

Loggerhead turtles killed by vessel and fishery interaction in Virginia, USA, are healthy prior to death

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ABSTRACT: Human-induced mortality of marine animals is a complex issue and the health of animals prior to human interaction is often questioned. We reviewed 74 cases of loggerhead turtles in the mid-Atlantic region of the US to determine whether fresh, dead loggerhead turtles that died from acute vessel or fishery interaction (acute VI/FI) were healthy animals prior to human-induced mortality. We used the presence of significant underlying pathology to determine health status and compared the body condition and adipose stores with turtles that died from other causes. We qualitatively and quantitatively assessed body condition using images, morphometrics, mass dissection data, % lipid and % triacylglycerol in the adipose tissue. We used the quantitative indices to validate the qualitative index. Acute VI/FI turtles were significantly less likely to have significant lesions indicating compromised health compared with turtles that died from other causes ($\chi^2 = 12.9012$, df = 1, p = 0.0003). There was a significant relationship between qualitative body condition and cause of death category, with acute VI/FI turtles more likely to exhibit normal body condition than turtles that died from other causes ($\chi^2 = 18.879$, df = 2, p < 0.0001). Values for 3 quantitative indices were significantly higher for acute VI/FI turtles compared with other causes of death, while 3 others were not different across turtles, most likely because there were some healthy turtles in the 'other' cause of death category. Our results suggest that the majority of acute VI/FI loggerhead turtles represented normal, healthy turtles in the population and were not compromised prior to human-induced mortality.

KEY WORDS: Sea turtle · Body condition · Human-induced mortality · Adipose · Stranding

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INTRODUCTION

Human-induced injury and mortality of marine species occurs globally. Of particular concern is the incidental capture of air-breathing megafauna in commercial fisheries, which contributes to declines in

populations of marine mammals, seabirds, and sea turtles (Stein et al. 2004, Campbell-Malone et al. 2008, Polidoro et al. 2008, Moore et al. 2009, Cassoff et al. 2011, Lewison et al. 2014, Wilson et al. 2014). Lewison et al. (2014) noted that the loss of predatory air breathing marine megafauna affects biotic inter-

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actions, disturbance regimes, species invasions, and nutrient cycling. They calculated by catch intensity on marine mammals, sea birds and sea turtles individually and cumulatively for 3 gear types, gillnets, longlines and trawls. Sea turtles had the highest by catch intensity globally. Mortality from vessel strikes was the second most common cause of human-induced mortality for global large whale deaths examined from 1970 to 2009 (van der Hoop et al. 2013). The impacts of vessel strikes on sea turtle populations are less well studied, but may be considerable in areas with large human populations and high boat traffic (Polidoro et al. 2008, Conant et al. 2009, Reimer et al. 2016). Additional studies on human interactions with sea turtles in densely populated regions are needed to fully understand the scope of anthropogenic causes of mortality (Wallace et al. 2013).

A portion of the western North Atlantic loggerhead turtle *Caretta caretta* distinct population segment (DPS) seasonally spends time in the ocean and/or estuarine waters of the mid-Atlantic U.S. (NMFS 2011a,b). Chesapeake Bay is the largest estuary in the region and is an important juvenile sea turtle habitat (Lutcavage & Musick 1985, Musick & Limpus 1997, Coles 1999, Mansfield 2006). The region is a busy fishing, shipping and recreational boating area, and sea turtles are frequently subject to interactions with human activities (Mansfield 2006, Murray 2009, Byrd et al. 2011, NMFS 2011a, Smolowitz et al. 2012). Stranding data can provide insight into threats faced by sea turtles in this region, and clarify sources of human-induced and natural mortality (Epperly et al. 1996, Orós et al. 2005, Chaloupka et al. 2008, Tomás et al. 2008, Casale et al. 2010, Byrd et al. 2011, Koch et al. 2013, Poli et al. 2014, Nicolau et al. 2016).

Most sea turtles that strand in Virginia are in a moderate to advanced state of decomposition and a definitive cause of death cannot be determined (Lutcavage & Musick 1985, Keinath et al. 1987). For a small subset of fresh animals, however, a thorough gross necropsy and histopathology assessment permit determination of cause of death as well as other underlying conditions. The Virginia Aquarium & Marine Science Center's Stranding Response Program (VAQS) has been conducting necropsies and submitting tissues for histopathology on stranded and incidentally caught loggerhead turtles since 2004. Having a dataset of animals whose tissues have been examined for significant underlying pathology as well as evidence of human interaction provides us with a unique opportunity to better understand the health of animals that die from acute interaction with vessels or fisheries.

Turtles that die from disease and/or debilitation are often anorexic, have heavy epibiota loads, and exhibit poor coloring and body condition (Flint et al. 2010, Work et al. 2015). It is possible that underlying health issues impact normal diving and foraging behavior and may increase vulnerability of turtles to human interactions. If this is the case, then turtles that ultimately die as a result of human interactions would exhibit signs of underlying health issues and poor body condition at post-mortem examination. Alternatively, if turtles that die from trauma due to vessel strikes or die in fishing gear interactions are not already compromised in some way, then they represent healthy animals and should be in normal body condition.

Body condition in animals is difficult to define and quantify and numerous indices have been used in fishes, amphibians, birds and small mammals (Reist 1985, Krebs & Singleton 1993, Brown 1996, Labocha & Hayes 2012, MacCracken & Stebbings 2012). Quantitative indices are usually based on non-invasive size and mass parameters that are designed to predict the amount of adipose tissue in an individual. One common index is the Fulton's *K* score, based on the length/mass relationship (first proposed by Heincke in 1908; see review in Nash et al. 2006), which is often used in fishes and has been referred to as the body condition index (BCI) in some sea turtle studies (Bjorndal et al. 2000, Work et al. 2004, 2015, Flint et al. 2010). The majority of the adipose depots in cheloniid sea turtles are encased within the rigid carapace and plastron, so traditional body condition indices based on external morphology may not be as effective as qualitative indices. Residuals from a regression of body mass on curved carapace length for live turtles (Jessop et al. 2004) and qualitative visual descriptions of the pectoralis muscle and color of the fat in dead turtles (Foley et al. 2007) have also previously been used to assess body condition. To date, there have been no studies to validate the use of morphometric or qualitative features as indices of adipose stores and body condition in sea turtles.

Body condition indices are designed to assess the quantity of adipose tissue, but adipose quality, measured by lipid content (% lipid) and triacylglycerol (TAG) content in the lipid (% TAG), may be more closely correlated with health (Napolitano & Ackman 1990, Ramsay et al. 1992, Stegall et al. 1999, Blair et al. 2000, Thiemann et al. 2006, McKinney et al. 2014). Adipose tissue in a healthy, robust individual would have a higher lipid content and a higher percentage of TAG (storage lipid and thus reflective of energy reserves) in the lipid than a compromised individual.

Turtle adipose is distributed throughout the body in robust sea turtles, with substantial depots associated with the carapace, appendage insertions, coelomic membranes, kidneys and gastro-intestinal tract (Pond 1998).

In this study, we used the presence of significant underlying pathology to determine health status of stranded and incidentally caught loggerhead turtles, and compared the body condition, muscle mass, and adipose stores of loggerhead turtles that died from acute vessel or fishery interaction (acute VI/FI) with those that died from other causes. If loggerhead turtles that died from acute VI/FI are in significantly better condition than those that die from other causes, then we can infer that vessel and fishery interaction affect primarily healthy animals. Additionally, we conducted a validation study to assess the reliability of quantitative and qualitative measures of body condition in loggerhead turtles to refine health assessments in this species.

MATERIALS AND METHODS

We reviewed images, gross necropsy reports and histopathology reports from fresh, dead loggerhead turtles examined at the Virginia Aquarium & Marine Science Center (VAQ) between 2004 and 2013. Although we do not know the exact date and time of death in most cases, we estimate that fresh dead turtles died up to 96 h prior to examination, depending on environmental conditions, primarily air and water temperature. All necropsy and pathology reports were produced by the authors and/or experienced colleagues. Most necropsy reports written from 2004 through 2013 were also reviewed by a veterinarian. In the pathology reports, each tissue was described and significant lesions that may have compromised a turtle's health or that likely contributed to the cause of death were summarized. Examples of significant lesions considered to be indicative of underlying health issues were those consistent with sepsis, myocarditis and/or pericarditis, hepatitis, parasitic and/or bacterial meningoencephalitis, arterial thrombus and perforated colon. Conditions such as mild to moderate parasitic infection were noted, but were not considered significant enough to affect a turtle's health. Acute pneumonia, with no obvious bacterial, parasitic or fungal etiology, was considered to be consistent with drowning, similar to humans where a diagnosis of drowning is basically one of exclusion of other causes of death (DiMaio & Dana 2007). We also reviewed morphometric data from live loggerhead

turtles captured or incidentally caught in net enclosures as part of a separate project to compare with the fresh dead turtle data.

We classified cases into one of 2 cause of death categories based on necropsy and pathology reports: (1) Acute VI/FI and (2) all other causes of death. Acute VI/FI included turtles that died from acute, traumatic injury resulting from a vessel strike, stranded turtles with evidence of drowning with acute trauma associated with entanglement or underwater entrapment in fishing gear, and observed, lethal takes from commercial fishing gear. The 'other' cause of death category included turtles that died from disease as indicated by significant lesions, animals that died with evidence of healing vessel trauma wounds, acute pneumonia consistent with drowning but with no evidence of entanglement, turtles with fish hooks chronically imbedded in the gastrointestinal (GI) tract, death from cold stunning, cases of chronic malnutrition, and cases where cause of death was not determined based on available evidence, but clearly was not attributable to acute VI/FI.

We qualitatively assessed body condition as normal, thin, emaciated, or could not be determined (CBD) using a standard set of images (dorsal, frontal, lateral and oblique view with the animal in ventral recumbency, and ventral and frontal view with the turtle in dorsal recumbency) and descriptions of external morphology in the necropsy reports. Scoring was similar to Heithaus et al. (2007) which was validated using the Fulton's *K* index described in Bjorndal et al. (2000), with Heithaus et al.'s 'poor' condition described here as 'emaciated,' their 'fair' and 'good' conditions combined into what we called 'thin,' and their 'very good' described here as 'normal' (see text and Fig. S1 in the Supplement at www.int-res.com/articles/suppl/m555p221_supp.pdf for description and images). If images were not available or were of poor quality, animals were not assigned a qualitative body condition and were listed as CBD. We did not capture images of live animals in dorsal recumbency, and head and neck images were usually taken with the head pulled tightly against the body, thus we were unable to conduct visual assessments on live turtles.

To quantitatively assess body condition using morphometric data, we used minimum straight carapace length (SCL-NN) and body mass to calculate a Fulton's *K* score for each individual, including live-captured loggerhead turtles, based on the formula $K = W/L^3 \times 10\,000$, where W is body mass in kilograms and L is SCL-NN in centimeters (Nash et al. 2006). Because sea turtles are not cubical as assumed by the Fulton's model, we developed a modified Ful-

ton's K formula which included other carapace morphometrics, replacing the L^3 value with carapace length \times carapace width \times [(right carapace depth + left carapace depth)/2]. For some live animals, we only collected one body depth measurement. The absolute difference between left and right body depths was 0.50 ± 0.50 cm (mean \pm SD); thus, when only one measurement was available, we included the single measurement twice. To test the assumption that the body condition index was insensitive to the influence of an animal's length on its body condition score, we ran a regression analysis for Fulton's K and SCL and checked for correlation between the residuals of the regression and SCL as conducted by Jessop et al. (2002).

We conducted mass dissections on a subset of fresh dead turtles, in which the mass of skeletal elements, muscle groups, viscera and adipose depots were documented. By comparing the mass of right versus left muscles, we determined that shoulder muscles were the most precisely and consistently collected muscle group. We developed a muscle mass index (MMI) by dividing the mass of the shoulder muscles (with coracoid process) by the mass of the carapace with all tissue removed to correct for size. The MMI was meant to assess protein catabolism as body condition declined. The adipose depots were difficult to dissect, and right and left appendage depots were rarely similar. Thus, we used the carapace depot to develop an adipose mass index (AMI) because it was the most precisely and consistently collected depot. The AMI was calculated by dividing the mass of the adipose adhered to the carapace by the carapace mass without any tissue attached. The AMI was designed to assess lipid oxidation. For both indices, we used the mass of the carapace cleaned of tissue instead of total body mass because it was likely to change with turtle size but not with cause of death (blood loss, seawater aspiration, etc.) or stomach contents.

We collected adipose tissue from depots associated with the carapace, appendage insertions, coelomic cavity and mesenteric membranes. We used a modified Folch procedure to extract lipid from a known mass of adipose tissue from 2 or more depots in each turtle and divided lipid weight by adipose wet weight to determine % lipid (Folch et al. 1957, used by Iverson 1988 and Koopman et al. 1996). Once extracted, the lipid was stored in hexane at 100 mg lipid ml^{-1} hexane at -10°C prior to lipid class determination. We used thin layer chromatography with flame ionization detection (TLC-FID; Iatroskan Mark VI, Mitsubishi Kagaku Iatron) to separate lipid samples into lipid classes. Samples were spotted in 1 μl

aliquots on chromarods that were developed in hexane:ethyl acetate:formic acid (94:6:1) for 25 min. Each sample was run in duplicate and lipid classes were quantified by FID. Lipid class peaks were identified and integrated using PeakSimple 329 Iatroskan software (SRI Instruments), based on lipid class standards (Nu Chek Prep, Elysian). Lipid class content was calculated by applying standard curves generated from known concentrations. To compare % lipid and % TAG within different adipose stores of the same individual and between individuals, we ranked the data and performed a 2-way ANOVA on the ranked data.

Statistical analyses were conducted using R (R Core Development Team 2011). We checked for differences in the cleaned carapace mass corrected for size among the visual body condition categories using ANOVA. We used Pearson's product moment correlation between carapace mass corrected for size and Fulton's K scores to check for indication of skeletal atrophy and to confirm that carapace mass was unlikely to be affected by overall body condition. We used a 2×2 contingency table (Chi-square) analysis to investigate the relationship between presence of significant lesions indicative of underlying conditions and cause of death category. A nonparametric Kruskal-Wallis test was used to compare AMI, MMI, Fulton's K , modified Fulton's K , % lipid and % TAG among the qualitative body conditions, and we used a nonparametric Tukey and Kramer post-hoc rank pairwise comparison (Sachs 1997) in the R package PMCMR (Pohlert 2014) to further assess significant results. We used Spearman's rank correlation to investigate relationships between the morphometric body condition indices (Fulton's K and modified Fulton's K) and AMI, MMI, % lipid and % TAG. The relationship between qualitative body condition and cause of death category was investigated using a 3×2 contingency table with post-hoc pairwise comparison using Bonferroni corrections, with $\alpha = 0.008$ (MacDonald & Gardner 2000).

We compared Fulton's K scores for acute VI/FI and other causes of death using a 1-sided Welch 2-sample t -test. A 1-sided non-parametric Wilcoxon rank-sum test was used to compare modified Fulton's K scores for acute VI/FI and other causes of death, as the data did not meet assumptions of normality (Shapiro-Wilk test of normality) and equal variance (Levene's test). We used the nonparametric Wilcoxon rank-sum test to compare % lipid, % TAG, AMI and MMI data between acute VI/FI and other causes of death, since these data were percentages or ratios. Finally, we used an ANOVA with Tukey's post-hoc pairwise

comparison to compare Fulton's K scores among live, apparently healthy turtles, acute VI/FI turtles and other causes of death.

RESULTS

We reviewed case reports of loggerhead turtles that stranded in Virginia or were observed lethal takes in the mid-Atlantic region to categorize cause of death (Fig. 1), document the presence of significant lesions that would indicate underlying conditions ($N = 70$), and assess body condition using quantitative and qualitative measures. We conducted mass dissections on a subset of the study animals and calculated MMI ($N = 40$) and AMI for those cases ($N = 42$). We determined % lipid for 34 turtles and determined % TAG for 22. In addition, we calculated Fulton's K for 14 live captured, presumed healthy, loggerheads.

Thirty-one turtles died as a result of a vessel strike ($N = 15$) or fishery interaction ($N = 16$) and were categorized as acute VI/FI, and 43 turtles died from other causes. Of the 31 acute VI/FI cases with histopathology results, only 1 turtle, an observed fishery take, had significant lesions that indicated underlying conditions may have compromised its health. In contrast, of the 39 turtles in the other cause of death category for which histopathology reports were available, 17 had significant lesions indicative of underlying conditions (Table 1). A Chi-square analysis indicated that there was a significant relationship between cause of death category and presence of significant lesions, with turtles in the acute VI/FI category being less likely to exhibit significant lesions indicative of underlying disease conditions compared with turtles that died from other causes ($\chi^2 = 12.9012$, $df = 1$, $p = 0.0003$).

Residuals from Fulton's body condition scores were not significantly correlated with turtle SCL ($r^2 = -0.01515$, $p = 1.0$; Fig. 2) indicating that the length of the turtles in this study (primarily large juveniles) did not affect body condition scores. Likewise, there was

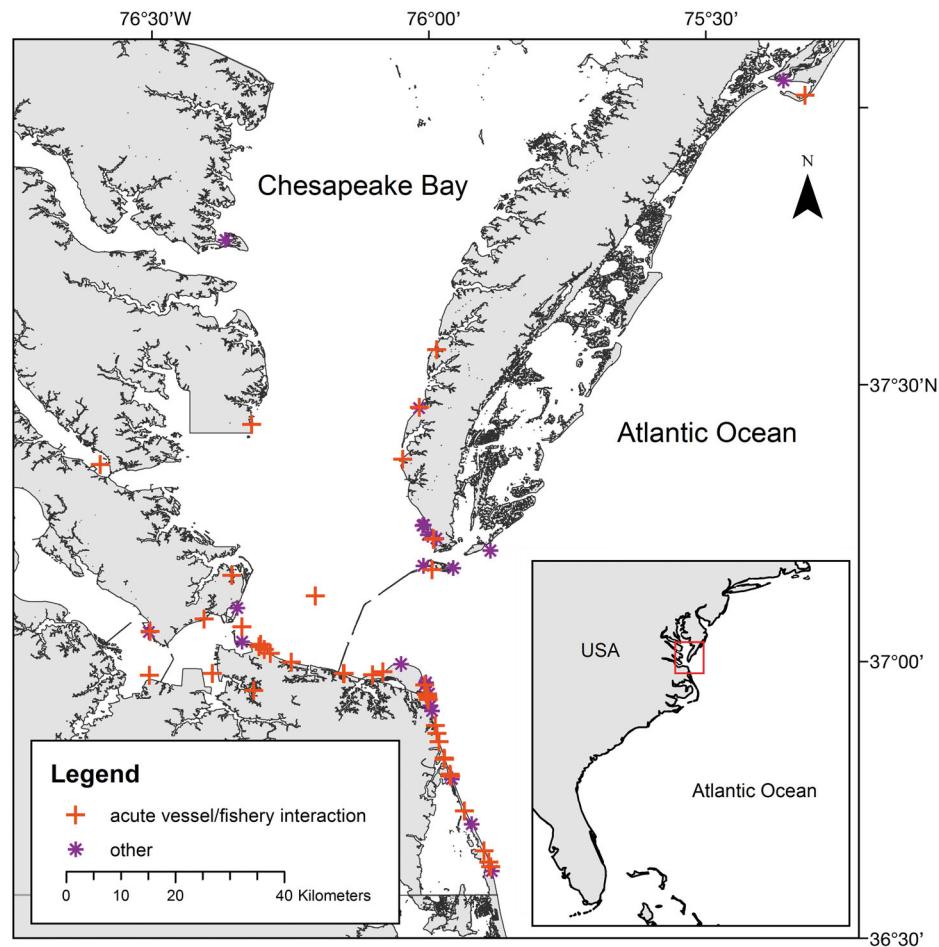


Fig. 1. Location of 60 stranded loggerhead turtles examined at the Virginia Aquarium & Marine Science Center between 2004 and 2013 that were included in the study of body condition prior to death. We did not have access to latitude and longitude for the 14 observed, lethal takes that were part of the study

Table 1. Results of analysis of necropsy reports and histopathology reports from fresh, dead loggerhead turtles examined at the Virginia Aquarium & Marine Science Center between 2004 and 2013, showing cause of death, presence or absence of significant lesions indicative of underlying ill-health conditions (based on histopathology results, available for 70 of the 74 turtles reviewed) and visual body condition assessment. VI/FI: vessel or fishery interaction; CBD: could not be determined

Cause of death	No. of turtles	Lesions indicative of underlying condition	Visual body condition		
			Normal	Thin	Emaciated
Acute VI/FI	31	1 of 31	27	1	0
Other causes	43	17 of 39	19	14	8
Total	74	18 of 70	46	15	8
					5

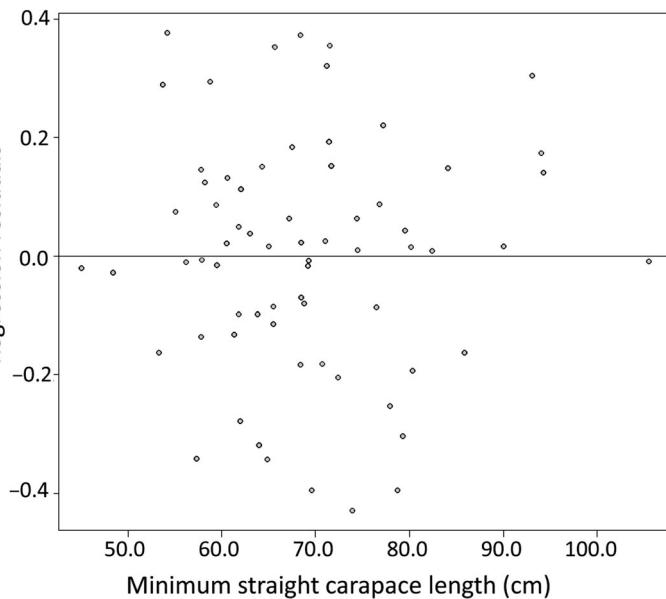


Fig. 2. Plot of residuals from regression of straight carapace length (SCL) of loggerhead turtles with Fulton's K score, used to assess body condition. There was no significant correlation ($r^2 = -0.015$, $p = 1.0$) indicating that Fulton's K scores were not related to carapace length

no relationship between visual body condition and cleaned carapace mass divided by SCL ($F_{2,37} = 0.353$, $p = 0.705$), nor was there a significant correlation between Fulton's K and carapace mass corrected for SCL ($t_{42} = -0.1574$, $p = 0.876$). Thus we felt that carapace mass did not change significantly with body condition and was an appropriate correction factor for AMI and MMI. We used the AMI and MMI to validate the visual body condition, Fulton's K and modified Fulton's K scores. We also compared the quantitative body condition scores among the 3 qualitative body condition categories. There were significant differences in both the AMI and MMI among qualitative body condition categories (Fig. 3). The mean AMI for turtles categorized as normal was significantly different from that of thin turtles, but AMI for the emaciated turtles was not significantly different from either normal or thin turtles (Fig. 3). The MMI for normal and thin turtles was significantly higher than it was for emaciated turtles. For the Fulton's K scores, all 3 categories were significantly different. There were no differences in modified Fulton's K scores among the qualitative body condition cate-

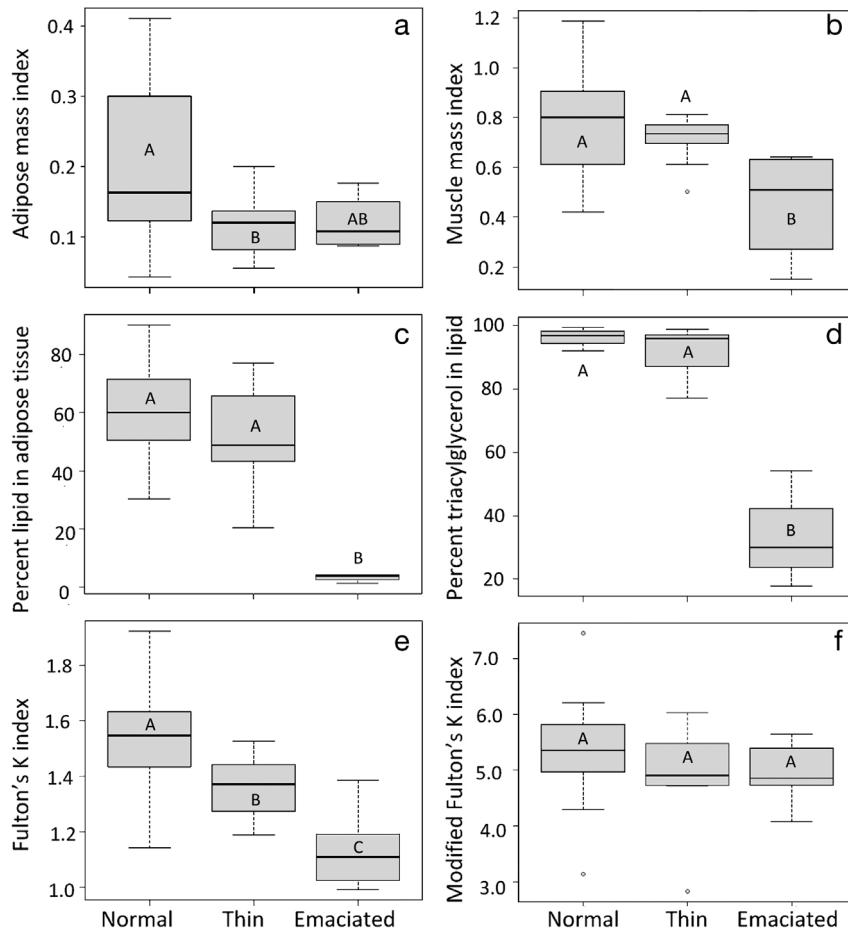


Fig. 3. Comparison of visual body condition assessment of loggerhead turtles (as normal, thin or emaciated) with (a) lipid quantity (adipose mass index); (b) muscle mass (muscle mass index); lipid quality measured by (c) lipid content (% lipid) and (d) triacylglycerol content (% TAG); and quantitative body condition scores: (e) Fulton's K and (f) modified Fulton's K . Visual body condition categories differed significantly in all measures, except modified Fulton's K , using Kruskal-Wallis rank-sum tests with Tukey and Kramer non-parametric post-hoc analysis. Different uppercase letters indicate significant differences among groups (Tukey and Kramer, $p < 0.05$). The area of each box represents the upper and lower 25% quartiles of the data on either side of the median (black bar) and the whiskers represent the range of the data excluding outliers, which are represented as open circles

gories. AMI was significantly positively correlated with both the Fulton's K ($R = 0.349$, $p = 0.026$) and modified Fulton's K ($R = 0.318$, $p = 0.023$).

Analysis of ranked % lipid data from 2 to 4 different adipose depots in each turtle indicated that % lipid was significantly different between individual turtles ($F_{3,25} = 6.3598$, $p = 0.003$) but was similar within the adipose depots of an individual turtle ($F_{3,25} = 1.4845$, $p = 0.28$). For the % TAG data, there were no significant differences in the same adipose depots among different turtles ($F_{4,22} = 13.2827$, $p = 0.21$) or between different depots in the same turtle ($F_{4,22} = 0.9703$, $p = 0.62$). Because there were no significant differences among the adipose depots within each individual turtle, we calculated mean % lipid in adipose tissue and mean % TAG in lipid for each turtle used in further comparisons. Percent lipid and % TAG were similar for normal and thin turtles, and both were significantly higher than emaciated turtles (% lipid: $\chi^2 = 11.778$, $df = 1$, $p = 0.003$; % TAG: $\chi^2 = 10.125$, $df = 1$, $p = 0.006$; Fig. 3). There was a significant correlation between Fulton's K and lipid quality (% lipid: $R = 0.397$, $p = 0.028$; % TAG: $R = 0.505$, $p = 0.029$), but not between modified Fulton's K and lipid quality (% lipid: $R = 0.016$, $p = 0.936$; % TAG: $R = -0.360$, $p = 0.156$).

All but one of the acute VI/FI turtles for which we were able to assign a qualitative body condition were categorized as normal (see Table 1). Of the turtles

that died from other causes for which we were able to assign a qualitative body condition ($N = 41$), 19 were visually normal, 14 were thin, and 8 were emaciated. There was a significant relationship between qualitative body condition and cause of death category, with acute VI/FI turtles more likely to exhibit normal body condition than turtles that died from other causes ($\chi^2 = 18.879$, $df = 2$, $p < 0.0001$). A post-hoc pairwise analysis of the data showed significant differences in the number of normal and thin turtles between the 2 cause of death categories ($\chi^2 = 12.331$, $p = 0.0004$, Bonferroni corrected $p = 0.016$) and in the number of normal and emaciated turtles ($\chi^2 = 9.3913$, $p = 0.002$, Bonferroni corrected $p = 0.016$), but no difference in the number of thin and emaciated turtles between the 2 categories ($\chi^2 = 0.5576$, $p = 0.45$, Bonferroni corrected $p = 0.016$).

The Fulton's K body condition scores were significantly higher for turtles that died from acute VI/FI compared with those dying from other causes of death (Table 2). Likewise, the modified Fulton's K body condition scores were significantly higher in acute VI/FI turtles compared with turtles in the other cause of death category. Neither % lipid nor % TAG was significantly different between acute VI/FI and other causes of death (Table 2). AMI was significantly higher in acute VI/FI turtles compared with other causes, but the MMI values were not significantly different between acute VI/FI and other causes of death.

Table 2. Results of t -tests between acute vessel or fishery interaction (VI/FI) cases and other cases. All comparisons were significant except for % lipid in adipose, % triacylglycerol (% TAG) in lipid and muscle mass index. When we eliminated the other/unknown cases and compared acute VI/FI with animals that died from disease/debilitation, the % lipid results were significantly different

Indicator of body condition	Mean value of indicator \pm SD (sample size) for cause of death: acute VI/FI other		Statistical analyses	
			Welch 2 sample t -test	
		t	p	
Fulton's K	1.58 \pm 0.19 (27)	1.37 \pm 0.18 (41)	-4.64	<0.0001
Modified Fulton's K	5.55 \pm 0.65 (22)	4.98 \pm 0.69 (36)	208	0.001
Lipid quality				
% Lipid in adipose	59.38 \pm 10.19 % (12)	48.13 % \pm 28.42 (21)	101	0.182
% TAG in lipid	95.38 \pm 2.44 % (7)	76.43 % \pm 30.07 (14)	40	0.268
Lipid quantity				
Adipose mass index	0.209 \pm 0.104 (17)	0.151 \pm 0.092 (21)	112	0.026
Muscle mass index	0.126 \pm 0.024 (20)	0.129 \pm 0.030 (24)	257	0.66

To further investigate the validity of the Fulton's K body condition index, we compared Fulton's K scores of acute VI/FI turtles (1.58 ± 0.19 , mean \pm SD) and turtles with other causes of death (1.37 ± 0.18 SD) with scores from live, apparently healthy, turtles (1.58 ± 0.18) captured as part of another study. There were significant differences in Fulton's K scores among the 3 categories ($F_{2,78} = 13.46$, $p = 0.0001$), and pairwise comparison indicated that scores for live and acute VI/FI turtles were similar and both were significantly higher than scores for turtles that died from other causes (Fig. 4).

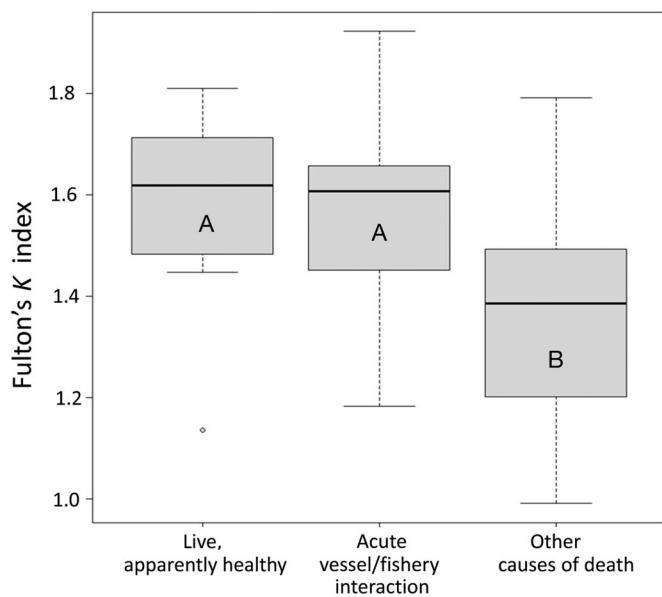


Fig. 4. Comparison of Fulton's K scores among live, apparently healthy loggerhead turtles caught for other research, turtles that died from acute VI/FI, and turtles that died from other causes. The turtles that died from other causes of death had significantly lower Fulton's K scores ($F_{2,78} = 13.46$, $p < 0.0001$) than the other 2 categories. Different uppercase letters indicate significant differences among groups (Tukey's post-hoc pairwise comparison, $p < 0.05$). See Fig. 3 legend for explanation of box-and-whisker plots

DISCUSSION

Presence of underlying conditions

Our results suggest the large majority of loggerhead turtles that die from acute VI/FI represent normal, healthy turtles in the population and, thus, are not predisposed to interaction with vessels or fishing gear because of underlying conditions. The turtles that died from acute VI/FI were significantly less likely to have lesions that would have compromised their health than those that died from other causes. This is similar to many cetaceans where the case definition, for example, 'death due to underwater entrapment in fishing gear' is often a diagnosis of last resort when an otherwise healthy animal has no obvious cause of death (Moore et al. 2013). One turtle categorized as acute VI/FI had prominent granulomatous enteritis and septic peritonitis that affected the intestine, pancreas and mesentery. This was the only turtle that died from acute VI/FI that appeared to be compromised prior to death, and it was an observed lethal take in a commercial trawl. Although the lesions in this turtle were serious, the cause of

death was drowning from forced submergence in trawl gear. Another interesting observation was that of the 14 confirmed fishery interaction cases that were either observed lethal takes or for which the turtle was removed dead from gear, 8 had no external marks indicating entanglement. Of these, 6 were observed takes in trawl gear, 1 was an observed take in a pound net and 1 was entangled in gill net gear. These observations strongly suggest that estimates of fishery interaction with these gear types based on presence of external marks on the carcass will result in an underestimate of interaction. In our 'other cause of death' category, 7 turtles had no significant lesions, no external lesions consistent with human interaction and no obvious cause of death. Because nearly half of the documented fishery interaction cases in our study had no external lesions consistent with entanglement, we suspect that the deaths of the 7 turtles with no significant lesions, as well as the 7 deaths due to drowning, could be due to fishery interactions, underscoring the fact that estimates of mortality of sea turtles from fishery bycatch using stranding data are most likely gross underestimates. These otherwise healthy turtles probably contributed to the high variability in quantitative measures for the 'other cause of death' category and may account for the lack of significant differences found for some of the variables investigated.

For loggerhead turtles in the 'other cause of death' category, we observed significant lesions consistent with encephalitis or meningoencephalitis (most often caused by severe parasitic infestation), sepsis, hepatitis/liver failure, and chronic pneumonia. Similar lesions have been documented for deceased sea turtles in Queensland, Australia (Flint et al. 2010) and Hawaii (Work et al. 2015). We also observed the following lesions and conditions suggestive of human interaction, but which could not be attributed directly to acute VI/FI by our conservative definition: ingested hook ($N = 7$), healing lesions consistent with vessel strike ($N = 3$), and acute pneumonia or aspiration of water consistent with drowning ($N = 7$). The exact cause of death for turtles with ingested hooks was difficult to determine post-mortem, but anorexia and/or lesions and infections resulting from untreated ingested hook and line can contribute to the decline and mortality of sea turtles (Orós et al. 2005, Parga 2012, Di Bello et al. 2013). Orós et al. (2005) found that approximately 19% of stranded loggerhead, green and leatherback sea turtles in the Canary Islands had ingested hooks and/or line, which were associated with ulcerative and fibrinous esophagitis, esophageal perforation, infiltrative bacteria,

and necrotizing enteritis. Di Bello et al. (2013) found that of 48 turtles with fishing line extending from the mouth, 15 were in extremely poor condition. In a discussion of the implications of longline fisheries interactions, Parga (2012) notes that turtles released from longlines with gear attached are at greater risk for post-release mortality than those released after removing all gear. In our study, of the 7 stranded turtles discovered with ingested hooks, 1 was emaciated, 3 were thin, and 3 were categorized as having normal body condition. The long-term implications of ingested or embedded fishing gear for sea turtles requires additional study. Likewise, the ultimate impacts of complications resulting from chronic injuries due to vessel strike are not well-understood. We observed debilitation and heavy infestation of parasites in the 3 turtles presenting with evidence of healing vessel strike, which ultimately may have contributed to the death of these animals.

Validation of body condition indices

Since body condition indices are designed to measure adipose quantity (McKinney et al. 2014, Brown 1996), we used the AMI to validate the Fulton's K , modified Fulton's K and visual body condition data. Necropsy observations and photographs suggested that normal turtles had larger adipose depots in all body regions; thus we assumed here that higher AMI scores, measuring the adipose associated with the carapace, were reflective of greater quantities of adipose throughout the body. The trends in AMI and MMI scores among the visual body conditions (Fig. 3) suggest that decline in body condition may be similar to that observed during typical mammalian starvation where an individual initially draws upon energy reserves in its adipose mass through lipid catabolism, and protein catabolism occurs later in decline (McCue 2010). This starvation scenario has been documented naturally and experimentally in mammals (deCalesta et al. 1977, Delgiudice et al. 1990) and some birds (Lindgård et al. 1992, Duerr & Klasing 2014). Stegall et al. (1999) noted a similar manner of decline in starving harbor porpoises, with blubber (lipid) stores declining initially and muscle mass being reduced later in the starvation process. Such a pattern would follow the typical sequence of events in a fasting endotherm after carbohydrate depletion, in which lipids are oxidized for fuel by many organs, or are converted into ketone bodies for the central nervous system (CNS). The CNS still has a glucose demand, which is met by gluconeogenesis occurring

from a low level of protein breakdown. Only later in fasting/starvation (Phase III) does protein use accelerate in mammals, and at this point the situation becomes terminal (Castellini & Rea 1992). Duerr & Klasing (2014), however, found that protein catabolism declined at a steady rate in 2 species of seabird regardless of starvation phase but that lipid catabolism only occurred initially until the birds were 56 to 66 % of wild mean mass (e.g. between Phases II and III). It is important to recognize that lipid and protein utilization during starvation in ectotherms may be different from that observed in endotherms (McCue 2007, 2010). Considerable changes in metabolic activity may accompany starvation in some reptiles as was demonstrated in 3 snake species which adjusted metabolic demands to match resource availability (McCue 2007). It is unclear whether loggerhead sea turtles have a similar ability to adjust their metabolic rate in response to resource availability although it is clear that the metabolic rate changes with other environmental conditions, particularly temperature (reviewed in Williard 2013). The influence of environmental temperature on metabolic processes and fuel utilization may account for some of the variability we observed in the AMI and MMI data. Although we did not observe skeletal changes in cleaned carapace mass, skeletal atrophy leading to overall loss of body mass could have occurred elsewhere in the turtles' skeletal system.

Lipid quality data had trends similar to that observed for MMI, with values for emaciated turtles being significantly lower than values for normal turtles (Fig. 3). The Fulton's K scores for normal turtles were significantly higher than thin and emaciated turtles. Thus, although there was variability in the trends of relationships between quantitative indices and the qualitative body condition categories, in all cases (except for modified Fulton's K where there was no significant difference) the healthy (normal) turtles were distinguished from animals that were in poorer condition (either thin, emaciated or both). Variability in the indices may be expected since a decline in body condition is due to multiple factors, including the mass and lipid composition of adipose tissue as well as the mass and perhaps composition of muscle tissue. Taken together, these data support the use of qualitative visual body condition assessment based on consistent observation of carcasses or images.

While quantitative data may be preferable, visual assessment can be used to distinguish healthy from unhealthy individuals if body length and total body mass data are not available to calculate body condi-

tion scores. Visual assessment is only useful, however, if animals are very fresh (e.g. not bloated) and are assessed in a consistent manner. To provide benchmark data for comparison we recommend using a standard set of images including: frontal view in both ventral and dorsal recumbency, lateral and oblique view in ventral recumbency and ventral view in dorsal recumbency (see the Supplement).

Both the Fulton's K and modified Fulton's K body condition scores were significantly positively correlated with AMI suggesting that as a sea turtle's body condition declines, the loss of total body mass is due, at least in part, to a corresponding loss of adipose tissue. The lipid quality values were also positively correlated with Fulton's K , suggesting that loss of adipose mass may be accompanied by loss of lipid and TAG in the adipose tissue. The weakness of the correlations between body condition scores and indices of lipid quantity and quality may reflect the difficulty in consistently dissecting sea turtles using a number of different prosector, but it may also indicate that while tissue quality declines, mass may not decline. Fresh turtles are a fairly rare occurrence in Virginia and mass dissections were conducted by experienced staff as each turtle was brought to the facility. Freezing fresh turtles for 1 or 2 prolonged necropsy sessions could have addressed the inconsistencies in dissection technique, but it would have compromised the pathology data which is much easier to assess when collected from unfrozen tissues. It is also possible that serous fat atrophy, where the adipose tissue has a watery or gelatinous consistency (Flint et al. 2009), inflated the mass of adipose tissue in turtles with poor body condition.

Between the 2 morphometric body condition indices, the Fulton's K data showed significant differences among the 3 visual body condition categories and were normally distributed where the modified Fulton's K values were neither significantly different nor normally distributed. This may have been due to smaller sample sizes for the modified Fulton's K data, but the use of body depth measurements could also have been problematic in the modified Fulton's K formula. Body depth in sea turtles changes drastically with bloating from decomposition in dead turtles and with breathing in live turtles. Although the turtles in this study were considered fresh dead, slight bloating may not have been obvious in thin or emaciated turtles. Even slight bloating would change the body condition score in the modified Fulton's K formula. For example, a change of 1.0 cm in body depth would change the modified Fulton's score of turtle from 4.86, the mean for emaciated turtles, to 4.90, the

mean for thin turtles. Since it requires fewer measurements, can be conducted on slightly bloated carcasses and is correlated with adipose quantity, the Fulton's K score may be a more appropriate morphometric measure of body condition in loggerhead turtles than the modified Fulton's K index we developed.

Adipose quantity as measured by an AMI supports use of both the qualitative and Fulton's K indices for analysis of body condition. For fresh animals for which it is not possible to collect body mass, the addition of 3 to 4 standard photographs can assist with assessing body condition visually. The Fulton's K body condition index relies on standard carapace length and body mass measurements that should be routinely and consistently collected. Body length and mass are less likely to dramatically change with decomposition compared with body depth, which clearly increases as a carcass begins to bloat from decomposition. Thus, as a morphometric index, Fulton's K is likely to be more useful than the modified Fulton's K for retrospective analyses of body condition based on data routinely collected by the Sea Turtle Stranding and Salvage Network (www.sefsc.noaa.gov/species/turtles/strandings.htm). While this study focused on stranded turtles that were not decomposed, a simple body condition index such as the Fulton's K index could probably be applied to intact, moderately decomposed carcasses to compare relative adipose quantity among a larger group of strandings. A controlled study to assess changes in length and mass with decomposition could determine whether Fulton's K is appropriate to apply to moderately decomposed, intact carcasses.

Measures of adipose quantity and quality between cause of death categories

Our results show that turtles that die from acute VI/FI are in better body condition, with greater adipose stores, than turtles that die from other causes (see Table 2). To further validate this result, we compared the Fulton's K scores from dead turtles in both categories (acute VI/FI and other causes) to those from live turtles captured as part of an ongoing research project. The mean Fulton's K values were not significantly different between acute VI/FI cases and live turtles, but both were significantly higher than scores for other causes of death. This result supports the hypothesis that turtles that die from acute VI/FI are otherwise healthy animals, and corroborates findings from previous studies (Orós et al. 2005, Flint et al. 2010, Work et al. 2015). Flint et al. (2010),

found that green sea turtles in Australia that died from traumatic injury had less incidence of heavy spirorchid infestation than turtles that presented with evidence of disease, and Orós et al. (2005) describe only lesions associated with the trauma of net entanglement and vessel impact for turtles where mortality was human induced. Work et al. (2015) found that green turtles in the eastern tropical Pacific were in poorer body condition if cause of death was due to nutritional or infectious/inflammatory causes versus trauma or physiological causes.

There were no significant differences in adipose quality between acute VI/FI and other causes of death, probably due to the high variance in the other cause of death category (see Table 2). This variance may be due to 2 factors. First, there were most likely otherwise healthy turtles that died as a result of fishery interaction included in the other cause of death category but were not considered acute VI/FI due to our conservative criteria. Since these undiagnosed acute VI/FI cases represented otherwise healthy animals, the other cause of death category had substantially higher variance than the VI/FI category (see 'Presence of underlying conditions' above). Furthermore, across cause of death categories, there were no significant differences in lipid quality between turtles that were qualitatively assessed as normal and thin. Since 61 of the 69 turtles were either normal or thin and only a few were considered emaciated, the imbalance in sample size and relatively high variance among emaciated turtles could contribute to the lack of significance in lipid quality among cause of death categories. Our results suggest that although adipose quantity changes as turtle body condition begins decline, adipose quality doesn't change significantly until turtles become very thin or emaciated (see Fig. 3). As such, while lipid quality provides insight into starvation in turtles, it is not the best variable to investigate a wider range of body conditions.

Management implications

The data analyzed for this study represent a decade of detailed necropsy and pathology assessments of fresh, dead loggerhead turtles that stranded in Virginia or were observed lethal takes brought to us with the help of federal fisheries observers. The lack of significant pathology and higher body condition scores of the loggerhead turtles that died from acute VI/FI suggests these animals were otherwise healthy and were not predisposed to vessel or fishery interactions because of underlying conditions. Of the 56

cases of stranded loggerhead turtles in the study (excluding the observed takes), at least 26 were healthy animals that died from acute VI/FI, and an additional 14 turtles were suspected to have been fishery interactions based on evidence of drowning or lack of significant lesions (see 'Presence of underlying conditions'). Thus, between 46 and 71% of the fresh, dead stranded turtles in Virginia from 2004 to 2013 died from human-induced mortality. These numbers are similar to other regions with high anthropogenic mortality, such as the Mediterranean coast of Spain (Tomás et al. 2008) where approximately 50% of sea turtle mortality (primarily loggerhead turtles) was found to be due to anthropogenic causes, mostly related to the longline fishery. Similarly, the majority of loggerhead and leatherback turtle mortality along the Atlantic and Mediterranean coasts of Portugal is due to fishery interaction (Nicolau et al. 2016). Nearly 63% of live loggerhead turtle cases admitted into a rehab center on Grand Canary Island from 1998 to 2014 were fishery related mortality with most being entanglements (Orós et al. 2016).

Fresh dead animals represented less than 4% of the total number of loggerhead turtles in the Virginia stranding record from 2004 through 2013. A few live stranded turtles later died and were included with the animals that stranded in fresh condition, but overall the turtles we were able to examine for cause of death based on veterinary pathology reports represented less than 5% of the total number of strandings. If documented causes of death for fresh, dead carcasses are representative of all stranded loggerhead turtles, then conservatively, between 793 (46%) and 1329 (71%) of the 1872 loggerhead turtles that stranded in Virginia from 2004 through 2013 may have died from human-induced mortality; this number represents approximately 100 loggerhead turtles per year in Virginia alone.

While external signs of fishery interaction in sea turtles are either cryptic or non-existent, signs of vessel interaction are usually obvious in stranded turtles. All of the 15 acute vessel interaction cases we reviewed exhibited no evidence of other significant lesions and were otherwise healthy turtles. In Virginia, we have consistently examined stranded turtles for signs of human interaction since 2009. From 2009 to 2013, 210 loggerhead turtles showed signs of vessel interaction, which represents 27% of all stranded loggerhead turtles, higher than eastern Spain (Tomás et al. 2008), Portugal (Nicolau et al. 2016) or Hawaii (Work et al. 2015). Interestingly, vessel interactions represented 25% (15 of 60) of the fresh, dead stranded loggerhead turtles we exam-

ined for this project. We view this as evidence that the rates of death by vessel interaction for fresh, dead loggerhead turtles may be similar to those for all stranded turtles. We believe that this rate of anthropogenic mortality is unsustainable, particularly if loggerhead turtles in other regions of the United States experience similarly high levels of fishery and vessel related mortality. Protected species managers need to continue to target vessel strikes and fishery interactions to mitigate high mortality in areas such as Virginia.

One explanation of the high vessel-related mortality is the high number of both commercial and recreational vessels in our region. The lower Chesapeake Bay and coastal ocean waters of southeastern Virginia are areas of high commercial shipping, naval activity, recreational and commercial fishing and pleasure boating. An alternative or complementary explanation may be related to loggerhead swimming and diving behavior. Loggerheads are a relatively slow moving sea turtle and have been shown to be more susceptible to threats, such as shark predation, than similarly sized green turtles in Shark Bay, Australia (Heithaus et al. 2008). Unpublished dive histogram data from tags deployed on loggerhead turtles in Virginia from 2009 to 2013 suggest that loggerheads in Chesapeake Bay spend from $22.1 \pm 29.8\%$ (mean \pm SD) in fall to $38.5 \pm 26.9\%$ in spring of their time within 1 m of the surface where they would be most susceptible to vessel strike (S. Barco et al. unpubl. data).

In Virginia, sea turtles are only present in the warmer months, primarily from May to October (Mansfield et al. 2009). The potential level of mortality of healthy turtles from human-induced mortality in the region appears to be quite high and may be one reason that loggerhead turtle population numbers appear to be consistently low and not increasing in some areas despite decades of protection and mitigation efforts (Work et al. 2010, Finkbeiner et al. 2011, Warden 2011,). This study demonstrates how consistently collected data and professional examination of stranded sea turtles can contribute to management and conservation efforts.

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