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CORRELATIONS OF CAPTURE, TRANSPORT, AND NUTRITION WITH SPINAL DEFORMITIES IN SANDTIGER SHARKS, *CARCHARIAS TAURUS*, IN PUBLIC AQUARIA

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Abstract: A number of captive sandtiger sharks (*Carcharias taurus*) in public aquaria have developed spinal deformities over the past decade, ranging in severity from mild curvature to spinal fracture and severe subluxation. To determine the frequency and etiologic basis of this disease, U.S. public aquaria participated in a two-stage epidemiologic study of resident sharks: 1) a history and husbandry survey and 2) hematology, clinical chemistry, and radiography conducted during health exams. Eighteen aquaria submitted data, samples, or both from 73 specimens, including 19 affected sharks (26%). Sharks caught off the Rhode Island coast or by pound net were smaller at capture and demonstrated a higher prevalence of deformity than did larger sharks caught from other areas via hook and line. Relative to healthy sharks, affected sharks were deficient in zinc, potassium, and vitamins C and E. Capture and transport results lead to two likely etiologic hypotheses: 1) that the pound-net capture process induces spinal trauma that becomes exacerbated over time in aquarium environments or 2) that small (and presumably young) sharks caught by pound net are exposed to disease-promoting conditions (including diet or habitat deficiencies) in aquaria during the critical growth phase of their life history. The last hypothesis is further supported by nutrient deficiencies among affected sharks documented in this study; potassium, zinc, and vitamin C play critical roles in proper cartilage-collagen development and maintenance. These correlative findings indicate that public aquaria give careful consideration to choice of collection methods and size at capture and supplement diets to provide nutrients required for proper development and maintenance of cartilaginous tissue.

Key words: Sandtiger shark, *Carcharias taurus,* spinal deformity, potassium, zinc, vitamin C, biochemical, clinical chemistry.

INTRODUCTION

The sandtiger shark (*Carcharias taurus*) is a popular exhibit specimen among public aquaria worldwide. At least 202 individuals are displayed by 39 institutions,[2] with a significant proportion of those individuals possessing spinal deformities of varying severity; however, spinal deformities have never been documented in wild *C. taurus*. The prevalence of spinal deformities in aquarium specimens prompted Berzins et al.[8,10] to launch a survey documenting the syndrome, which revealed 33 cases, representing a third of *C. taurus* held in aquaria at that time.[1] This work led to clinical description of the syndrome[14,35] and many etiologic hypotheses.

Survey results demonstrated spinal curvature characterized by scoliosis and kyphosis of the spinal column, with single or multiple incremental subluxations and occasional compressed spinal bodies. Skeletal tissue revealed ineffective remodeling, cartilaginous proliferation, excessive mineralization of vertebrae, and interspinal degeneration. The surrounding epaxial muscle revealed degeneration and fibrosis, and gingival hyperplasia, jaw protrusion, and curled pectoral fins were sometimes associated clinical signs.[9,14] Behavioral sequelae included loss of forward speed, use of one of the pectoral fins to equilibrate posture, stalling, and subsequent sinking to the tank floor.[35]

Several causative cofactors of spinal deformity in *C. taurus* have been hypothesized[8,35] over the past decade, including nutritional deficiencies (especially vitamin C and zinc), spinal stress caused by high growth rates, capture-transport trauma, atypical swimming behavior induced by aquarium habitats, and infection. This study examined correlations among capture, transport, and nutrition with occurrence of spinal deformity in *C. taurus*.

Capture and transport

The spinal lesions found in *C. taurus* typically occur anterior to the cranial dorsal fin,[8,35] at or near the angular vertex about which sharks swim or thrash. Sharks may endure trauma to the spine during capture, transport, or both as a result of thrashing or gravitational loading on the spinal column in the absence of the buoyant force of water (i.e., while being lifted out of the water).

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Subclinical fractures or subluxations instigated by these events may become exacerbated over time as a result of 1) other localized forces that impinge upon the spinal axis in this location during normal locomotion, 2) poor healing capability of elasmobranch cartilage,[4] and 3) impairment of healing via abnormal collagen formation in sharks with nutritional deficiencies (see "Nutrition" below).

Nutrition

The authors of previous studies have suggested that deficiencies in vitamin C and zinc may result in abnormal cartilage development and maintenance. Collagens are the primary protein constituent of connective tissues such as cartilage, bone, ligament, and tendon and are responsible for the tensile properties of these tissues. Hydroxylation is the process by which tropocollagen triple helices are formed from polypeptide subunits. These triple helices are subsequently bundled to form collagen fibrils, which are organized into the collagen fibers of connective tissues.[19,20,30,32] Hydroxylation is dependent on a series of reactions involving enzymes that require vitamin C as a cofactor.[22,33,36] Vitamin C deficiency, therefore, results in a series of connective tissue disorders in many taxa, including spinal deformities.[42] Scorbutic teleosts characteristically present with lordosis and scoliosis of the spinal column, characterized by severe spinal dislocations, hyperplasia of cartilagenous tissue, and other connective tissue abnormalities.[5,15,26,27,44] Furthermore, excessive concentrations of vitamins A and E may also interact to inhibit vitamin C absorption.[11,15]

Zinc is also an essential element in processes of collagen synthesis and turnover. It is a critical component in a number of metalloenzymes,[25] in particular alkaline phosphatase, an enzyme that plays important roles in the maturation and degeneration of epiphyseal plate cartilage (in chicks),[43] and bone collagenase, which is crucial to collagen turnover.[40] Zinc also promotes the anchorage of cartilage oligomeric matrix protein (important in cartilaginous cell growth and matrix development) to collagens I and II within the extracellular matrix of cartilage.[37]

Newborn and infant rhesus monkeys (*Macaca mulatta*) deprived of zinc present with delayed skeletal maturation and defective mineralization similar to that seen in human rickets.[25] Chicks (*Gallus gallus domesticus*) deprived of zinc present with shortened and thickened leg bones and swollen, twisted joints characterized histologically by hypertrophied chondrocytes that are diffusely distributed in the extracellular matrix in areas of the proliferating region of the epiphyseal plate remote from blood vessels.[40,43] Skeletal deformities associated with zinc deficiency may be exacerbated as a result of abnormal weight gain as well. Zinc-deficient rats (*Rattus norvegicus*) shifted incorporation of proline, an important amino acid involved in protein synthesis, away from skin protein and skin collagen and into liver and plasma proteins, which was closely correlated with body weight gain in these animals.[7]

Objectives

A new survey was launched to investigate the etiology of spinal deformities in this species. Data were requested on collection and transport locale and methods, size at capture, morphometrics, diet, and vitamin supplementation. Respondent institutions were requested to provide radiographs for confirmation of deformity and blood for leukocyte differential, hematocrit, clinical chemistry, and vitamin assays. From these data, correlations between clinical signs and the etiologic hypotheses discussed above were elucidated.

MATERIALS AND METHODS

Participating U.S. aquaria holding *C. taurus* individuals were requested to collect data and tissue samples in two stages along the clinical timeline of both apparently normal as well as grossly affected sharks.

Stage I

The Stage I survey collected both historical and husbandry data. Husbandry personnel were requested to complete the survey on living sharks. The survey requested data on history (sex, acquisition date, collection method and locale, and time to onset of deformity, if affected) and morphology (length, weight, girth, Fulton condition factor [K] as kg/m^3)[3] at acquisition, at onset of disease (if affected), and at euthanasia, as well as additional morphologic data over time for growth comparisons. Data on clinical signs of disease (including location of abnormality; presence of kyphosis, scoliosis, or both; the presence or absence of gingival hyperplasia; jaw protrusion; curled fins; open sores or lesions; chronically flared gills; and photographs) and diet (food items fed, mass per feeding, frequency of feeding, and type and amount of vitamin supplementation) were requested as well.

Stage II

The Stage II survey requested nonlethal diagnostics of sharks during regularly scheduled health exams. Radiographs of spinal columns were requested (in both dorsoventral and lateral views) for illumination and confirmation of spinal deformities. Phlebotomy was requested for hematocrit, leukocrit, leukocyte differential, and assays of plasma for clinical chemistry, including zinc and concentrations of vitamins A, C, and E. For hematocrit and leukocrit values, blood was collected in heparinized microhematocrit tubes and spun for 5 min at 2,000 *g*. Leukocyte differentials (% of 100 leukocytes counted) were obtained by counting heterophils, fine eosinophilic granulocytes, coarse eosinophilic granulocytes, lymphocytes, and monocytes visualized from blood smears stained with HemaStat (Fisher Scientific, Pittsburgh, Pennsylvania 15275-1126, USA) at ×1,000. Each slide was read twice, and the means of each leukocyte type were calculated. For clinical chemistry, remaining blood was spun in lithium heparin tubes for 15 min at 1,200 *g*. An aliquot of serum was prepared with an equal amount of cold 10% metaphosphoric acid solution and stored at −80°C until submission to the Montefiore Medical Center (Bronx, New York 10466-2604, USA) for vitamin C (ascorbic acid) concentration assay. Serum ascorbic acid was measured spectrophotometrically using 2,4-dinitrophenylhydrazine as chromogen,[16] a method that has been shown to correlate highly with high-performance liquid chromatography (HPLC) analysis.[16,38] Two additional aliquots of serum were stored at −80°C; one aliquot was subsequently submitted to the Montefiore Medical Center for vitamins A and E assays, measured by reversed-phase HPLC and ultraviolet–Vis detection.[39] The second additional aliquot was subsequently submitted to Idexx Laboratories (St. Petersburg, Florida 33716-2305, USA) for a clinical chemistry panel of 14 analytes (albumin, albumin:globulin ratio, alkaline phosphatase, aspartate transaminase, calcium, chloride, cholesterol, creatinine, globulin, glucose, phosphorous, potassium, total protein, and zinc).

Statistical analysis

χ^2 tests were employed to assess associations of incidence of spinal deformity with sex, other connective tissue abnormalities, capture method, locale, food items fed in the most recent year, and vitamin supplementation. Patterns were observed between capture location and size at capture; this relationship was tested in a one-way analysis of variance (ANOVA). A two-way ANOVA was employed to assess differences in K between healthy and affected animals, partitioning out the effect of sex. A repeated-measures ANOVA was employed to assess number of different food items fed per year and percentage of body weight fed weekly (on an as-fed basis) to healthy vs. affected animals over a 4-yr period from the time of the survey to 3 yr prior. Clinical chemistry parameters, including vitamin and zinc concentrations, were tested between healthy and affected animals with either a *t*-test or a Mann–Whitney *U*-test, depending on the normality of data distributions. Hematocrits and leukocrits were arcsine transformed, leukocyte differentials were square-root transformed; both sets of data were tested between healthy and affected animals with *t*-tests. Analyses were completed with Excel (v. 2007; Microsoft Corporation, Redmond, Washington 98052-7329, USA), Analyse-it (v. 2.22; Analyse-It Software, Ltd., Leeds, LS3 1HS, United Kingdom), and Minitab (v. 15.1.30.0; Minitab, Inc., State College, Pennsylvania 16801-3008, USA).

RESULTS

Eighteen institutions responded to the Shark Spine Study, returning Stage I survey data from 54 healthy sharks and 19 with spinal deformity (representing a prevalence rate of 26%) and Stage II blood samples for hematology and clinical chemistry from 23 healthy sharks and 10 with spinal deformity.

There was no association between spinal deformity and other connective tissue abnormalities ($\chi^2 = 1.39$, df = 1, $P > 0.10$). Among sharks without connective tissue abnormalities, 19.6% presented with spinal deformities, while 33.3% of sharks with connective tissue abnormalities also presented with spinal deformities. There was no association with sex (males, 27.1%; females, 26.6%; $\chi^2 < 0.001$, df = 1, $P > 0.99$).

In consideration of the contribution of collection methods to spinal deformity, animals caught by pound net were more likely to develop spinal deformity (44.4%) than were animals caught by hook and line (8.3%, $\chi^2 = 7.84$, df = 1, $P < 0.01$). Animals captured off the Rhode Island coastline were more likely to develop spinal deformity (46.6%) than were animals captured in other areas (New Jersey–Delaware coastlines, 5.9%; Maryland–Virginia coastlines, 16.6%; $\chi^2 = 7.88$, df = 2, $P < 0.025$; Fig. 1). However, all sharks caught in pound nets were caught off the Rhode Island

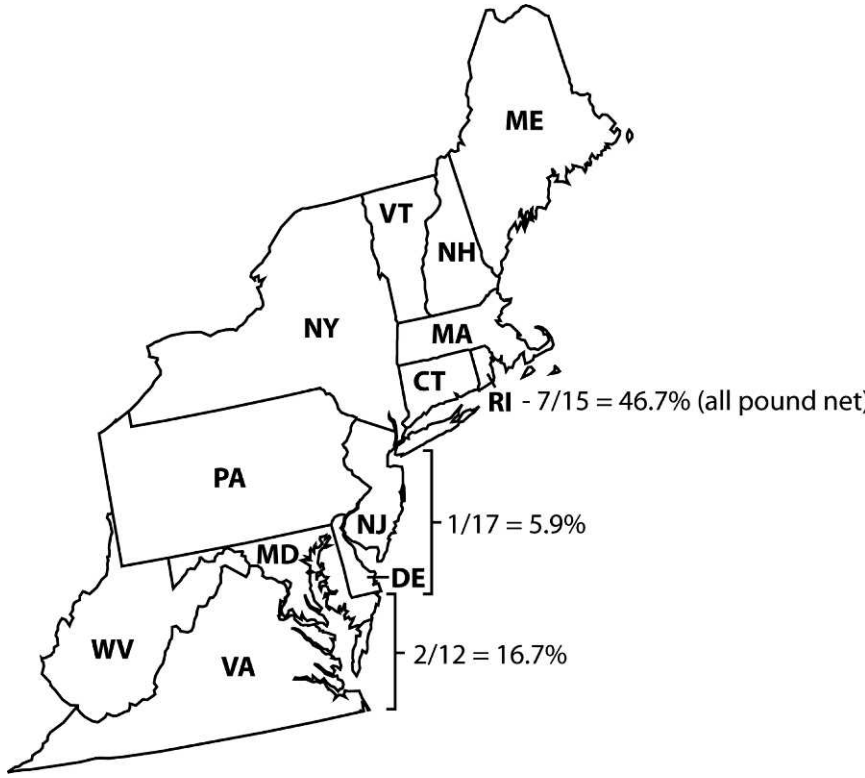

Figure 1. Prevalence of spinal deformity among captive sandtiger sharks (*C. taurus*) in public aquaria, by original collection locale along the northeast U.S. coastline.

coastline, and all sharks caught in Rhode Island were captured by pound net ($n = 15$), indicating differences in collection methods among collectors operating in different locales. Sharks captured by pound net in Rhode Island were also smallest at capture (mean ± standard error of the mean total length = 121.24 ± 3.39 cm in RI vs. 188.96 ± 10.56 cm in other locales; ANOVA, $F_{7,22} = 8.72$, $P < 0.001$), and sharks with spinal deformities were also smaller at capture than were healthy sharks in general, regardless of collection locale or method (121.79 ± 8.04 cm vs. 185.95 ± 10.30 cm, $t = 4.91$, df = 27, $P < 0.001$).

Once in aquaria, animals presented with spinal deformity by a median of 4.0 yr (interquartile range: 3.0–8.8 yr). Affected sharks demonstrated a higher K than healthy sharks (ANOVA, $F_{1,55} = 4.51$, $P = 0.038$; Table 1); this was absent of any significant differences in 1) the types of foods fed in the most recent year ($\chi^2 = 5.92$, df = 13, $P > 0.90$; Fig. 2), 2) the number of different food items fed annually over a 4-yr period (healthy sharks were fed an average of 3.6 ± 0.1 different food items annually, while affected sharks were fed an average of 3.7 ± 0.2 different food items annually; ANOVA, $F_{1,107} = 039$, $P = 0.532$), or 3) the amount of food sharks were fed per kilogram of body mass per week (healthy sharks were fed 2.25 ± 0.17% of body mass on an as-fed basis weekly, while affected sharks were fed 1.87 ± 0.13% of body mass weekly; ANOVA, $F_{1,81} = 3.26$, $P = 0.069$). Approximately half (52.6%) of the sharks received vitamin supplementation with either Mazuri Vita-Zu Shark/Ray Tabs I (5M24) or II (5MD8, PMI Nutrition International LLC, St. Louis, Missouri 63166-6812, USA); in some surveys it was not clear which of the two products was offered, but in general, supplementation was not associated with the presence or absence of spinal deformity ($\chi^2 = 0.38$, df = 1, $P > 0.50$). However, sharks with

Table 1. Average Fulton condition factor (K, kg/m^3)^3 of sandtiger sharks, *C. taurus,* in public aquaria, with and without spinal deformity, by sex (mean ± standard error).

Condition	Male	Female
Healthy	6.16 ± 0.15	7.09 ± 0.29
Affected	6.78 ± 0.54	8.06 ± 0.87

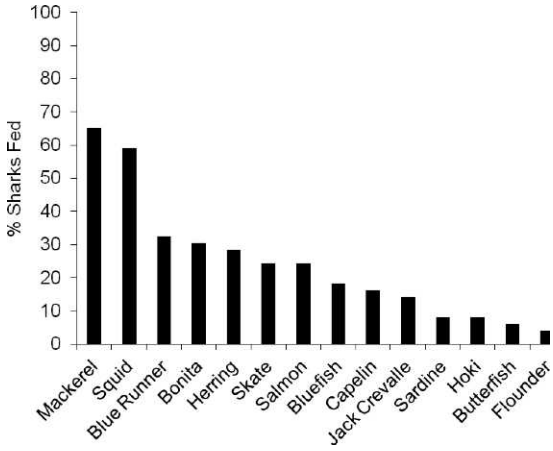

Figure 2. Prevalence of food items fed to captive sandtiger sharks (*C. taurus*), in public aquaria.

spinal deformities demonstrated lower serum concentrations of several nutrients, including vitamins C and E, potassium, and zinc (Table 2).

Hematologic results were unremarkable. There were no significant differences in percent representation of leukocyte types among the leukocyte population between healthy and affected sharks or in hematocrit or leukocrit (Table 3).

DISCUSSION

Capture and transport

Factors associated with initial capture from the wild are strongly associated with the development of spinal deformity in *C. taurus*. Sharks that were caught at small size were often captured by multiple collectors off the coast of Rhode Island by means of pound net and showed a higher prevalence of deformity among the population in public aquaria. This leads to three alternative hypotheses.

First, sharks caught off the Rhode Island coastline may be genetically predisposed to the development of spinal deformities. This hypothesis seems unlikely given the genetic mixing opportunities likely to result from its migratory behavior. *Carcharias taurus* has demonstrated maximum migration distances of 1,187 km off

Table 2. Clinical chemistry values of healthy and affected sandtiger sharks (*C. taurus*). For parametric data, reference ranges are reported as 90% and 95% confidence intervals (CIs). For nonparametric data, the interquartile (IQ) range is reported as a reference range. Subscripts for *t*-test statistic represent degrees of freedom and for *U*-test statistic represent sample sizes of each category.

	Healthy			Affected		
	Mean ± standard error	Reference range 90% CI	Reference range 95% CI	Mean ± standard error	Test statistic	P[a]
Parametric						
Albumin (g/dl)	0.46 ± 0.02	0.42–0.50	0.41–0.51	0.46 ± 0.03	$t_{20} = 0.071$	0.944
Albumin:globulin ratio	0.15 ± 0.02	0.12–0.17	0.11–0.18	0.16 ± 0.02	$t_{20} = 0.375$	0.712
Calcium (mg/dl)	13.91 ± 0.28	13.45–14.38	13.36–14.47	13.63 ± 0.19	$t_{20} = 0.652$	0.522
Chloride (mEq/L)	270.7 ± 10.6	253.3–288.1	249.9–291.4	242.1 ± 8.8	$t_{20} = 1.704$	0.104
Cholesterol (mg/dl)	74.8 ± 4.5	67.4–82.2	65.9–83.7	63.6 ± 9.1	$t_{20} = 1.245$	0.227
Globulin (g/dl)	3.22 ± 0.15	2.97–3.47	2.92–3.52	2.97 ± 0.15	$t_{20} = 1.016$	0.322
Glucose (mg/dl)	34.0 ± 1.6	31.4–36.6	30.9–37.1	32.7 ± 2.7	$t_{20} = 0.434$	0.669
Phosphorous (mg/dl)	6.40 ± 0.23	6.02–6.78	5.94–6.86	5.90 ± 0.39	$t_{20} = 1.155$	0.262
Potassium (mEq/L)	4.75 ± 0.26	4.32–5.19	4.24–5.27	4.09 ± 0.14	$t_{20} = 2.230$	**0.037**
Total protein (g/dl)	3.68 ± 0.15	3.43–3.93	3.38–3.98	3.43 ± 0.16	$t_{20} = 0.996$	0.331
Vitamin A (μg/dl)	18.75 ± 1.83	15.74–21.76	15.17–22.34	14.07 ± 3.69	$t_{19} = 1.269$	0.220
Vitamin C (mg/dl)	0.622 ± 0.047	0.545–0.699	0.531–0.713	0.412 ± 0.114	$t_{19} = 3.260$	**0.002**[b]
Vitamin E (mg/dl)	0.789 ± 0.062	0.688–0.891	0.668–0.911	0.412 ± 0.098	$t_{19} = 3.260$	**0.004**
Zinc (mg/L)	0.76 ± 0.05	0.68–0.84	0.66–0.85	0.47 ± 0.04	$t_{19} = 2.067$	**0.026**[b]
Nonparametric	Median	IQ range		Median	Test statistic	P
Alkaline phosphatase (U/L)	6	5–12		5	$U_{15,7} = 37.5$	0.332
Aspartate transaminase (U/L)	31	18–40		12	$U_{15,7} = 26.0$	0.066
Creatinine (mg/dL)	0.0	0.0–0.1		0.0	$U_{15,7} = 41.0$	0.448

[a] Significant *P*-values are in bold.
[b] One-tailed test.

Table 3. Hematologic parameters of sandtiger sharks (*C. taurus*). The interquartile (IQ) range is reported as the reference range. Subscripts for *t*-test statistic represent degrees of freedom.

Parameter	Healthy		Affected		
	Median	IQ range	Median	Test statistic	*P*
Hematocrit (%)	25.7	22.5–29.5	23.5	$t_{21} = 0.873$	0.393
Leukocrit (%)	2.8	2.0–6.9	1.7	$t_{21} = 1.688$	0.106
Leukocyte differential					
Coarse eosinophilic granulocytes (%)	21.0	17.0–28.0	23.0	$t_{21} = 0.006$	0.995
Fine eosinophilic granulocytes (%)	8.5	3.8–15.0	14.0	$t_{21} = 0.334$	0.742
Heterophils (%)	16.5	9.0–22.3	14.0	$t_{21} = 0.913$	0.372
Lymphocytes (%)	38.5	32.3–48.3	45.5	$t_{21} = 0.386$	0.703
Monocytes (%)	1.5	0.8–2.5	2.3	$t_{21} = 0.890$	0.383
Heterophil:lymphocyte ratio	47.2	23.2–66.8	39.1	$t_{21} = 0.630$	0.536

the east coast of the United States,[23] 1,897 km off the east coast of South Africa,[13] and 1,150 km off the east coast of Australia.[6] The geographic locales categorized in this study all fall well within the range of these reported maximum migration distances, rendering a hypothesis of genetic isolation implausible in a range of this scale.

Second, the methods associated with pound-net capture and transport may cause more trauma to the spinal column of sandtiger sharks than would be suffered during hook-and-line capture and transport. At first consideration, a pound net is a rather nonstressful method of capture. Sharks simply swim into a large, netted trap that features ample swimming space.[12] However, the process of gathering and transferring sharks from the crib of the net onto a holding tank onboard a vessel is a potentially traumatic operation depending upon the extent of thrashing during gather and transfer and the number of sharks caught at any one time. Transfer in the absence of the buoyant force of water places previously unencountered stress on the spinal column. While sharks caught via hook and line are likely to be exposed to a similar and potentially traumatic transportation chain (e.g., from vessel to temporary holding tank to transport truck to destination, etc.), the difference lies in the transfer from the collection gear to the vessel; landing a shark from a hook and line may constitute a less traumatic operation.

Finally, sharks caught by pound net off the Rhode Island coastline were also smaller than sharks collected in other locales and typically by hook-and-line methods. The prevalence of spinal deformities in this cohort may be alternatively explained by the small size (and presumably young age) at which these sharks were brought into public aquaria. These animals were exposed to aquarium living conditions and diets that may have contributed to the development of spinal deformity early on in their life histories, during a critical phase of growth. This hypothesis is supported by the relatively early onset of affliction and deficiencies in nutrients that are critical to proper cartilage development and maintenance among sharks in public aquaria presenting with spinal deformities.

Hypotheses 2 (capture method) and 3 (size at capture) are not necessarily mutually exclusive. Capture and transport during a critical growth phase may cause initial injury, which could be exacerbated over time by inadequate living conditions and diets.

Nutrition

To the authors' knowledge, the first published reference ranges of clinical chemistry parameters of *C. taurus* are presented in this study. Haulena[17] reported several identical clinical chemistry parameters in up to seven *C. taurus*, all juveniles of 2–3 yr in age and all held at one public aquarium. A comparison of Haulena's data against that of the current study reveals striking differences. Of measured biochemical parameters in common, Haulena's measurements of chloride, potassium, and total protein concentrations were below this study's reported reference ranges, while calcium, cholesterol, glucose, and phosphorous concentrations were above this study's reported reference ranges. These comparisons may reveal differences in 1) biochemical status of juvenile vs. adult sharks or 2) nutritional status of sharks held in this singular public aquarium vs. the average nutritional status of 15 sharks held among the six public aquaria included in Stage II of this study.

Despite similarities in diets consumed between healthy and affected sharks, sharks with spinal deformity were deficient in several nutrients that play key roles in cartilage development and maintenance. Potassium plays an important role

in regulating the membrane potentials of chondrocytes.[28,34] Although a discrete connection between chondrocyte membrane potential and spinal deformity is unclear, it is possible that altered membrane potentials could affect the cellular transport of biochemical agents involved in the development, maintenance, or mineralization of the extracellular matrix. The roles of vitamin C and zinc in cartilage development and maintenance, as well as clinical signs of deficiency, were outlined in the 'Introduction.' Originally it was hypothesized that excess concentrations of vitamins A, E, or both could inhibit vitamin C absorption. However, no excess concentrations of these nutrients were found among affected sandtiger sharks. To the contrary, affected sharks were also deficient in vitamin E. The role of vitamin E in animal metabolism is unclear, but in general, it is a fat-soluble antioxidant that stops the production of reactive oxygen species formed when fat undergoes oxidation.[18,31,41] Vitamin E deficiencies are characterized by ataxia, myopathies, neuropathies, retinopathy, and impairment of the immune response.[21,24] There is no evidence to date that a deficiency in this nutrient affects cartilage development and maintenance.

Affected sharks demonstrated higher body conditions without a concomitant increase in dietary intake. This may be partially explained by differences in the activity budget of healthy vs. affected sharks and also by the correlation between this finding and zinc deficiency in affected sharks. This correlation parallels Bates and Evans' findings in rats.[7] Zinc deficiency thus not only inhibits proper cartilage development and maintenance but may also shift deposition of nutrients into body storage compartments, leading to increased weight gain, which may place excessive force on a damaged spinal column, exacerbating the disease. Furthermore, the spinal columns of diseased sharks exhibited reduced flexural stiffness, necessitating greater muscular force generation to produce equivalent locomotive thrust, compared to healthy sharks, thereby compounding the effects of decreased activity budgets and excessive weight gain. Reduced flexural stiffness in diseased sharks is attributed to inadequate quantity and distribution of mineral within vertebrae, which may be related to the aforementioned nutritional deficiencies.[29]

Recommendations

Careful consideration should be given to the methods used to source sandtiger sharks for aquarium collections. Aquarium professionals should seek collaboration with experienced collectors who specialize in live shark capture and transport and who exercise great care along the chain of custody, from landing of the shark on board the vessel, to temporary holding, to final transport to the aquarium destination. Along the collection and transport timeline, the goal must be to minimize or eliminate the opportunity for trauma to occur, which could introduce a spinal fracture that may become exacerbated over the long term, developing into a spinal deformity. This process could include completely and evenly supporting the weight of the animal during removal from the water.

Given correlations between size at capture, nutritional deficiencies, and prevalence of spinal deformity, public aquaria may decide to source adult wild-caught sharks to avoid the potential for spinal deformities to develop among juvenile sharks subjected to exacerbating conditions in aquaria during a critical growth phase.

Supplementation of diets with vitamins C and E, potassium, and zinc is encouraged, as these nutrients play important roles in collagen-cartilage development and maintenance (except for vitamin E) and were deficient among sharks with spinal deformity. Many institutions supplemented diets with Mazuri Vita-Zu Shark/Ray tabs I (5M24) or II (5MD8), but neither supplement contains potassium, and only Tab II contains zinc (at 8.0 mg per 1.5-g tablet). Concentrations of vitamins C and E vary between the two versions, but additional supplementation focusing on these deficient nutrients is encouraged, and additional studies are warranted to determine recommended supplement dosages. Collectively, these efforts may ensure the long-term welfare of aquarium populations, thereby reducing dependence on wild stocks for aquarium exhibits.

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