymatic activities correlated with POP levels in the green turtles, except CAT correlated positively with endrin sulfate. At the other site (Bahía Magdalena), different correlations were seen with a smaller sample size of 11 turtles. *Trans*-chlordane was significantly and positively correlated with lipid peroxidation, GST, CAT, and GPx. ΣHeptachlors were positively correlated with lipid peroxidation, and ΣDDTs were correlated with CAT. Most of these correlations are positive and suggest that the more highly exposed turtles to OCPs have greater oxidative stress and are expressing a stronger antioxidant enzyme response.

Several sea turtle studies have measured vitellogenin (VTG), a yolk protein precursor produced in the liver normally only by adult oviparous females in response to estradiol, which then circulates in the blood and is deposited into egg yolk. VTG expression is possible in juveniles and males when they are exposed to estrogen or an estrogen-mimicking compound, and this has been

documented in juvenile sea turtles after exposure to estradiol (Heck et al., 1997). In red-eared sliders, 2,4′-DDT and PCBs can mimic estrogen and turn on the expression of VTG in males (Palmer and Palmer, 1995; Smelker and Valverde, 2012), verifying that VTG can be a biomarker of estrogenic exposure. Zaccaroni et al. (2010) measured this protein biomarker in plasma of 61 juvenile to adult loggerhead turtles of both sexes from Italy with the goal of using it as a biomarker of exposure to endocrine-disrupting contaminants. A percentage of loggerhead turtles considered juveniles were expressing this protein, concluding that they were either precocious or had been exposed to an estrogenic compound. However, no contaminants were measured in these animals for comparison. Similarly, a study that screened over 400 loggerheads from North Carolina also discovered precocious females as well as a male (confirmed by laparoscopy) expressing VTG (Keller et al., 2003). POP concentrations measured in some of these animals were on the higher end of loggerhead exposure (Keller, unpublished data), but a statistical analysis of the data has not yet been performed.

Casini et al. (2010) have tested a suite of nonlethal biomarkers using blood and skin biopsies from live loggerhead turtles from the Mediterranean Sea. They have shown cytochrome P450 1A induction in blood lymphocytes and skin biopsies exposed in vitro to POPs. Additionally, they are investigating correlations between POPs and plasma lipid peroxidation, genotoxic effects in whole blood using the comet assay and erythrocytic nuclear abnormalities, plasma liver enzyme activities, VTG, and butyrylcholinesterase activity.

11.5 CONCLUSIONS

This review leaves no doubt that sea turtles are exposed to POPs, some species and locations more so than others. While this field has been exponentially growing since 1974, large data gaps still exist in understanding POP exposure to sea turtles globally, and future studies need to improve sample sizes and use analytical approaches with appropriate sensitivity and that assure quality data. POP concentrations in sea turtles are driven by the same factors that influence POP accumulation in other species, such as lipid content, trophic status, age, and sex. Interesting spatial and temporal trends in POP concentrations are emerging in sea turtle studies, and tissues stored in formal specimen bank programs would allow for more of these types of studies.

For decades, sea turtle researchers and conservationists have suspected that environmental contaminants cause or contribute to several sea turtle health issues and diseases, ranging from green turtle fibropapillomatosis (Aguirre and Lutz, 2004; Foley et al., 2005; Herbst and Klein, 1995) to low hatch success in leatherback sea turtles (Bell et al., 2003). However, too few studies have directly examined these questions, and preliminary results are weak, inconsistent, or just correlative, emphasizing that much more research is warranted. One important and consistent confounding factor shown in at least three sea turtle studies is that body condition affects POP concentrations in ways that might confuse conclusions regarding causes of health issues (Keller et al., 2004a, 2007; Orós et al., 2009). Future studies should always consider this factor from initial sampling design to final data interpretation. Finally, the preliminary studies showing correlations between POP concentrations and health parameters or toxicological biomarkers are the first line of evidence suggesting that chronic environmental exposure to POPs could be causing sublethal effects on sea turtle populations. These studies pave the way for future more directed studies that should strive to include endpoints that are important to conservation managers, such as the vital rates of survival at each stage, growth at each stage, and reproductive output by both sexes. These kinds of data would allow managers to understand the relative risk of POP effects to other better known threats, like fisheries bycatch and harvest for meat or eggs. As a top priority, toxicity testing should be focused on sensitive life stages as well as subpopulations that are more exposed. For example, a study could assess POP effects on mortality and fitness of loggerhead hatchlings from North Carolina or the Mediterranean Sea, where they are more exposed. This is just one example of the vast research gaps available to scientists eager to approach this field.

DISCLAIMER

Certain commercial equipment or instruments are identified in the chapter to specify adequately the experimental procedures. Such identification does not imply recommendations or endorsement by the NIST nor does it imply that the equipment or instruments are the best available for the purpose.

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