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Pollutants and the health of green sea turtles resident to an urbanized estuary in San Diego, CA

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ABSTRACT

Rapid expansion of coastal anthropogenic development means that critical foraging and developmental habitats often occur near highly polluted and urbanized environments. Although coastal contamination is widespread, the impact this has on long-lived vertebrates like the green turtle (*Chelonia mydas*) is unclear because traditional experimental methods cannot be applied. We coupled minimally invasive sampling techniques with health assessments to quantify contaminant patterns in a population of green turtles resident to San Diego Bay, CA, a highly urbanized and contaminated estuary. Several chemicals were correlated with turtle size, suggesting possible differences in physiological processes or habitat utilization between life stages. With the exception of mercury, higher concentrations of carapace metals as well as 4,4′-dichlorodiphenyldichloroethylene (DDE) and γ chlordane in blood plasma relative to other sea turtle studies raises important questions about the chemical risks to turtles resident to San Diego Bay. Mercury concentrations exceeded immune function *no-effects* thresholds and increased carapace metal loads were correlated with higher levels of multiple health markers. These results indicate immunological and physiological effects studies are needed in this population. Our results give insight into the potential conservation risk contaminants pose to sea turtles inhabiting this contaminated coastal habitat, and highlight the need to better manage and mitigate contaminant exposure in San Diego Bay.

1. Introduction

Nearshore ecosystems support high levels of biodiversity across a wide range of taxa (Gray, 1997). However, many coastal areas are also subject to intense human activities that can severely degrade habitat quality. Resulting effects on coastal species are difficult to quantify because reduced habitat quality rarely leads to immediate mortality and may take years to manifest in long-lived species. Though coastal urbanization is widely cited as a threat to marine megafauna, currently few studies exist on the effects of pollutants and other stressors related to habitat alteration.

Marine turtles rely on nearshore areas as critical foraging and developmental habitats (Morreale and Standora, 2005), and may be particularly sensitive to the alteration of these ecosystems due to their delayed maturation and longevity (NMFS, 1998). Populations have declined in many regions (Chaloupka et al., 2004), making marine turtle conservation a high priority (NMFS, 1998). Traditional conservation actions have focused on direct threats to populations (e.g. harvest and incidental catch) with little consideration given to sublethal risks from coastal contamination (but see Keller and McClellan-Green, 2004; Keller et al., 2004a; Day et al., 2007; van de Merwe et al., 2010a). Recent attention to the importance of marine spatial planning highlights the need for accurate data on cumulative impacts across threats (Crowder and Norse, 2008). Consequently, evaluating effects of anthropogenic factors is now a top global research priority for marine turtle conservation, with the specific impacts of pollution on marine turtles identified as an area needing study (Hamann et al., 2010).

Chemical contaminants such as metals and persistent organic pollutants (POPs), e.g. pesticides, flame retardants and polychlorinated biphenyls (PCBs), make their way into coastal environments from a range of industrial, agricultural and urban sources (Sindermann, 2006). These chemicals can exert lethal and sublethal effects in wildlife, including alteration of neurological and immune function, growth, and reproduction (Beyer et al., 1996). For species like marine turtles, the required experimental toxicology research to determine these relationships is not feasible because the manipulation of long-lived endangered species is rarely permitted. A

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substantial number of studies have quantified chemical pollutants in sea turtle tissues, however, the majority are post-mortem analyses of stranded turtles or fisheries bycatch (for reviews see Pugh and Becker, 2001; Storelli and Marcotrigiano, 2003). Although these data are valuable, such specimens may not be representative of the overall population, and cannot provide information on physiological effects.

Recent studies have demonstrated the advantages of non-lethal methods for monitoring contaminants and health in protected species (Keller et al., 2004b; Day et al., 2007; van de Merwe et al., 2010b). Blood concentrations of many pollutants can be a proxy for recent exposure, while keratinous scutes reflect a longer-term signature because they incorporate elements over time. With the advent of precise instruments capable of detecting pollutants at very low levels, pollutants in blood and shell can now be accurately determined to monitor levels in wild populations. When paired with quantitative health assessments, these methods have the potential to identify relationships between contaminants and physiological condition (Keller et al., 2004a; Day et al., 2007), providing information on the conservation risk that contamination poses to sea turtles.

San Diego Bay (CA, USA) is the natural northern range limit for the east Pacific green turtle (*Chelonia mydas*) along the Pacific coast of North America, and harbors a resident population of post-pelagic juveniles and adults (Dutton and McDonald, 1990). Though the bay has been identified as critical habitat and foraging area for the green turtle, it is also highly urbanized and listed as an impaired water body (Fairey et al., 1998). Development activities such as dredging reintroduce chemicals from historical sources (e.g. PCBs) back into coastal food webs, while present-day pollution in San Diego Bay stems from a large variety of commercial and residential activities (Fairey et al., 1998). Of particular concern are polybrominated diphenylethers (PBDEs), used predominantly as flame-retardants. Many PBDEs have recently been banned due to a growing body of evidence that they have toxic and bioaccumulative effects (Hites, 2004), but previously manufactured products containing PBDEs are still in widespread use and contribute to the growing environmental reservoirs of these chemicals (Ross et al., 2009). The bay provides protection from other threats green turtles face throughout their range, but chronic pollutant exposure in this estuary may pose a threat to sea turtles inhabiting this and other contaminated coastal regions. Utilizing a combination of non-lethal techniques, we quantify levels of contaminants in the San Diego Bay green turtle population and consider possible sublethal effects to this long-lived endangered species residing in a highly urbanized nearshore environment.

2. Methods

2.1. Study site and sample collection

This study was conducted in San Diego Bay, CA (N32°40.0′ W117°13.7′), a semi-enclosed estuarine system encompassing over 57 km^2 (Fig. 1). The bay is bordered by San Diego, a densely populated metropolis with 2.9 million people, and is the terminus of three watersheds encompassing over 660 km^2. Connected to the Pacific Ocean by a narrow northwest channel, water residence time is largely driven by tidal pumping. Depths range between 5–15 m, and temperatures vary seasonally from approximately 13–25 °C (Delgadillo-Hinojosa et al., 2008).

Live green turtles were captured between November 2007 and March 2009 using large mesh gillnets deployed from a National Marine Fisheries Service vessel across three areas in the South Bay channel of San Diego Bay (Fig. 1). Thirty-one unique individuals were captured, with seven turtles being captured two of more times (total = 41). We classified turtle life stages according to Seminoff et al. (2003), including juveniles (sex undetermined) and adults (male and female). As part of a broader ecological study examining the demography and foraging ecology of green turtles, individuals were brought ashore for morphological measurements and tagging. At this time, blood and carapace tissue were sampled according to modified protocols of Owens and Ruiz (1980) and Day et al. (2005), respectively (see Appendix).

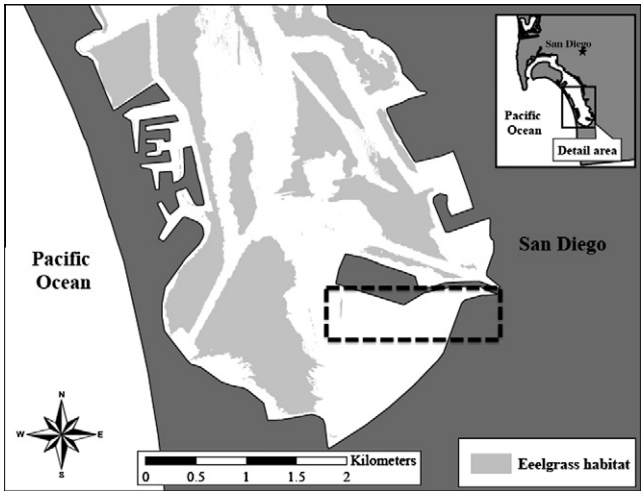

Fig. 1. Map of study area in south San Diego Bay, CA, USA (N32°40.0′ W117°13.7′). Dark gray shading denotes land surrounding the bay. Water area is shown in white, with submerged eelgrass habitat depicted by light gray shading. Green turtles utilize all regions of the Bay, particularly the southern portion (inset) and eelgrass habitats. Dashed black box denotes South Bay channel where turtles were captured.

2.2. Contaminant analyses and health assessments

We conducted trace metal analyses for whole blood, red blood cells, and carapace at Scripps Institution of Oceanography (University of California, San Diego) and the Institute for Integrated Research in Materials Environments and Society (California State University, Long Beach). We used inductively coupled plasma mass spectrometry (ICP-MS) and cold vapor atomic fluorescence spectrometry for trace metals and mercury, respectively, according to modified methods of Deheyn and Latz (2006). Percent recovery of standard reference materials ranged from 59.9% to 155% (Tables A1 and A2). Blood plasma was analyzed for a panel of persistent organic pollutants (POPs) at Mississippi Chemical Laboratory (Mississippi State, MS) according to modifications of EPA Methods 3545, 3620B, and 8081A. Spiked sample recoveries ranged from 57.3% to 110.0% (Table A3). Clinical health panels were conducted by a reptilian specialist at IDEXX (Irvine, CA) within 24 h.

2.3. Statistical analysis

We conducted all statistical analyses using SYSTAT 12 (Chicago, IL). We determined contaminant level differences among juveniles, adult male and adult females with one-way analyses of variance (ANOVA) for each element or chemical. For metals, we used paired *t-tests* for each element to determine differences between sample matrices. We used Pearson and Spearman correlation coefficients to identify relationships (1) within and across sample types (i.e. red blood cells, whole blood, carapace), and (2) among curved carapace length (CCL, an indicator of age), health markers, and chemicals. Recaptures were only sampled if caught after a minimum of 1 month to limit non-independence. Because we used blood

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samples to indicate recent exposure and carapace as a longer-term indicator, we averaged recapture carapace samples. For chemicals that exhibited correlations within sample matrices, we applied Principal Components Analysis models (PCA). This approach served to reduce issues of collinearity (Graham, 2003) and allowed us to examine overall trends of contaminant loadings and their relationships with CCL and health markers. Best fit PCA models for each sample matrix retained metals that correlated to a PC factor >|0.7| for PC factors with eigenvalues >1. All statistical analyses were performed with significance on the basis of an α of 0.05. Unless otherwise stated, values represent means ± one standard error, and stated *p*-values are the highest encountered among all comparisons.

3. Results and discussion

3.1. Contaminant levels

We detected all metals and numerous POPs in the majority of San Diego green turtles (Table 1). A number of POPs of concern, most notably total PCBs, were not detected due to higher LLOD of analytical methods available for this study. Preliminary analyses using techniques with improved LLOD have detected these chemicals in San Diego turtles (R. Lewison, unpublished data), indicating that further investigation of POPs using more sensitive methods is necessary. The majority of metals preferentially associated with red blood cells relative to whole blood (Mn, Fe, Co, Cu, As, Se, Cd, Hg, Pb; $p \leqslant 0.02$). These findings are concordant with those of Day et al. (2005), suggesting that measurement of whole blood may introduce substantial error into concentration calculations due to variable hematocrit concentrations. However, to date all other studies in sea turtles have measured metals in whole blood, making it necessary to conduct cross-study comparisons. Thus, it is in the best interest of future studies to measure both blood matrices or standardize whole blood values to hematocrit. Carapace concentrations were significantly higher than both blood matrices for all metals ($p \leqslant 0.046$) except iron and selenium. These findings support previous work proposing that the carapace acts as a sink where metals are stored in a detoxified state, indicative of longer-term exposure (Sakai et al., 2000a). However, the toxicokinetics are likely metal specific and need future study to facilitate use of carapace tissues as non-lethal biomarkers of metal bioaccumulation.

San Diego turtles exhibited lower levels of mercury in blood and carapace than those reported in loggerheads on the US. Atlantic coast, Kemp's ridleys in the Gulf of Mexico, and green turtles in Japan, but were comparable to levels in green turtles in Australia and Florida (Table A4). For other metals, whole blood concentrations were similar to all published studies, but carapace loads were higher in San Diego. Detected POPs such as 4,4′-DDE, γ chlordane, and PBDEs were also higher than most other studies. These comparisons are challenged by differences in analytical techniques and small sample sizes, and should be revisited in future investigations of San Diego turtle plasma POPs.

Pollutant concentrations identified in our study raise important questions about the longer-term physiological risks to turtles resident polluted coastal habitats. Omnivores such as green turtles are frequently assumed to have lowered exposure risks relative to carnivorous species, a hypothesis supported by our blood metal data and previous studies using organ contaminant loads (Caurant

Table 1
Descriptive statistics for (a) metal (whole blood, $N = 30$; red blood cells, $N = 27$) and (b) POP ($N = 20$) concentrations detected in one or more individuals sampled. Concentration values for whole blood, plasma and red blood cell (ng g^{-1} wet weight); carapace (ng g^{-1} dry weight). *N* represents number of independent samples above level of detection for the instruments (LLOD). Samples below LLOD were replaced with ½ LLOD for calculations. Standard error (SE) not calculated for chemicals with <3 samples above LLOD (listed n/a).

Element	Whole blood		Red blood cells		Carapace	
	N > LLOD	Mean (median) ± SE	*N* > LLOD	Mean (median) ± SE	*N* > LLOD	Mean (median) ± SE
(a) Tissue type						
Aluminum (Al)	20	146 (46.7) ± 34.2	24	140 (118) ± 26.1	31	641 000 (375 000) ± 109 000
Arsenic (As)	30	157 (102) ± 25.9	27	289 (202) ± 61.4	31	1470 (1000) ± 230
Cadmium (Cd)	19	13.2 (2.35) ± 4.20	24	29.9 (7.28) ± 10.6	31	443 (432) ± 53.7
Copper (Cu)	30	749 (727) ± 45.7	27	934 (887) ± 40.2	31	7090 (4960) ± 992
Lead (Pb)	30	1260 (967) ± 222	27	3260 (1900) ± 626	31	7230 (3590) ± 2330
Manganese (Mn)	30	463 (248) ± 89.1	27	1010 (626) ± 169	31	48 700 (29 900) ± 7040
Mercury (Hg)	30	1.01 (0.74) ± 0.16	15	2.13 (1.43) ± 0.42	31	47.5 (13.8) ± 14.5
Selenium (Se)	30	776 (346) ± 253	27	1520 (646) ± 673	31	1680 (881) ± 306
Strontium (Sr)	30	725 (652) ± 56.2	27	316 (251) ± 42.6	31	41 100 (32 300) ± 5720
Silver (Ag)	12	1.63 (0.34) ± 0.53	13	2.61 (0.34) ± 1.06	31	574 (333) ± 90.56
Tin (Sn)	11	170 (4.53) ± 138.6	14	63.5 (54.6) ± 13.1	31	3700 (1600) ± 1340

Lower limits of detection (LLOD) for ICP-MS were calculated as 3σ of the laboratory blanks and ranged between 0.381–10.4 ng g^{-1}.

Contaminant	*N* > LLOD	Mean (median) ± SE	Range
(b) Blood plasma			
γ HCH	20	0.915 (0.841) ± 0.092	0.460–2.45
Heptachlor epoxide	1	0.216 (0.200) ± n/a	<LLOD – 0.516
α Chlordane	1	0.221 (0.200) ± n/a	<LLOD – 0.620
γ Chlordane	12	0.554 (0.646) ± 0.073	<LLOD – 1.16
4,4′-DDE	14	0.736 (0.750) ± 0.097	<LLOD – 1.56
PBDE #47	2	0.200 (0.200) ± n/a	<LLOD – 0.760
PBDE #99	2	0.200 (0.200) ± n/a	<LLOD – 0.730
PBDE #153	1	0.200 (0.200) ± n/a	<LLOD – 0.220
PBDE #154	1	0.200 (0.200) ± n/a	<LLOD – 0.230
Moisture (%)	20	92.5 ± 0.425	86.3–94.6
Lipid (%)	20	0.462 ± 0.135	0.126–2.77

All compounds quantified include: hexachlorobenzene, α-hexachlorocyclohexane, γ- hexachlorocyclohexane, β- hexachlorocyclohexane, oxychlordane, γ-chlordane, α-chlordane, heptachlor epoxide, trans-nonachlor, cis-nonachlor, endrin, mirex, total toxaphene, dieldrin, total PCBs, 2,4′-DDE, 4,4′-DDE, 2,4′-DDD, 4,4′-DDD, 2,4′-DDT, 4,4′-DDT, PBDE-28, PBDE-47, PBDE-99, PBDE-100, PBDE-153, PBDE -154, and PBDE-183. Compounds not shown in table indicate all 20 samples tested were below the limit of detection. LLOD was 0.4 ng g^{-1} (w.w.) for all chemicals except PBDEs (0.2 ng g^{-1}), total toxaphene (10 ng g^{-1}), and total PCBs (2 ng g^{-1}).

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et al., 1999; Sakai et al., 2000a). However, carapace concentrations of many metals as well as plasma levels of several POPs were higher in San Diego turtles than both carnivorous and omnivorous sea turtles populations from other regions. The majority of these pollutants have already been identified as contaminants of concern in San Diego Bay due to high concentrations in sediments and biota (Fairey et al., 1998), with DDE and possibly PBDEs linked to seabird reproductive failures (Zeeman, 2004). Elevated temperatures due to warm water from power plant discharge in south San Diego Bay may also lead to higher foraging rates throughout the year in this population (Eguchi et al., 2010). Thus, increased consumption rates in a highly contaminated food web may result in San Diego turtles having an elevated risk for chemical exposure.

3.2. Correlative trends

Only a few POPs measured in plasma were detected in an adequate number of samples to calculate Spearman correlations, and all associations were weak (γ HCH and γ chlordane $r = 0.03$, γ chlordane and 4,4′-DDE $r = -0.11$, γ HCH and 4,4′-DDE $r = 0.25$). In contrast, multiple correlations existed for metals, and patterns were element dependent (Table A5). Concentrations of single metals were generally correlated between whole blood and red blood cells, however, aluminum, copper, and strontium showed virtually no relationships. Associations between different metals were observed, but only a few elements exhibited consistently high correlations in both blood matrices (As and Se $r \geqslant 0.70$, Cd and Se $r \geqslant 0.69$). These findings suggest that sample choice may impact results, possibly due to hematocrit variation.

Many metals were highly correlated within the carapace, but overall few metals were correlated between blood and carapace. Carapace mercury loads were correlated with mercury blood concentrations ($r \geqslant 0.76$), supporting similar findings of Day et al. (2005). Carapace mercury was also correlated to selenium in both blood matrices ($r \geqslant 0.74$). These associations may be the result of selenium playing a role in mercury detoxification processes, as has been observed in other species (Khan and Wang, 2009). Yet selenium and mercury were not highly correlated within blood tissues, indicating that complex physiological mechanisms drive these relationships.

Due to the lack of correlation among plasma POPs, we conducted PCA only for metals. Best-fit models for each sample type produced PC factors corresponding to groups of metals with $\geqslant$75% variance explained (%VE; each metal listed factor loading $\geqslant +0.7$; whole blood factor 1: Cu, As, Se, Cd (41%VE), factor 2: Al, Ca, Sr (36%VE); red blood cell factor 1 As, Se, Cd (49%VE); Sr, Ca (43%VE); carapace factor 1 Al, As, Ca, Co, Cu, Fe, Sr, Cd (78%VE)). These results indicate that metals within each factor positively associate with one another, making it likely that turtles with higher concentrations of one metal are likely to also have increased contaminant loads for other metals within each PC factor. The strong factor loadings coupled with the high %VE also support that PC factor scores are representative composite variables appropriate to further investigate metal load trends with turtle size, sex, and health markers.

3.3. Size and sex trends

Numerous metals in blood matrices exhibited strong negative correlations with turtle CCL (Table 3; As, Cu, Cd, Se, Hg: $r \leqslant -0.64$), while lead and aluminum had moderate positive associations ($r \geqslant 0.42$). In contrast, several carapace metals showed moderate positive correlations with CCL (Al, Mn, Cu, Pb: $r \geqslant 0.36$) and mercury was strongly negatively correlated ($r = -0.90$). Lead and mercury were the only metals exhibiting consistent relationships with CCL across all sample types, and mercury associations were much stronger (Hg: $r \geqslant -0.80$, Pb: $r \geqslant 0.41$). Whole blood PC factor 1 and red blood cell PC factor 1 (composite

Table 2
Blood serum biochemical and hematology values for all turtles sampled (2007–2009). Symbols indicate variables for which 95% CIs were different in this study compared to published studies of healthy green turtles. (✪ = Aguirre and Balazs (2000), ✚ = Bolten and Bjorndal (1992)).

Parameter	N	Mean	Median	SE	Range
SCL (cm)	31	86.22	94.40	3.25	48.25–110.4
CCL (cm)	31	90.50	99.25	3.26	52.10–116.5
Weight (kg)	31	103.7	121.0	9.59	16.00–231.0
White blood cell count ($\times 10^3\ \mu L^{-1}$)	31	11.97	12.10	0.99	2.30–21.90
Heterophil count (μL^{-1})	30	2668	2404	208.7	598.0–5544
Lymphocytes count (μL^{-1})	30	7251	8242.0	623.8	120.0–13 140
Eosinophils count (μL^{-1})	26	1756	1160.0	398.6	0.00–7035
Basophils count (μL^{-1})	13	85.38	0.00	54.98	0.00–700.0
Monocytes count (μL^{-1})	31	566.3	318.0	140.6	0.00–3870
Hematocrit (PCV) (%)	26	38.19	38.00	1.64	28.00–69.00
Alkaline phosphatase (ALP) ($U\ L^{-1}$)	22	73.05	33.50	32.17	11.00–709.0
Alanine aminotransferase (ALT) ($U\ L^{-1}$)	22	5.50	3.00	1.46	2.00–30.00
Aspartate aminotransferase (AST) ($U\ L^{-1}$)	31	153.3	150.0	6.74	77.00–287.0
Creatine phosphokinase (CPK) ($U\ L^{-1}$)	31	1312	795.0	312.6	325.0–9780
Lactate dehydrogenase (LDH) ($IU\ L^{-1}$) ✚	22	239.0	181.0	40.21	75.00–938.0
Total protein ($g\ dL^{-1}$) ✚✪	31	5.81	5.90	0.18	4.00–7.60
Albumin ($g\ dL^{-1}$) ✚✪	31	2.16	2.20	0.07	1.50–3.10
Globulin ($g\ dL^{-1}$) ✪	31	3.65	3.70	0.12	2.20–4.70
Cholesterol ($mg\ dL^{-1}$) ✪	22	228.6	207.0	23.63	102.0–647.0
Glucose ($mg\ dL^{-1}$)	31	95.94	97.00	3.07	68.00–143.0
Calcium ($mg\ dL^{-1}$)	31	8.24	8.25	0.26	3.80–12.00
Phosphorus ($mg\ dL^{-1}$) ✚✪	31	8.98	8.90	0.24	6.80–11.40
Calcium/Phosphorus	31	0.94	0.90	0.05	0.35–1.67
Potassium ($mEq\ L^{-1}$)	31	4.53	4.20	0.17	3.50–7.90
Sodium ($mEq\ L^{-1}$)	31	155.0	155.0	0.89	140.0–167.0
Uric acid ($mg\ dL^{-1}$)✚	31	1.08	1.00	0.10	0.30–2.80
Blood urea nitrogen ($mg\ dL^{-1}$) ✚✪	29	22.38	19.00	1.59	10.00–43.00
Chloride ($meq\ L^{-1}$)	15	111.5	108.0	2.77	99.00–144.0

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metal variables) exhibited negative relationships with CCL ($r \leqslant -0.80$), reinforcing that smaller turtles have higher loads of a suite of metals in blood. No other PC factors were correlated with turtle size.

Negative relationships between size and metal loads have been reported in green turtles from other regions (Gordon et al., 1998; Sakai et al., 2000b), hypothesized to be the result of the ontogenetic shift to herbivory that juvenile green turtles undergo as they migrate into neritic habitats and enter a period of rapid growth (Bjorndal, 1997). This process may contribute to the trends results, however, the negative correlations we observed are unlikely to be solely the result of lingering pelagic food web signatures because many juveniles were resident to San Diego Bay months or years prior to being sampled for this study (NMFS, unpublished data). Recent stable isotope analyses have also determined that San Diego resident turtles are actually omnivorous, exhibiting the highest invertebrate consumption rates reported for this species (Lemons et al., in press). Finally, we observed positive correlations with certain metals (e.g. lead) that contradict this hypothesis. Our observed patterns could instead be due to physiological processes, such as biotransformation and elimination, which may change with turtle maturity or exposure acclimation over time (e.g. up-regulation of metallothionein). Metal specific changes in these processes could potentially lead to differential patterns and should be considered in future studies. Alternately, our results may stem from differential resource use of foraging habitats between juveniles and adults within San Diego Bay. There is often high spatial variability in contaminant profiles within the bay due to differences in input sources and development activities (Deheyn and Latz, 2006), and such spatial segregation behavior between life stages has been observed in green turtles in Mexico (Lopez-Mendilaharsu et al., 2005). Investigation of this hypothesis via tracking studies is critical, as it may have important management implications because adequate protection of foraging areas for all life stages within San Diego Bay is necessary to conserve this population.

ANOVAs determined that metal loading differences between juveniles, adult females, and adult males were mostly explained by turtle size, i.e. juveniles differed from adults with few differences between adult males and adult females. In whole blood females had significantly higher concentrations of aluminum, calcium, and strontium ($p \leqslant 0.04$), and in red blood cells lead and iron were significantly higher in males ($p \leqslant 0.002$). Metal maternal transfer has been documented in sea turtles (Ikonomopoulou et al., 2011), so it is possible that higher lead levels in males is the result of the ability of females to lower burdens via egg deposition. PC factors (composite metals variables) for all samples types did not differ between sexes, suggesting that overall metal loads are similar for adult males and females. No significant trends in any POPs were detected with turtle size or sex.

3.4. Contaminants and health

The majority of hematology and biochemical health markers were similar to reference ranges for green turtles (Table 2). Observed differences may be due altered physiological conditions in San Diego turtles or simply natural variability among populations. Calcium:phosphorus ratios were inverted from typical mineral balance ratios found in other reptiles (65% animals < 1; mean 0.95 ± 0.05), where healthy animals generally have ratios from 1.0 to 2.0 (Hernandez-Divers et al., 2005). These are the first health data for this population, and will serve as a baseline for future studies.

Blood mercury, cadmium, and selenium were correlated with several health markers (Table 3a), however, interpretation of these relationships is confounded by covariance with turtle size. Further research using larger sample sizes within each turtle size class will validate the importance of these associations for San Diego turtles. Significant correlations with other metals and POPs not confounded by covariance included markers of hematology (RBC Ag, lymphocytes $r = 0.52$; 4,4′-DDE, hematocrit $r = 0.79$), organ function and tissue damage (Mn, AST $r \geqslant 0.44$), overall health markers (γ HCH, total protein $r = -0.49$) and ion regulation (Mn, phosphorus $r = 0.42$). Composite blood metal variables (PC factors) showed only a few significant relationships across health marker categories (e.g. RBC Factor 2, lymphocytes $r = -0.57$), indicating that associations are chemical specific and do not exhibit overall trends for recent metal exposure. Some carapace metals also covaried with CCL, but health marker patterns overall were very dissimilar to those observed in blood matrices (Table 3b). Silver and selenium were negatively correlated with multiple white blood cell types ($r \leqslant -0.35$) and positively associated with several enzymes ($r \geqslant 0.38$), while carapace PC factor 1, a biomarker of longer-term overall metal load, positively correlated with several indicators of overall health.

The contrasting trends between blood and carapace associations with health markers reinforces that these tissues reflect different timescales of exposure and possibly physiological processes. In chronically contaminated coastal systems like San Diego Bay, recent chemical exposure may not be acute enough to elicit obvious response patterns in large vertebrates. Over time, however, physiological acclimatization and associated energetic demands of chronic chemical biotransformation and elimination could result in altered biochemical and hematological profiles, potentially explaining our observed trends between increased carapace metal loads and health markers. Alternatively, correlative trends may not depict relationships accurately if physiological responses involve biological thresholds and are non-linear. Risk assessments comparing values to established biological threshold concentrations are typically effective under such conditions, but are problematic for marine turtles because few chemical thresholds have been established for reptiles. Of thresholds established for loggerheads by Keller et al. (2006) and Day et al. (2007), blood mercury and 4,4′-DDE concentrations in San Diego turtles exceeded lymphocyte proliferation *no-effects* levels, suggesting that immunological effects may be a concern at current concentrations of these pollutants. San Diego concentrations were generally similar or lower than thresholds in birds and mammals (Beyer et al., 1996), but differences among taxa and tissue types make interpretation of these comparisons uncertain.

There is evidence that chemical exposure in marine turtles may cause abnormalities in embryonic development (van de Merwe et al., 2009), disrupt endocrine balance (Ikonomopoulou et al., 2009) and alter other important metabolic functions (Peden-Adams et al., 2002; Keller and McClellan-Green, 2004). Our results do not identify definitive cause-effect relationships of coastal contamination on San Diego turtle health, but rather provide ecologically relevant data to inform hypotheses warranting further investigation. Correlations between pollutants and white blood cell counts coupled with chemical concentrations exceeding immune function *no-effects* levels underscore the need for immunological studies in this population (e.g. lymphocyte proliferation and cytokine gene expression). Additionally, associations between carapace metal loads and enzyme concentration increases support future investigation of physiological responses and energetic trade-offs with chronic sublethal chemical exposure.

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Table 3
Pearson and Spearman correlation coefficients between chemicals and health markers. Only relationships with $p \leqslant 0.1$ are depicted for ease of interpretation. Correlation calculations use concentrations of metals in (a) red blood cell (RBC), whole blood (WB), and (b) carapace, and POPs in blood plasma. Bolding designates $p \leqslant 0.05$. For abbreviation definitions see Table 2.

(a)	Parameter	CCL	Sample type	Al	As	Cd	Cu	Pb	Hg†	Mn	Ag	Se	Sr	PC Factor 1	PC Factor 2
	CCL		RBC	**0.42**	**-0.73**	**-0.87**		**0.50**	**-0.83**			**-0.85**		**-0.86**	
			WB		**-0.78**	**-0.89**	**-0.64**	**0.46**	**-0.80**			**-0.88**		**-0.89**	
Cell Counts	Heterophils (27)		RBC							-0.40^{S}					
	Lymphocytes (27,30)		RBC												**-0.57**
			WB					-0.36			**0.52^{S}**				
	WBC Count (30)		WB								**0.41^{S}**				
	Monocytes (27,30)	**0.44^{S}**	RBC			-0.38^{S}									
			WB		**-0.42^{S}**	-0.32^{S}	**-0.60^{S}**							**-0.39^{S}**	
	Eosinophils (26)		RBC								0.45^{S}				
			WB								**0.46^{S}**				
	HCT (26)		WB								**-0.40^{S}**				
Organ/Tissue Function	ALP (22)	**-0.56^{S}**	RBC		0.37^{S}	**0.49^{S}**						0.42^{S}		**0.46^{S}**	
			WB		**0.42^{S}**	**0.54^{S}**			**0.52^{S}**			**0.52^{S}**		**0.48^{S}**	
	ALT (22)		WB				**-0.48^{S}**								
	AST (27,30)	-0.31^{S}	RBC			0.34^{S}	-0.35^{S}			**0.47**					-0.38
			WB			**0.37^{S}**				**0.44**					
	CPK (27,30)	-0.33^{S}	RBC			**0.49^{S}**		-0.35^{S}				**0.44^{S}**		**0.46^{S}**	
			WB			0.39^{S}			**0.42^{S}**			0.37^{S}	**-0.39^{S}**	0.36^{S}	
	LDH (22)		RBC				**0.44^{S}**		-0.61			0.39^{S}			
Overall Health Indicators	Glucose (27,30)		RBC						**0.58**						
			WB					-0.34	**0.66^{S}**						
	Total Protein (27,30)	**0.49^{S}**	RBC						-0.56					**-0.48**	
			WB		**-0.54**	**-0.61^{S}**	**-0.40**	**0.44**				**-0.53**			
	Albumin (27,30)	**0.401^{S}**	RBC			**-0.46^{S}**									
			WB		**-0.44**	**-0.44^{S}**	**-0.45**					-0.36			
	Globulin (27,30)	**0.44^{S}**	RBC	0.40^{S}		**-0.50^{S}**		**0.51**	-0.56			-0.36^{S}		**-0.49**	
			WB		**-0.53**	**-0.55^{S}**		**0.53**	**-0.59^{S}**		-0.33^{S}	-0.56			
	A:G ratio (27,30)		RBC					**-0.59^{S}**		**-0.53^{S}**					
			WB			0.45^{S}		**-0.62^{S}**				**0.53^{S}**			
	Uric Acid (27,30)		RBC	**0.50^{S}**			0.39^{S}	0.36			**-0.57^{S}**				
			WB					0.40			**-0.54^{S}**				
	BUN (27)		RBC	-0.36^{S}											
	Cholesterol (22)	**0.55^{S}**	RBC	0.42^{S}								-0.42^{S}			**0.61**
			WB			**-0.56^{S}**			**-0.48^{S}**						
Ion Regulation	Phosphorus (27,30)		RBC	**0.51^{S}**				0.36		**0.42**					
			WB					**0.38**		0.36					
	Potassium (27,30)	**-0.40^{S}**	RBC		0.37^{S}	0.38^{S}						0.40^{S}		**0.48^{S}**	
			WB						0.36^{S}						
	Sodium (30)		WB					-0.36^{S}							

(b)

	Parameter	CCL	Al	As	Cd	Cu	Pb	Mn	Hg	Se	Ag	Sn	PC Factor 1	γ HCH	γ chlordane	4,4′ DDE
	CCL		**0.38**			**0.36**	**0.41**	**0.56***	**-0.90**	-0.30						
Cell Counts	WBC Count (31)									**-0.50^{S}**	**-0.43**					
	Heterophils (31)			-0.36^{S}	-0.36^{S}						**-0.42**				-0.57^{S}	
	Lymphocytes (30)				-0.31^{S}					**-0.61^{S}**	**-0.35**					
	H/L Ratio (30)									0.35^{S}						
	Monocytes (31)	**0.44^{S}**														
	Eosinophils (26)									**-0.59^{S}**	**-0.63**			-0.51^{S}		
	HCT (26)			0.38^{S}	**0.59^{S}**	**0.46^{S}**				**0.55^{S}**	**0.55**		**0.45^{S}**			**0.79^{S}**
Organ/Tissue Function	ALP (22)	**-0.56^{S}**	**-0.49^{S}**			-0.41^{S}		**-0.63^{S}**	**0.54^{S}**					0.50^{S}		
	ALT (22)				**0.46^{S}**	0.46^{S}										
	AST (31)	-0.31^{S}														
	CPK (31)	-0.33^{S}						-0.32^{S}	0.34^{S}	0.35^{S}						
	LDH (22)									**0.44^{S}**						
Overall Health Indicators	Glucose (31)			**-0.39**		-0.34	-0.31^{S}		**0.41^{S}**							
	Albumin (31)	**0.41^{S}**		0.33	**0.53^{S}**	**0.49**	**0.44^{S}**				**0.38**		0.35			0.49^{S}
	Total Protein (31)	**0.50^{S}**	**0.50**	**0.42**	**0.53^{S}**	**0.60**	**0.55^{S}**	**0.47**	**-0.49^{S}**		**0.43**	**-0.37^{S}**	**0.46**	**-0.49^{S}**		
	Globulin (31)	**0.44^{S}**	**0.52**	**0.39**	**0.37^{S}**	**0.55**	**0.51^{S}**	**0.51**	**-0.52^{S}**		**0.38**	-0.32^{S}	**0.44**			
	A/G Ratio (31)		**-0.27^{S}**					-0.43^{S}	**0.53^{S}**	**0.44^{S}**				**0.59^{S}**		
	Uric Acid (31)			**0.53**	**0.58^{S}**	**0.38**				**0.40^{S}**	**0.68**	**-0.58^{S}**	**0.46**			0.51^{S}
	Cholesterol (22)	**0.55^{S}**	**0.45**	0.42		**0.46**	**0.56^{S}**	**0.46**					0.43			
Ion Regulation	Calcium (31)											-0.33^{S}				
	Potassium (31)	**-0.40^{S}**														
	Sodium (31)	-0.32														
	Chloride (15)		-0.43								**-0.56**					

N for each correlation denoted in parentheses for (RBC,WB). If only one number is listed, it corresponds to the only tissue type listed for that parameter.
S = Spearman rank correlations; † One adult female outlier with high mercury levels in all tissues was removed from correlation calculations and sample size for Hg RBC correlations = 15.

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4. Conclusion

San Diego Bay is typical of many coastal areas where protection from harvest and bycatch make it critical habitat for species vulnerable to these threats elsewhere. Our study demonstrates that San Diego turtles are exposed to pollutants known to exert negative physiological effects in other species. If this urbanized estuary is to serve as critical habitat it must be managed to consider these risk factors. Many compounds detected in San Diego turtles have been banned in the United States for several decades, but remain as legacy pollutants in Bay sediments (Deheyn and Latz, 2006; Fairey et al., 1998). Dredging occurs frequently for shipping lanes and other development projects, resulting in the release of legacy chemicals back into coastal food webs. The prevalence of these activities in San Diego Bay may be partially responsible for the high levels of contaminants observed in resident green turtles. Therefore, in addition to regulating contemporary pollutant inputs from commercial and residential activities, protection of critical turtle habitat needs to include consideration of these activities. Our results highlight the need for future monitoring of both contemporary and legacy pollutants in San Diego Bay wildlife to attain robust population estimates of contaminant burdens, and suggest that they cannot be dismissed as potential risk factors for longer-lived vertebrates inhabiting polluted coastal habitats. Continued monitoring of contaminant loads coupled with immunological and physiological effects studies will increase our understanding of pollution impacts on marine turtles inhabiting contaminated coastal habitats.

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Appendix A. Supplementary material

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.chemosphere.2011.04.023.

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