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THE PHYSIOLOGICAL EFFECTS OF CATCH AND RELEASE ANGLING ON THE POST-RELEASE SURVIVORSHIP OF JUVENILE SANDBAR SHARKS (*CARCHARHINUS PLUMBEUS*)

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THE PHYSIOLOGICAL EFFECTS OF CATCH AND RELEASE ANGLING
ON THE POST-RELEASE SURVIVORSHIP
OF JUVENILE SANDBAR SHARKS
(*CARCHARHINUS PLUMBEUS*)

BY
ABBIEY LEIGH SPARGO

A THESIS SUBMITTED IN PARTIAL FULFILLMENT OF THE
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2001

MASTER OF SCIENCE THESIS
OF
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Abstract

A post release survivorship study of juvenile sandbar sharks (*Carcharhinus plumbeus*) was conducted in Delaware Bay during 1999-2000. A total of 104 sharks were captured and sampled for changes in blood chemistry after exposure to exhaustive exercise. Of these, 24 sharks were angled in the field, sampled, tagged, and released. The remaining 80 sharks were transported to a holding tank, allowed to recover, and half were angled to exhaustion. To quantify recovery, blood samples were taken from these fish at 0, 1.5, 3, 6, 10, 14 and 24 hours, then tagged and released. Blood was obtained by caudal puncture and analyzed immediately for blood gasses and glucose. Serum samples were sent to a commercial laboratory for the determination of blood metabolites, proteins, and electrolytes. Blood levels of lactate, PCO_2 , glucose, K^+ , Ca^{2+} , Mg^{2+} , and CK were elevated following the stressor, while pH and HCO_3^- levels declined. Most metabolites returned to baseline within 6-10 hours. Moreover, 5 sharks were recaptured 0.03-12 months after release over the course of the study. These preliminary data indicate that sandbar sharks are able to physiologically recover after the exhaustive exercise associated with rod and reel angling and therefore, catch and release fishing may not severely impact neonatal and juvenile sandbar sharks in important nursery areas.

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Introduction

The sandbar shark (*Carcharhinus plumbeus*) is one of the sharks commonly found off the East Coast of the United States during the summer. They are highly migratory and usually inhabit warm temperate coastal waters (Branstetter 1990; Kohler et al. 1998). Due to their wide range, proximity to shore and excellent flesh, they have become a target for intense commercial and recreational fishing pressure. These sharks do not reach maturity until around 150-152 cm fork length (FL), and maturity can occur anywhere from 6-18 years or even longer (Springer 1960; Pratt and Castro 1990). Sandbar sharks can be considered K strategists with a low reproductive potential characterized by: late sexual maturity, one to two year reproductive cycles, small number of young and limited number of nursery grounds such as Delaware Bay (Springer 1960; Branstetter 1990; Musick 1995). The primary nursery grounds of the Western North Atlantic sandbar shark population ranges from South Carolina to New Jersey (Merson and Pratt 2001). Based on their life history strategies, the sandbar shark is highly susceptible to overfishing and stock depletion. Commercially, through 1995, sandbar sharks contributed 2/3 of the coastal shark fisheries catch in the North Atlantic (Musick 1995). During 1990-2000 the NMFS commercial landings data indicate that 15,727,340 pounds (lbs.) of sandbar sharks were caught (NMFS Commercial Data 2000). The recreational catch for sandbars during 1998-1999 was 2,377,148 lbs., which corresponds to approximately 55,000 sharks (Cortes 2000). The sandbar shark catch is second only to the blue shark (*Prionace glauca*) in U.S. recreational catches (Musick 1995).

Fisheries managers have implemented various regulations aimed at sustaining both the commercial and recreational shark fisheries. The last Shark Evaluation Workshop (SEW) held in 1998 proposed that the minimum size be raised to 140 cm and the total large coastal catch be reduced to 816 metric tons per year (Cortes 2000). The current recreational management plan for the sandbar shark allows only one shark to be kept per vessel per trip with a minimum size of 137cm FL (FMP 2000). More drastic measures have been taken for species that are considered to be in trouble. A Fisheries Management Plan for sharks of the Atlantic Ocean was developed in 1993 and a number of revisions have occurred since its inception. With the final implementation in July 1, 2000, 19 sharks were included on a prohibited species list. The wording of the current plan states that all fish caught must be released in such a manner as to ensure their maximum survival, with one stipulation that fish caught must be unhooked and released without ever leaving the water (FMP 2000). As any of these sharks caught incidentally cannot be kept, it is important to understand the amount of stress that these animals face during their capture and subsequent release.

The sandbar shark is not on the prohibited species list, however, other large coastal carcharhinids with similar life history strategies are, for example the dusky shark (*Carcharhinus obscurus*). The sandbar shark, therefore, is a good model to determine the effects of catch and release fishing and results can most likely be used as guidelines for similar species.

For recreational fisheries especially, catch and release angling has been widely promoted to preserve stocks, notably Atlantic salmon (*Salmo salar*). If recreational anglers are to continue fishing, a large percentage of fish must be returned to the waters

unharmful for future recapture (Wydoski et al. 1976; Schisler and Bergersen 1996).

While there is support for this plan, there is relatively little known about the ramifications of non-consumptive angling on the post-exercise survival and physiology of any species captured in this manner (Wilkie et al. 1996). As Wood et al. (1983) said, "it is important for research regarding exercise stress to be conducted since the results could render pointless the legislated return of trawled or angled fish to the water".

While studying the effects of stress during exhaustive exercise for cartilaginous and bony fishes, researchers have observed a number of physiological disturbances. These disturbances include: (1) a depletion of glycogen reserves and the build-up of lactate in white muscle caused by anaerobic glycolysis coupled with the depletion of ATP and creatine phosphate stores; (2) a marked decrease in blood pH resulting from metabolic (H^+) and respiratory (pCO_2 elevation) acidoses; (3) a profound disturbance of ionic, osmotic, and fluid volume homeostasis with hemoconcentration and increased plasma electrolytes; and (4) a rapid increase in circulating catecholamines, corticosteroids, and glucose levels (Wood 1991). Fish that experience these changes may require hours or days to fully recover (Kieffer et al. 1995).

Sharks can experience several types of environmental stressors: including those acutely caused through catch and release angling (Wood 1991), and those chronic changes related to long-term captivity (Barton 1997). The concept of stress itself has experienced many revisions. Brett (1958) provided a good working definition: "stress is a physiological state produced by an environmental factor that extends the normal adaptive responses of the animal, or disturbs the normal functioning to such an extent that the chances for survival are significantly reduced". Angling is considered to be the

most stressful capture method as it involves exercise, possible fright and air exposure (Milligan 1996), and is one of the most severe forms of exhaustive exercise (Booth et al. 1995). Exhaustive exercise is defined as the point at which the fish can no longer sustain burst swimming speeds, but may react with brief swimming motions if provoked (Wood 1991). Exhaustion occurs rapidly, with progression from steady state aerobic metabolism to anaerobic glycolysis within seconds or at most a few minutes (Wood 1991).

Anaerobic glycolysis in the white muscle fuels burst speed swimming; glycogen stores are reduced while the concentration of lactate is increased (Milligan and Girard 1993). The longer the exhaustive demand lasts, the greater the glycogen depletion and lactate accumulation (Driedzic and Hochachka 1978).

The literature on exercise physiology is growing rapidly, but as much as 95% of the total published work reports studies focusing on salmonids and other freshwater species (Lowe and Wells 1996). It has been well documented that exhaustive angling invokes detrimental physiological changes in salmonids (Wood et al. 1983; Parkhouse et al. 1988; Tufts et al. 1991). Kieffer et al. (1995) were among the first salmonid researchers to describe the relationship between the time taken to land a fish and the amount of physiological disturbances experienced; they found longer fight times invoked more profound physiological disturbances which extended recovery time.

Despite the wealth of information on salmonids and other freshwater species, current information on large pelagic teleosts and elasmobranchs is limited. Although the majority of knowledge is salmonid based, it would not be scientifically sound to directly apply the results and conclusions obtained to other species (Lowe and Wells 1996).

There is only limited literature regarding the responses of sharks to various stressors. Early studies such as Piiper et al. (1972) and Butler et al. (1978) focused on different species of dogfish because they were easy to capture and keep in captivity. Piiper et al. (1972) investigated the balance of H^+ ions and lactate ions in the blood of the larger spotted dogfish (*Scyliorhinus stellaris*) after severe exercise. They found that part of the H^+ generated in conjunction with lactate was not dispersed into the surrounding water, but was retained and buffered in the tissues. With advancements in aquarium techniques (Gruber and Keyes 1981), larger and more active sharks can now be transported and studied in captivity (Smith 1992). Bushnell et al. (1982) reported an increase in hematocrit and arterial pO_2 content during exercise in the lemon shark (*Negaprion brevirostris*). Most recently, Manire et al. (2001) analyzed serological changes associated with gill-net capture and restraint in the bonnethead (*Sphyrna tiburo*), blacktip (*Carcharhinus limbatus*) and the bull shark (*Carcharhinus leucas*).

Cliff and Thurman (1984) attempted to determine the pathological and physiological effects of stress during capture and transport in the juvenile dusky shark (*Carcharhinus obscurus*). They found much the same results as other exercise related studies (Rasmussen and Rasmussen 1968; Wells et al. 1986; Barham and Schwartz 1992). The sharks experienced an increase in lactate, glucose, electrolytes (Na^+ , K^+ , Cl^- , Ca^{2+} , Mg^{2+}) and enzymes such as creatine kinase (CK), coupled with a decrease in pH from both metabolic and respiratory acidosis. They also concluded that dusky sharks require approximately 24 hours to attain full metabolic recovery. Skomal and Chase (1997) concluded that short-term survivorship is not compromised in blue fin or yellow

fin tuna and blue sharks when exposed to greater than average stress levels during angling and handling.

To investigate the relationship between exhaustive exercise and recovery rates for the sandbar shark, a two-phase study was undertaken. The first phase involved a field study that would mimic the natural conditions facing sandbar sharks when they are subjected to angling and would quantify the effects of exhaustive exercise. The second phase used sharks in captivity to experimentally reproduce the recovery phase that would naturally occur after exposure to exhaustive exercise. The purpose of this study, was to quantify physiological changes in blood chemistry that occur during catch and release angling in sandbar sharks and to assess recovery and survivorship. This study attempted to assess blood parameters associated with stress and also the effect of independent environmental variables on the stress reaction. In the study, I test two hypotheses: the first is that there is a direct relationship between the length of time of capture and the physiological response; and secondly that the sandbar shark will take less than 24 hours to reach full physiological recovery.

Methods

Field Study

Shark Capture

To replicate as closely as possible the natural conditions facing juvenile sharks in Delaware Bay, DE (38.8° N 75.1° W), a field study was undertaken that exposed juvenile sandbar sharks to differing levels of exhaustive exercise. The purpose was to determine resting conditions and to quantify the effects of prolonged angling on these sharks. The group of juvenile sharks found in Delaware Bay consists of many different ages. Neonates or young of the year, and can be differentiated by the presence of an umbilical scar (Merson and Pratt 2001). Juvenile fish were considered to be in their second year of life or older. Both neonate and juvenile sharks were captured with rod and reel gear from the NOAA Vessel Stillwell, a 22 ft Boston Whaler, from the NOAA-NMFS Laboratory, Narragansett, RI. The fishing gear consisted of spin tackle with 12-pound test line; #12 circle hooks to minimize damage to the fish. Previously frozen mackerel strips used for bait. Individual sharks were fought for a specific time period in order to gather information over a wide range of times. After a specified fight time had been reached, the shark was brought on board. Each shark was measured for size (fork length, FL, a straight line measurement from the snout to the caudal fork, and total length, TL, the snout to tail tip, to the nearest cm), weighed (to the nearest kg using a hanging scale), and the sex was determined. Blood was removed (3-4 ml) by direct caudal puncture, a standard National Marine Fisheries Service (NMFS) blue rototag was attached to the first dorsal fin, tag information was recorded as quickly as possible, and

the shark was released. For each fish captured and released, the following data were also recorded: date, location of capture, tag number, time out of the water (in minutes), the time (in minutes) until the blood was tested, release condition, sea surface temperature (°C), air temperature (°C), and salinity (ppt). The release condition was determined from a scale utilized by (Pratt, Personal Communication 1998) and biologists from the NMFS Cooperative Shark Tagging Program during their fieldwork. The scale categorizes the behavior of the animal upon release ranging from one to five. One indicates that the shark is able to quickly swim away; two indicates that the fish slowly swims away without equilibrium loss; three, the fish swims slowly with equilibrium loss; four, the fish is alive but shows erratic movements such as sinking or floating; and finally number five, where the fish does not move and appears dead.

Tank Recovery Study

Shark Capture

The second phase of the study was implemented using large outdoor tanks owned by the National Aquarium in Baltimore located at the edge of the Broadkill River, a few hundred meters from Roosevelt Inlet off Delaware Bay, Lewes, DE. These tanks enabled the recovery process after exhaustive exercise to be studied. Neonate and juvenile sharks were captured at similar sites as the field study (38.8° N 75.1° W) using longline gear. The longline gear consisted of a mainline of 1000ft 5mm braided nylon line, 25-50 gangions with #12 circle hooks that was soaked for thirty minutes using frozen mackerel strips as the bait. The gear was set and tended from the NOAA vessel Stillwell. Once a shark was retrieved on the line during haul back, it was brought

quickly on board, measured for size (as previously mentioned), the sex was determined, an individual colored tag was attached and the shark was placed in a non-aerated transport tank. The sharks were transported to shore (1-5 minutes) and placed in one of two circular holding tanks. Each tank measured 20 ft in diameter and contained approximately 27-m³ water. The tanks utilized open systems with filtered water drawn from Broadkill River inlet, Lewes, DE; each tank held a depth between 3-4ft water, were at ambient temperature and natural photoperiod. A mix of size classes and sexes were placed in each tank, and the sharks were allowed to acclimate for 2-3 days without food before the experiment began.

Experimental Design

Neonate and juvenile sharks were assigned into different experimental groups based on their size. Each of these groups was divided into two categories of control and stressed fish. The stressed sharks were subjected to recovery analysis at four time periods after angling. The corresponding controls, while not angled, were also tested at the same time periods for comparison to pre-exercise levels.

In the 1999 season, blood samples were taken at 0, 3, 6 and 24-hour intervals after the stressor was applied. The 2000 field season repeated 0 and 3 hours, but replaced the other two times with 1.5 and 10 hours or 1.5 and 14 hours for neonates and juveniles, respectively. The repetition of the time periods was to duplicate previous measurements and the addition of new time periods was to obtain a more complete picture of the recovery phase between 0 and 24 hours.

All sharks to be stressed were caught one at a time from the tanks using two hand nets in order to minimize capture time. They were then carried about 100 feet to the angling site (Broadkill River inlet), a circle hook was placed through their jaw, and they were angled for ten minutes. Once the angling period was over, the angled sharks were taken from the water, the circle hook was removed and the sharks were immediately returned to the holding tanks; with the exception of the zero time period sharks that were blood sampled, tagged with a standard NMFS rototag and released.

All sharks returned to the holding tanks were recaptured after the designated time had elapsed and blood was removed through direct caudal puncture. A standard NMFS blue rototag was attached, and the shark was released into Broadkill River inlet. As a stressed fish was sampled and released, a corresponding control shark was captured from the tank, blood sampled, tagged and released. To avoid sampling complications because of the neonatal sharks' small size, they were sampled only once. The juvenile sharks were each serially sampled four times. The sampling process for each juvenile shark (control or stressed) involved angling (if a stress shark), removal of blood, recovery time in the holding tank, and at each subsequent sampling period, recapture, re-sampling and placement in holding tank. After the fourth and final sample was removed (14 or 24 hours depending on year), the sharks were tagged and released.

Blood Sampling

The sharks were bled in both the field and tank recovery study through caudal puncture using a 20-gauge needle with 3-4 milliliters (ml) of whole blood was removed for each sample. Of the total volume removed, 1 ml was analyzed immediately for pH

and blood gases (HCO_3^- , pCO_2 and pO_2) using a portable blood gas analyzer, made by Diametrics Medical (Phillips Medical Systems, Andover, MA). The analyzer uses pre-calibrated cartridges and whole blood to determine pH and the blood gases mentioned above. The temperature must be constant at 37°C . A two-point calibration occurs when the cartridge is inserted into the machine; the first calibration is hard-wired into the cartridge and works with a specific code while the second is a temperature and chemical calibration once inserted into the machine. The pCO_2 and pH use a gel calibrant while the pO_2 works using barometric pressure sensors. If the cartridge is not functioning properly, the IRMA will reject the sample and a new cartridge and blood sample must be used.

The packed cell volume (hematocrit) was determined through replicate samples centrifuged at $12600\times g$ for 3 minutes in heparinized microhematocrit capillary tubes and then read against a HCT card. The hematocrit is measured to determine the percentage of blood as cells versus serum, a change in hematocrit indicates that red blood cells are being actively added or removed from the body. Lactate samples were prepared through deproteinization of two 0.5 ml samples of the whole blood with 1.0ml 8% perchloric acid (HClO_4) and spun for 10 minutes at $13600\times g$. The supernatant was removed and frozen in liquid nitrogen for later analysis using a Sigma kit # 826B and following methods per Bergmeyer (1983). The remainder of the blood was dispensed into two serum separation tubes, allowed to clot, and spun at $1500\times g$ for five minutes. The blood serum was sent to the IDEXX Veterinary Diagnostic Laboratory (N. Grafton, MA) for commercial analysis of 29 variables that included standard veterinary tests for liver and kidney function. These tests include the following constituents: glucose, bicarbonate

(tCO₂), creatine kinase, electrolytes (Na⁺, K⁺, Cl⁻, Ca²⁺, P), enzymes (LDH, CK, AP, GGT, ALP), and total proteins. The IDEXX lab checks and if necessary recalibrates three times a day. Each sample was run against mammalian and if possible fish quality control (QC) standards that followed standard operating procedures (SOP) for the determination of those constituents.

Statistical Analysis

Field Study

All of the statistical analyses were conducted in SPSS version 10 (SPSS Inc. Chicago, IL). The blood parameters obtained during the field study were analyzed by regression (linear, exponential, logarithmic, power, and quadratic) using various independent variables as estimators of physiological disturbance. Those independent variables include fight time (min), size (weight in kg; FL and TL in cm), salinity (ppt), air and sea surface temperature (°C), time out of water (min) and time until blood sampled (min). A principle component analysis (PCA) of the field data was also conducted to examine correlations between the variables. The 29 variables from the serum profile conducted at the IDEXX Veterinary Services Lab were screened for outliers and checked for completeness using the Mahalanobis distance (SPSS Base 10, 1999). Based on detection limits, five variables (bilirubin (total, direct and indirect), creatinine, and LDH) were excluded from the rod and reel analysis because their levels were below detection. In addition to the serum variables from the IDEXX lab, the data collected in the field from the IRMA and other methods, the blood gas, hematocrit and lactate values were also included in the PCA analysis. A Varimax rotation was applied

to determine which variables comprised each component and correlation values of less than 0.3 were excluded. Each component produced a factor score when the analysis is run; these factor scores were subjected to a one-way analysis of variance (ANOVA) to determine if they were significant at the 95% confidence level ($p < 0.05$).

Tank Recovery Study

As the two size classes were treated independently during the tank recovery experiment, they were also subjected to statistical analysis individually. Both sets of data were subjected to descriptive analyses and tests for normality, Kolmogorov-Smirnov and Shapiro-Wilk; neither data set needed transformation. A PCA analysis was run on the neonate tank data with the blood gas variables (pH, pCO₂, HCO₃⁻, pO₂), hematocrit, blood lactate, and the 29 variables from the IDEXX lab were included in this analysis. The PCA included only the 1999 dataset; the serum for the 2000 sampling season was exposed to ambient temperatures and was no longer viable. The significant groups from the PCA were subjected to a one-way analysis of variance (ANOVA) to determine their significance. The blood parameters were also individually subjected to a two-way ANOVA. Some of the variables that were found to be significant through the PCA (excepting electrolytes, enzymes and tCO₂) were obtained from the blood gas analyzer and were available for both the 1999 and 2000 field seasons. An independent samples t-test was conducted for the difference between the two years. To compare between stressed and unstressed animals the means of each time period sampled for the two groups (control and stress) were calculated together with the 95% confidence intervals and significance at $p < 0.05$ and plotted.

For the juvenile tank recovery data, a repeated-measures test (two-way ANOVA) was conducted because the sharks had been serially sampled. The repeated measures-split plot design examines the “within-subjects test” by testing the effects of each time interval (0 and 3 hours) versus the effects of each group (control and stress); while the “between-subject test” shows the relationship between the two groups. The experimental design in 1999 used different individuals for the initial controls (0 hours) then the repeated measures controls (3, 6, 24 hours). Therefore, for the purpose of the repeated measures analysis, the 1999 control data was excluded while the 2000 control series was tested against the stress data for both years. Before this assumption was made, a t-test was employed to test the difference between the years for each variable at each time period. Since the serum samples for the 2000 season were lost, the repeated measures only includes information on the blood gases and hematocrit and lactate that were collected in both years. Graphs of each blood parameter over time for both groups were constructed for the juvenile tank recovery data; the graphs presented are means with 95% confidence intervals and significance at $p < 0.05$.

Results

Field Study

The field study was conducted during July 1999 and July 2000. The sharks captured during this phase of the study were caught at sampling sites just off shore from Broadkill Beach in Delaware Bay. The sharks were caught using rod and reel gear and blood sampled. Twenty neonates with sizes ranging from 56-62 cm TL, and 1.2-1.6 kg weight and four juveniles with sizes ranging from 74-143 cm TL and 2.8-20 kg weight were caught during the two year field study. The fight times ranged from 10 seconds to 20 minutes and 12 seconds. Two sharks were eliminated from the statistical analysis (9.1 and 18.9 minutes), as they had blood data values that were out of range for this study. These values were considered abnormal due to non-study related stressors; one shark exhibited fresh teeth marks on the anterior portion of its body and the other was trapped in the mesh of an old gillnet. These two sharks along with other outlying data will be discussed later. The capture of three sharks in less than 20 seconds enabled baseline sampling of sandbar sharks in the field (Table 1). The sharks usually fought for the first 3-5 minutes and then became weaker; when the line was let out, they would run and tire easily again. Once a shark was boated, the blood sampling and tagging procedure took less than two minutes minimizing additional capture stress to the animals. Means and SE were determined for all of the blood data obtained during the field study (Table 2).

Of all the regression analyses conducted, the linear model produced the most significant results (highest R^2 value) for each variable tested. All 34 dependent variables (IDEXX serum data and whole blood data from the IRMA) were tested against the independent variables listed in the methodology for significance. The following seven

dependent variables were significantly related to an increase in fight time: pH, tCO₂, pCO₂, lactate, blood glucose, calcium and uric acid: implicating its role as an indicator of changes in metabolic activity (Figure 1, Table 3). The pH and tCO₂ (HCO₃⁻, mEq/L milli-equivilants per liter or mM, millimoles per liter) were both negatively correlated with increasing fight time ($R^2 = 0.775$, $p = 0.000$ and $R^2 = 0.568$, $p = 0.000$ respectively). PCO₂ (mmHg) and lactate (mM) were the most positively related to fight time with 76% and 48% of the variability in PCO₂ and lactate concentration, respectively, being explained by the variability in fight time. ($R^2 = 0.764$, $p = 0.000$ and $R^2 = 0.482$, $p = 0.001$ respectively). Blood glucose (mg/dl) ($R^2 = 0.286$, $p = 0.022$), calcium (mEq/L) ($R^2 = 0.327$, $p = 0.013$) and uric acid ($R^2 = 0.308$, $p = 0.017$) were also positively related to fight time although the relationships were not as statistically strong.

Of the remaining independent variables, regression analysis against the same 34 dependent variables indicated significance for only four dependent variables that were related to size (weight, FL, and TL). The dependent variables positively correlated with weight were albumin (g/dl) ($R^2 = .654$, $p = 0.000$; Table 3), albumin to globulin ratio ($R^2 = .564$, $p = 0.000$; Table 3), and ALT (U/L) ($R^2 = .479$, $p = 0.001$; Table 3) (Figure 2). The same dependent variables showed significance for FL and TL (albumin (g/dl), globulin (g/dl), A:G ratio and alanine amino transferase (ALT) (U/L)) (Table 3; Figures 3 and 4). The remaining independent variables were found to have no significance related to the rod and reel data.

An independent-sample t-test was conducted on the dependent variables to test for differences between the two sexes and between different release conditions. Between the sexes, only phosphorus showed any significance ($p = 0.009$), with the males

(n = 9) having a greater concentration than the females (n = 10) (6.478mg/dl and 5.54mg/dl respectively). Between release conditions one and two, a significant difference was observed for the mean pH values with release condition one having a slightly higher pH (pH = 7.30, n=14) than release condition two (pH = 7.12, n=5). The release condition was not recorded in 2000. One of the 20 sharks in the 1999 field study was excluded from statistical analysis as it was considered to be an outlier.

A principle component analysis (PCA) was conducted on the serum data obtained in the field (Table 4). The rod and reel field data indicated 8 important components that accounted for a total of 87% of the total variation in fight time. All 8 components were tested, but only component 2 showed significance ($p = 0.029$). Component 2 was comprised of the following 7 variables: pH, $p\text{CO}_2$, $t\text{CO}_2$, lactate, glucose, Ca^{2+} and uric acid, and encompasses the same variables that show significance with increasing fight time in the regression analysis (Figure 1).

Tank Recovery Study

A total of 80 sharks were caught and held for the tank recovery study. The actual experiments used 48 neonatal sharks and 14 juveniles. Some of the other sharks that were collected were used to assess the possible effects of chronic tank stress with and without the addition of angling stress. The descriptive statistics for the blood chemistry data for the neonate and juvenile sharks used during the tank recovery experiments were determined (Table 5 and 6, respectively).

The two size classes were treated independently for statistical purposes; however, a comparison was run between the control groups and stress groups for both size classes.

Independent sample t-tests indicated a significant difference between the neonate and juvenile stress groups for lactate and glucose levels (Table 7). The magnitude of the disturbance for both variables was greater for the stressed juveniles. Independent sample t-tests for the neonate and juvenile indicated a significant difference between the control size classes for HCO_3^- and pO_2 (Table 7). No significant differences were observed with temperature or salinity for either the tank or the inlet where the angling took place. Also, no sex differences were found with the neonatal sharks or the juvenile sharks for the tank recovery study.

The neonate and juvenile groups were subjected to independent statistical tests based on sampling regime, a PCA for the neonate groups and a repeated measures test for the juvenile groups. These results will be described first for each size class. The remaining results for both groups including acid-base balance, metabolites and enzymes will be presented together systematically for direct comparison.

Neonate

A PCA was conducted using the 1999 serum data obtained from the IDEXX laboratory. A total of 10 components accounted for 90% of the variation, a Varimax rotation was performed and the rotated component matrix was tabulated for visual use (Table 8). A one-way ANOVA was conducted to test the significance of the factor scores for three categories: time, group and their interaction (time and group) (Table 8). Time was recorded as the four stages of recovery ($t = 0, 3, 6, 24$ hours), while group referred to the differences between the control and the experimental (stressed) samples. From the ANOVA, three components were significant. Component 1 comprised the

following variables: pH, pCO₂, tCO₂, lactate, K (mEq/L), CK (U/L), AST (U/L) and the ratio of Na/K and was significant for group (p = 0.016), time (p = 0.000) and group and time (p = 0.000) differences. Component 4 showed significance for differences between the two groups (p = 0.005) and was comprised of the following variables: glucose, HCO₃⁻, phosphorus, Ca²⁺, albumin, A: G ratio, gamma glutamyl transferase (GGT) and magnesium (mEq/L). Finally, component 9 showed significance for the interaction between group and time (p = 0.018) and was comprised of alkaline phosphatase (U/L), amylase (U/L) and ALT (U/L).

The serum variables were individually subjected to a two-way ANOVA to test if there were differences between group, time or group and time (Table 9). Five variables were found to be significant for either group or group and time interaction: phosphorus (mg/dl), alkaline phosphatase (U/L), amylase (U/L), magnesium (mEq/L), and AGAP). These five components were then tested for significance over time for each group, only alkaline phosphatase, gamma-glutamyl transferase (GGT), and magnesium showed any significance and individual graphs were constructed for these three variables. Independent sample t-tests were run for the acid-base chemistry, and, electrolytes and enzymes (Table 10 and 11 respectively). Because of the inclusion of two years of data for the acid-base chemistry, the time periods 0 and 3 hours both have six samples for each group. The other time periods (1.5, 6, 10 and 24 hours) only have a sample size of three for each group (Table 10). The electrolyte and enzyme data has a sample size of three for all the time periods as this data was only collected in 1999 (Table 11).

Juvenile

The repeated measures test was used for the metabolites obtained from the portable blood gas analyzer; pH, lactate, $p\text{CO}_2$, HCO_3^- , $p\text{O}_2$, and hematocrit and included data from 1999 and 2000 (Table 12). The pH was significant for the time, group, and time and group effect ($p = 0.024, 0.005, 0.002$), lactate was significant between the two times, 0 and 3 hours ($p = 0.003$) and $p\text{CO}_2$ between the two effects (time and group, $p = 0.026$). There was no significant relationship between the two groups for HCO_3^- ($p = 0.064$), $p\text{O}_2$ or hematocrit.

Each serum parameter (1999) was tested for significance over time using an independent samples T-Test for the two groups (control and stress) (Table 13). All significant parameters were plotted with means and 95% confidence intervals over the recovery time periods. Independent sample t-tests were run for the acid-base chemistry, and, electrolytes and enzymes (Table 14 and 15, respectively).

Acid-Base Balance

The results of the acid-base significance over time for the neonates and juveniles are presented in tables 10 and 14, respectively. The neonate sharks experienced a sharp decline in the pH of the stressed group after the 10-minute exercise period (Figure 5). The mean pH of the control group was 7.415, while the stress group was 7.101 with a significant difference between these two groups ($p = 0.000$). The drop of 0.3 pH points indicates a doubling of the H^+ ion concentration in the blood. Within 1.5 hours, the stress group had recovered to within 0.04 pH units of the control group. Both groups then display the same pattern after 1.5 hours and have overlapping confidence intervals.

The pH for the juvenile sharks follows a similar pattern over time as the neonates (Table 14). There was an immediate significant decline in the pH levels from 7.447 (control) to 7.157 (stress) which was a drop of 0.29 pH units ($p = 0.000$). This decline again indicates a doubling in the H^+ ion concentration in the blood (Figure 6). At 1.5 hours the values from tested sharks have converged with the control values (7.336 and 7.347 respectively). At 1.5 hours through 24 hours, there was no significant difference between the two groups, and the stress levels have converged with the control values in the range of 7.45.

There was a significant increase in the mean value of pCO_2 for the neonate stress group at the first time period (0 hours) (Table 10, Figure 5). The mean of the control group was 9.433 mmHg while the stressed group rose to 16.42 mmHg with a 74% increase ($p = 0.000$). Within 1.5 hours the levels of each group were not significantly different from each other, and had overlapping means after 3 hours. The levels of pCO_2 in the blood dropped off from the initial time period for both groups and remained consistent until 10 hours ($p = 0.049$). The group means re-merged and remained similar through 24 hours.

There was a significant sharp increase (84%) from baseline, in the value of pCO_2 for the stressed juveniles at 0 hours ($p = 0.033$), followed by a subsequent decline over time (Figure 6). Even though all the pCO_2 values were slightly variable, both groups were consistent with each other and decreased over time and by 24 hours, the values have returned to pre-stress levels. It appears that the baseline values for pCO_2 fall in the range of 7 – 11 mmHg for both groups. Only the 1.5 hour juvenile control sample was out of this range and was slightly elevated at 12.63 mmHg.

There was an immediate slight decrease in the concentration of HCO_3^- (mM) at the first time period for the stress group as expected from the results of the field study (Figure 5). The value for the control group was 5.93 mM while the stress group fell to 5.05 mM. This difference was significant ($p = 0.023$) even though there was an overlap in the confidence intervals. The mean values for the two groups were not considered significantly different after the initial sampling (Table 10).

There was an initial 13% decrease in the juvenile values for HCO_3^- in the stress levels ($p = 0.035$) (Figure 6). The stress group remained below the control group over the duration of the study; however, there were no significant differences between the two groups for the remaining time periods.

Although $p\text{O}_2$ is not normally considered an important indicator in acid-base status as the results can be highly variable depending on the blood sampled, there was a significant difference ($p = 0.014$) between the neonate control and stress groups at 0 hours (Figure 5). The $p\text{O}_2$ (mmHg) levels declined immediately but recovered to control levels by 1.5 hours. The means for each group and the confidence intervals followed a similar pattern over the next 24 hours and were not considered significantly different at any other time period (Table 10).

The juvenile $p\text{O}_2$ data was variable and no significant differences were recorded for any time periods except 24 hours ($p = 0.032$) (figure 6). At 1.5 hours, the $p\text{O}_2$ concentration rose by 140% above baseline and steadily declined until 6 hours when the values between groups became consistent.

Blood Lactate, Packed Cell Volume and Blood Glucose

The packed cell volume (hematocrit) increased in the neonate stress group from the first time period until 3 hours, at which point the value for the stressed sharks fell below the control values (Table 10, Figure 7). There was a significant difference between the two groups at 0 hours ($p = 0.003$); the mean of the stressed group was elevated 17% over the control group. The levels were consistent until the 24 hours sampling when the stress group was 15% lower than the control group ($p = 0.008$).

Juvenile hematocrit levels increased immediately ($p = 0.020$) by 19% above baseline levels after stressing (Table 14). The levels converged by 1.5 hours and the two groups remained consistent through 24 hours (Figure 8). The mean level of hematocrit for both groups was exactly the same at 24 hours, unlike the neonate data.

Neonate blood lactate (mM) showed an immediate rise in concentration at 0 hours followed by an elevated trend until 3 hours (5.02 - 7.09 mM) (Figure 7). The lactate concentration decreased through 6 hours until the stressed and control groups were consistent at 10 hours. A significant difference was noted at 0 and 3 hours ($p = 0.003$ and 0.008 respectively). The control sharks did not deviate from the baseline level of 1.0 mM except the 1.5 hour sample (range = 0.783-0.969, Table 10).

Immediately following the stressing event, there was a significant rise in juvenile blood lactate concentration ($p = 0.010$) (Figure 8). The rise in lactate continues from the initial time period through 3 hours, after which the level begins to drop. There was again a significant difference between control and stress values at 3 hours ($p = 0.010$). At 14 and 24 hours the mean values for both groups were almost identical and within range of the pre-stress conditions (Table 14).

Glucose results were only obtained for the 1999-sampling season so only four time periods were included (0, 3, 6 and 24 hours). Immediately after stress, there was no increase in the concentration of neonate blood glucose (Figure 7). The levels then rose dramatically between sampling. At 3 hours, the concentration of glucose for the stress group was 55% higher than that of the control group ($p = 0.032$). This pattern was consistent through 6 hours (58% increase), but not considered statistically significant at 6 hours ($p = 0.08$). By the final sampling time of 24 hours, the blood glucose levels had returned to match the control values (Table 10).

Immediately after stress there was a slight increase in the concentration of juvenile blood glucose (Figure 8). The levels then rose dramatically between samplings, and at 3 hours the concentration of glucose for the stress group was 65% higher than that of the control group ($p = 0.032$). This pattern was consistent through 6 hours with an 88% increase ($p = 0.016$) at that time. By the final sampling point, the blood glucose levels had fallen to within the pre-stress range and were not statistically significantly different (Table 14).

Serum electrolytes

All the neonatal and juvenile serum variables (only 1999) were checked for outliers and were tested for significant differences over recovery time (Table 11 and 15, respectively). For the juvenile data, two control data points were eliminated in each analysis because of insufficient sample size (Table 15). The time periods of 0 and 24 hours for the control group had a sample size of two data points, while the 3 and 6-hour samples and all of the stress samples had a sample size of three data points.

The concentrations of sodium and chloride ions for the neonate sharks were not significantly different during activity or recovery; the levels approximated 250 mEq/L (Table 11 and Figure 9). Concentrations of serum sodium for the juvenile sharks rose significantly during the ten-minute stress period ($p = 0.032$, Figure 10). The level continued to rise through 3 hours and then by 6 hours the levels of this electrolyte showed full recovery as they returned to baseline values. The 24 hour data points show variation between the means that was due to a lack of three data points for this time period as previously mentioned. The levels of serum chloride (mEq/L) did not differ significantly with stress and remained in the range of 250 – 300 mEq/L over the course of the 24 hours (Figure 10).

Potassium ions (mEq/L) for the neonates rose slightly (15%) after the 10-minute stress period followed by steady decline back to the control values by 3 hours. The values between the two groups remained close after 3 hours, and the mean values were identical at 24 hours (Figure 9).

Levels of juvenile potassium (mEq/L) were not statistically significant over time; however, there was variation within the two groups. There was an immediate 22% rise in the stress group that continued to a 42% increase at 3 hours. The concentration began to drop after 3 hours and was fully recovered by 24 hours (Figure 10).

The concentration of serum calcium (mg/dl) for neonate sharks showed a significant increase from baseline values at 0 hours ($p = 0.012$). The overall means of the stress group remained elevated through 3 hours but by 6 hours had dropped to the same level as the control group and stayed consistent with baseline levels (12-14meq/L) through 24 hours (Figure 9).

The juvenile serum calcium (mg/dl) was significantly higher for the stressed group after the 10-minute stressing event ($p = 0.001$), and again at 3 hours ($p = 0.009$) (Figure 10). At 6 hours the stress level had dropped below the control group and was not statistically different. The values from tested sharks were again higher than the control at 24 hours but no significant difference was observed.

The neonatal serum magnesium (mEq/L) showed a similar pattern to the neonatal calcium for the first few hours (Figure 9), but unlike calcium, magnesium remained elevated throughout the entire 24-hour recovery analysis. At 0 hours, there was a significant variation between the two groups ($p = 0.05$). Baseline values for magnesium appear to be around 2.0 mEq/L.

The concentration of magnesium (mEq/L) for the juvenile sharks between the two groups was not different (Figure 10). The stress group increased 24% over control values at 3 hours. The values from tested sharks continued to rise through 6 hours but showed almost full recovery by 24 hours.

Enzymes and Metabolites

The concentration of creatine kinase (CK) in the neonate blood serum did not show any statistical significance (Table 11), however; there was an immediate 143% increase in the stressed samples over control levels after the 10-minute stressing event (Figure 11). After the immediate rise, there was a decline back to control values by 3 hours and the levels remained consistent through 24 hours.

The juvenile CK concentration rose in the stressed sharks, from <20 U/L to >150 U/L, after the 10-minute stressing event ($p = 0.045$) (Table 15). The levels of the stress

group had dropped back to baseline levels by 3 hours and then remained consistent with the control values (Figure 12).

There were also significant changes in the enzyme GGT in the neonate sharks, but not for the juvenile sharks. There was an immediate significant decrease in the concentration for the stress levels ($p = 0.013$) (Figure 11). The levels increased after 0 hours and both groups were not considered different after the initial sample.

The concentration of alkaline phosphatase (AP) for the neonatal sharks was not elevated initially but at 3 hours a maximal level of 200% over resting values was reached (Figure 11). The levels started to decline slightly, a significant difference was observed at 6 hours ($p = 0.041$). At 24 hours the average value of the stressed group had returned to the control levels (10 U/L).

The juvenile alkaline phosphatase, AP, concentration doubled in concentration after the stressing event ($p = 0.028$). The 3-hour values from treated sharks then dropped with a concurrent increase in the control values ($p = 0.042$). Both groups were not significantly different from 6 through 24 hours (Figure 12).

There was no difference observed in the phosphorus for the neonates, however, there was a difference for the juveniles. The amount of phosphorus (mg/dl) for the juvenile sharks increased 42% after the stressing event ($p = 0.043$). The phosphorus data followed a similar pattern as the juvenile AP and was significantly different at 3 hours ($p = 0.022$). The two groups converged to below pre-stress values by 24 hours (Figure 12).

Creatinine (mg/dl) also did not differ for the neonates but did for the juveniles. Creatinine followed a similar pattern with an increase in the stress group immediately following stress, and a subsequent decrease in values from tested sharks and rise in

control values (Figure 12). At 3 hours, the control values were significantly higher than the stress group and the pre-stress conditions ($p = 0.008$). From 6 hours through 24 hours, the two groups, although not converged, followed the same pattern.

Recovery and Post-Release Survivorship

Recovery from exhaustive exercise varies dramatically in different species. Recovery times for standard exercise physiology variables were compared for teleosts and elasmobranchs in different species after exhaustive exercise (Table 16). Of the 104 sharks caught and released during the 1999-2000 seasons, 9 were recaptured (Table 17), which represents a recapture rate of 8.6%. Time at liberty ranged from 1.45 hours – 367 days, the distance traveled ranged from 0.01 – 219 miles in various directions. Sharks from both years and both phases of the project were recaptured. During 2000 we recaptured SB9906, a 1999-rod and reel sample, and then used this shark in the 2000 tank recovery study. The recapture information shows that this shark traveled only 8 miles SouthEast (SE) of its original capture site. However, this information might be misleading because this shark probably over-wintered off of North Carolina and then returned to Delaware Bay one full year later. The shark, SB0043, that was captured 92 days or 3 months later (October 2000), was found off of Cape Hatteras, N.C., one of the common wintering grounds for juvenile sandbar sharks.

Discussion

Field Study

It is well known that angling induces physiological stress (Wood et al. 1983) as pH and $t\text{CO}_2$ (HCO_3^-) decrease with continued exercise, $p\text{CO}_2$ increases. The differences in the levels of $p\text{CO}_2$ and HCO_3^- , from baseline values indicate respiratory and metabolic acidosis. In this study seven important variables: pH, $t\text{CO}_2$, $p\text{CO}_2$, lactate, glucose, Ca^{2+} and uric acid were found to be significant. These same variables comprise the second group of the principle component analysis, which was found to be significant versus fight time. The majority of these variables are related to each other and specifically to a change in pH, and are common indicators of stress (Mazeaud et al. 1977; Wilkie et al. 1997). Two variables show a decreasing trend with increasing fight time, pH and $t\text{CO}_2$ or HCO_3^- . The pH decreases dramatically from 7.45 to 7.05, indicating more than a doubling of the hydrogen ion (H^+) concentration in the blood. The use of glycogen and formation of lactic acid produces lactate and H^+ , which enter the blood stream from the white muscle. This increase contributes to intracellular acidosis (Tietz 1976). The $t\text{CO}_2$ also decreased with continued angling, which resulted in metabolic acidosis. The increase of acid in the blood causes the HCO_3^- to be used in the buffering system, this HCO_3^- is not adequately replaced and so the serum concentration falls; less H^+ ions are buffered and pH also falls (Abelow 1998; Figure 1).

In this study the other five variables that are significant show an opposing trend with increasing stress: they increased over time (Figure 1). $p\text{CO}_2$ and blood lactate exhibit the most linear trends with fight time. This is expected because $p\text{CO}_2$ rises during hyperactivity due to an increase in the muscle production of CO_2 and an

inhibition of excretion of CO₂ at the gills (Cliff and Thurman 1984). During anaerobic conditions, the white muscle readily produces lactic acid to further glycolytic activity (Fudge 1995; Wardle 1981). The secondary stress effect of an increase in circulating glucose levels during exhaustive exercise is due to the primary stress effect of increasing circulating catecholamines (Smith 1992). Calcium levels are expected to increase during continued stress because ionic balances in cell barriers have been perturbed. In mammals, increasing Ca²⁺ levels with stress are an expected consequence of acidosis (Cliff and Thurman 1984). In this study, uric acid also increases with increasing fight time. Uric acid is a waste product derived from purines in the diet and is regularly excreted and absorbed by the body (Tietz 1976). Uric acid can also be a precursor in the synthesis of urea, although the pathway is not thought to be a major one; urea synthesis and excretion are thought to be acid-base independent (Ballantyne 1997; Shuttleworth 1988). A constant rise in the concentration of uric acid was also found for skipjack tuna (*Katsuwonus pelamis*) for 48 hours after capture (Bourke et al. 1987).

The effect of Independent variables on the stress response

A number of studies have been conducted to determine the effects of body size on changing metabolic parameters (Kieffer et al. 1996). Ferguson et al. (1993) found that changes in acid-base and metabolite status were clearly related to body size in salmonids. In this study, increasing body size did not influence acid-base parameters (Figures 2-4). The proteins albumin, globulin, A: G ratio and the enzyme alanine amino transferase (ALT), however, did show differences with size related variables. Albumin is one of the most important non-bicarbonate buffers in the body and the increase in

concentration with increasing size could be based on greater acidosis and an increased need for additional buffering capacity (Abelow 1998). Increased levels of ALT may indicate a disruption in gill and cardiac tissues, which might occur if the larger fish experienced a greater level of stress (Wells et al. 1986). Any conclusions drawn from the rod and reel data concerning size variables are at best preliminary. The small juvenile sample ($n = 3$) versus neonates ($n = 19$) could play a role in the differences observed. Of the three sharks that drive the relationships with size, two of them had the same TL, 78 cm and Weight, 3.3 kg. None of these sharks are considered outliers on the fight time regressions and none were recaptured. These three juvenile sharks might not be indicative of true concentrations in larger sharks because only a small sample size was observed.

A difference between blood parameters in the sexes has been found for newborn smooth dogfish (*Mustelus canis*). Barham and Schwartz (1992) found differences in lactate, glucose and hematocrit levels between the sexes. No differences in those three parameters were found in this any phase of this study, however, there was a difference between the sexes for phosphorus in the field study. The male serum levels were higher than the females although there was no other significant difference between the sexes and the number of males and females used. There is no reference to a discrepancy between sexes for phosphorus in the literature. The reason for the difference remains unclear.

The use of release condition might be an appropriate indicator of how survivorship could be impacted after release. With just one scale change in release condition (from a one to a two), the pH decreased from 7.30 to 7.12, which indicates a

58% increase in the H^+ concentration. As mentioned in the results, one outlying data point was withheld from the release condition analysis. A sample size of one would not have produced valid results from any statistical analysis. This sample was only used for comparative purposes. The neonatal sandbar shark in question was fought for 6 minutes, had a comparable time out of water as the other sharks in the study, however, it had a release condition of a 4. This condition indicates that the shark is alive, but shows erratic movements, sinks or floats. The shark in question swam away weakly at the surface then rolled over onto it's back and sank next to the boat. The pH recorded for this shark was 7.162, which is not significantly different from the release condition 2's mean value. The potassium, however, was 6.0 mM, which indicates that the fish was experiencing hyperkalemia, an increase in blood potassium. Values above 7.0 mM of potassium cause bradycardia, and can be fatal to fish (Cooper and Morris 1998). Due to the lack of differences in the handling, it is reasonable to propose that the decrease in pH and increased level of potassium could be due to the fresh teeth marks on the anterior portion of the fish.

Release condition has been used to assess stress in three species of shark during capture by gillnets (Manire et al. 2001). The scale used in the gillnet study is also based on five parameters and each level is closely related to the release conditions used in this study. Manire et al. (2001) found both inter- and intraspecific differences in blood serological parameters; they concluded that species-specific and individual differences from gillnet capture are closely related to the degree of struggling and the animals respiratory physiology.

No other independent variables, such as temperature (1999: 26.2, 26.6 and 27°C, 2000: 23, 22.2, and 21.8°C mean values in the bay, tanks and river respectively for both years), salinity (1999: 25-30.2 ppt, 2000: 17-28.4 ppt) time out of water (less than 2 minutes) or others listed in the methods, had a significant effect on rod and reel samples. The salinity was not considered statistically significant for the blood chemistry but was different for the two years due to high quantities of rain (freshwater) during the 2000 sampling effort. An explanation for the lack of significance with these independent variables is that all of the samples were caught in the same area and processed efficiently thereby minimizing any experimental differences.

Although one rod and reel shark captured in 2000 was excluded from analysis because of an independent environmental variable it was not one analyzed for in this study. The shark in question was fought for 18.9 minutes, was a female juvenile (124cm FL, 143cm TL), and was experiencing a stress reaction due to a long-term stressor. This shark probably had swum into a gillnet, become partially gilled, but somehow managed to free herself and part of the net. The gillnet was around the body and had cut into the pectoral fins about 1-2 inches on each side. Even though this shark experienced the second longest fight time, the acid-base chemistry was lower than expected. The values for this shark is as follows; pH, 7.295; pCO₂, 13.2 mmHg; HCO₃⁻ 6.3 mM; lactate, 1.61 mM; and hematocrit, 27. All of these values are within the range of control fish or those stressed for 10 minutes; which is unusual because of the length of the fight time. These values could have been produced one of two ways, the effect of this chronic stressor had “seasoned” this shark to physiological changes or the shark did not actively fight and so did not experience lactacidosis. It is known that if fish are subjected to the same

stressor, subsequent exposures will not have as great of a magnitude of physiological disturbance (Moyle and Cech Jr 1996; Barton 1997). For example, the first time a fish is manually chased around a tank there will be an appreciable rise in lactic acid, but not on the second day even if subjected to the same level of stress.

Tank Recovery Study

The utilization of holding tanks allowed the process of metabolic recovery after exercise to be explored. The neonatal sharks provide a snapshot of the physiological changes at each time period, while the juvenile sharks provided an opportunity to directly assess the changes in each shark as recovery progressed. The trend during and after exercise has been shown to be the same for all fish species studied to date. It has been found that pH, HCO_3^- , ATP, creatine phosphate and glycogen decrease during stress, while pCO_2 , lactate, hematocrit, glucose, creatine kinase (CK) and electrolytes (Na^+ , K^+ , Cl^- , Ca^{2+} , Mg^{2+}) all increase during or after exercise (Cliff and Thurman 1984; Kieffer et al. 1994; Moyes and West 1995; Cooper and Morris 1998).

The sharks included in the control analysis showed significance between the size classes for HCO_3^- at 1.5 hours; the neonatal sharks experienced a decrease from resting values while the juvenile sharks experienced a concomitant increase above resting values. A similar pattern was followed for the control sharks at 6 hours with pCO_2 . The decrease in neonate bicarbonate concentration at 1.5 hours is due to an extended capture time for one of the three samples tested. The elevation in pCO_2 for the control juvenile samples at 6 hours is also due to an extended and therefore more stressful capture event for one of the three juvenile sharks tested. This shark struggled out of the nets twice and

fell against the side of tank possibly hitting its head; it did not survive through the 24 hour sampling time period. Because these differences were caused through experimental error they are not considered as physiologically important as the changes within the stress group.

A significant difference between the sizes was noted at 1.5 and 3 hours after the stressing event for the concentration of lactate. The juvenile values were double the concentration of lactate obtained by the neonatal sharks. The doubling of the lactate concentration for larger fish is a common finding, indicating that the larger fish have an increased anaerobic capacity, which can be defined as the total mass specific production of lactate on exhaustion (Goolish 1991; Ferguson et al. 1993; Kieffer et al. 1996). This could be due to a difference in their metabolic scope: the difference in metabolic rate between rest and maximal exertion. Salmon show a size dependency with larger fish having significantly greater performance capabilities (Schmidt-Nielsen 1984). In contrast to the lactate data, the juvenile sharks had a lower level of circulating glucose at 0 and 3 hours then the neonates but overall the juveniles reached a higher blood glucose concentration. The lower levels of circulating glucose might be an artifact of size, Cliff and Thurman (1984) who worked with comparable sized juveniles did not see the highest level of blood glucose until 24 hours post-exercise.

Although the size classes were treated independently during the study and for statistical analysis, the conclusions drawn from the results will be presented together. The greatest difference between the two groups was the number of samples taken (one from each neonate and four from each juvenile) and the final time during the 2000

season at which they were taken: 10 hours for the neonates and 14 hours for the juveniles.

Acid-Base Balance

The changes found in the acid-base status after exercise for sandbar sharks is similar to those reported in other active elasmobranch and teleost fish (Piiper et al. 1972; Cliff and Thurman 1984; Perry et al. 1985; Tufts et al. 1991). For both the neonate and juvenile sandbar sharks, the pH decreased immediately following stress (Figures 5 and 6). The range for both size classes was from 7.447 (resting values) to 7.101 (after stress). This is a 100% increase in the H^+ concentration in the blood. The values recovered by 1.5 hours and were not significantly different after this point. The lower limit for pH values is 6.8, once a fish falls below this value it will no longer survive (Abelow 1998). With an accompanying decline in HCO_3^- (resting, 6-7 mM to stress, 4 mM) and an average increase of 80% in pCO_2 , mixed acidotic distress is indicated with both metabolic and respiratory components playing a part.

The immediate decrease in the concentration of HCO_3^- (from 6 mM to 5 mM) for both size classes after exercise indicates metabolic acidosis. Again after 1.5 hours, there were no longer significant differences between the groups. Bicarbonate is one of the most important buffering systems in the body; within the pH range of this study HCO_3^- can contribute up to 90% of the buffering capacity (Abelow 1998). Bicarbonate functions as a buffer by accepting H^+ ions to form carbonic acid, considered a weak acid, therefore has less effect on the blood pH. Carbonic acid can disassociate into carbon dioxide and water, the carbon dioxide that results is primarily excreted through the gills,

while bicarbonate reabsorption occurs in the kidney (Moyle and Cech, Jr 1996). As noted by Cliff and Thurman (1984) the resting levels of bicarbonate obtained (7 mM) are remarkably lower than mammalian systems, therefore non-bicarbonate buffers might play a greater role than expected.

The increase in $p\text{CO}_2$ levels during exercise indicates respiratory acidosis (from 9 mmHg to 17 mmHg). An immediate rise in $p\text{CO}_2$ has been known to increase levels of circulating catecholamines and plasma potassium, and change the excretion of acid through the urine (Tietz 1976). The recovery of pH, HCO_3^- and $p\text{CO}_2$ after 1.5 hours indicates rapid recovery from the initial acidosis. An interesting side note is the significant differences observed for the neonates between groups at 10 hours for pH and $p\text{CO}_2$. This difference is not considered to be physiologically significant because the values for the surrounding time periods indicate recovery has already occurred and is consistent. The values presented and trends are similar to the results found by Cliff and Thurman (1984) for the juvenile dusky shark (*Carcharhinus obscurus*).

Blood lactate, Packed Cell Volume and Blood Glucose

Despite the rapid recovery of the pH values, the concentration of lactate in the blood continued to rise until the maximal value was reached at 3 hours (5 mM for neonates and up to 15 mM for juveniles) (Figures 7 and 8). The neonates were recovered by 10 hours and the juveniles recovered by 14 hours. The 1.5-hour sample lacks significance for both size classes because the control values are elevated. The elevation occurred because of an extended capture time, which caused the sharks to experience some level of exercise in the tank while trying to avoid the nets. One of the

most useful parameters to base the rate of recovery of carbohydrate status is lactate. Salmonids and elasmobranchs can be considered lactate releasers as the concentrations of blood lactate increase 10-20 fold after exercise (Milligan and Girard 1993; Moyes and West 1995). Lactate values in the rainbow trout (*Oncorhynchus mykiss*) reached a peak of almost 17.6 mM (Milligan and Wood 1986a; Kieffer et al. 1994) while newborn smooth dogfish reached a maximum concentration of 1.7 mM (Barham and Schwartz 1992). Elasmobranch lactate levels after exhaustive exercise range from 3.9-18 mM (Cliff and Thurman 1984; Manire et al. 2001).

Anaerobic metabolism occurs during exhaustive exercise. The first physiological change is a decrease in the creatine phosphate stores combined with a decrease in muscle ATP. Once this process has started, glycogen is reduced by as much as 90% in the white muscle (Milligan 1996). Lactate levels rise dramatically in conjunction with the decreasing glycogen. As glycolysis occurs, there is a buildup in the NADH concentration, and without reduction of the NADH to NAD^+ glycolytic activity could no longer proceed. The conversion of pyruvate to lactate converts the NADH to NAD^+ , thereby replenishing the NAD^+ pool allowing glycolysis and ATP generation to continue (Fudge 1995).

The initial acidosis is well into recovery before the lactate concentration has reached its maximal value. There have been other reports of the discrepancy between pH and lactate levels in many fish (Cliff and Thurman 1984). The continued increase in the lactate concentration even after the stressor has ended is due to additional leaching out of the muscle. One reason for this discrepancy is the differential release rate of lactate and H^+ from the white muscle into the blood (Wood et al. 1983). This process is

advantageous to the fish because the lactate anion is less toxic than the H^+ and so does not require as great a buffering capacity (Abelow 1998). It is also advantageous because the fish does not have to subjugate its need for aerobic metabolism to favor anaerobic clearance (Fudge 1995).

Despite the mechanism of differential release, most of the lactate and H^+ ions produced together are never released into the blood, but instead metabolized *in-situ* through glyconeogenesis, with little or no interaction with the Cori cycle (Milligan 1996; Milligan and Girard 1993). This process is unlike mammalian systems where the primary fates are through the liver in the Cori cycle or through oxidation in the red muscle (Milligan and Girard 1993). The mechanism of glyconeogenesis within the white muscle is still not fully understood, two key enzymes PEPCK (phosphoenolpyruvate carboxykinase) and pyruvate carboxylase are not detectable in fish muscle except in marlin (*Makaira nigricans*) (Milligan 1996). The current theory put forward by Moyes and West (1995) is that reversal of the pyruvate kinase reaction of 0.05% it's forward direction would be more than sufficient to allow glyconeogenesis to occur directly within the white muscle. Direct enzymatic studies are still necessary in this area to determine the exact pathway of lactate disappearance and glycogen resynthesis.

There are varying references in the literature regarding packed cell volume concentration. Bushnell et al. (1982) state that increases in hemoglobin have been found in teleosts after exercise but not in elasmobranchs. Temporary increases in hematocrit also occur, however, during periods of extreme demand in order to maximize the amount of oxygen getting to the tissues. In fact, hematocrit levels are often used as a measure of

the blood oxygen carrying capacity (Wells et al. 1986). It is known that splenic contraction can dump red blood cells into the blood in order to increase the amount of oxygen reaching the tissues (Jones and Randall 1978). The oxygen carrying capacity of hemoglobin is altered in the presence of high carbon dioxide or low pH; hemoglobin changes shape, reducing its affinity for oxygen and delivering more oxygen to the active tissues (Moyle and Cech, Jr 1996). This process is known as the Bohr effect. It has been questioned whether elasmobranchs have efficient oxygen transport and extraction systems. The whole blood oxygen saturation of some elasmobranchs displays weak cooperativity, a moderate Bohr effect, a high oxygen affinity, a weak or absent Haldane effect and no Root shift (Ballantyne 1997). The lemon shark (*Negaprion brevirostris*) exhibits a fixed acid Bohr effect ($\log P_{50}/pH$) of -0.36 ; which is comparable to the range found for elasmobranchs of -0.25 to -0.49 (Bushnell et al. 1982; Shuttleworth 1988). The mako shark (*Isurus oxyrinchus*) does not exhibit a Bohr effect, which is contrary to data obtained from other large pelagics (Wells and Davie 1985). There is no known Bohr coefficient for the sandbar shark, as they have similar life history strategies and activity, I would assume it falls within the range of other large coastal carcharhinids.

In this study, the hematocrit increased immediately in the stress group for both size classes through 3 hours (15 –28). This rise functions to increase the amount of oxygen getting to the tissues after the anaerobic activity has ended and the fish is experiencing aerobic metabolism. Both groups converged after 3 hours and remained consistent through 24 hours. The juvenile levels for both groups were elevated over the neonates; the juveniles were not as easily captured from the tanks and might have experienced more stress than the smaller sharks. Greater levels of stress would lead to a

larger oxygen debt and a greater oxygen demand in the tissues after the removal of the stressor. As discussed by Ferguson et al. (1993) larger individuals in some species may experience greater physiological disturbances than smaller individuals when subjected to the same stressor. Hematocrit ranges have been found between 6-43 for various sharks (Bushnell et al. 1982; Schwartz and Jensen 1993; Wells and Davie 1985).

The rise in the concentration of blood glucose is considered a secondary stress effect stimulated primarily by the release of catecholamines from the chromaffin tissue (Wells et al. 1986; Wood 1991). A rise in glucose concentration after exercise has been found in many other studies (Cliff and Thurman 1984; Bourke et al. 1987; Lowe and Wells 1996); it is also known that asphyxia and struggling disturbs glucose levels in elasmobranchs (Mazeaud et al. 1977). In this study the blood glucose for both size classes followed a similar pattern. There were no initial changes in concentration after angling, but the sharks then experienced hyperglycemia at 3 hours (up to 10 mM), this continued through 6 hours and baseline levels were reached by 24 hours. The blood contains a small and unknown fraction of the total body glucose pool that it is not the best indicator of changes in blood sugar; it would be better to determine the levels within the white muscle (Schwalme and Mackay 1991). Concentration of insulin and glucagon are also components of hyperglycemia after exhaustive exercise that have not been well studied (Milligan 1996). Exact recovery times of blood glucose differ between studies; in this study just 1999 data was collected and so it can only be said that recovery occurs somewhere between 6 and 24 hours. This data contradicts the findings obtained by Cliff and Thurman, (1984) who observed the highest levels of blood glucose at 24 hours in juvenile dusky sharks. Blood glucose concentrations rise before an increase is seen in

lactate, and in teleosts glucose remains elevated from 12 hours through several days (Schwalme and Mackay 1985; Wells et al. 1986). Blood glucose up to 10 mM has been reported for the marine teleost, the blue mao mao (*Scorpius violaceus*) (Lowe and Wells 1996). Elasmobranch blood glucose ranges from 1.5-20 mM (Murru 1984; Cliff and Thurman 1984; Stoskopf 1993; Barham and Schwartz 1992).

Water and Salt Balance

Due to technical difficulties resulting from a failed sample storage unit, the serum data presented only included the 1999 sampling season, so changes between 0 and 3, and 6 and 24 hours were not evaluated. There was, however, notable data from the 1999 season that bears discussion. Wells et al. (1986) noted rapid changes in electrolyte composition indicating that angling had dramatic effects on the water and salt balance in marine fishes. Wood (1991) observed that exercise stress caused profound ionic and osmotic disturbances, haemoconcentration generally occurs and the concentration of all the major plasma electrolytes increase. The results of the current study are consistent with these observations, as almost all of the electrolytes experienced some level of disturbance. Corticosteroid increase, a primary effect of stress, is known to affect active ion transport across the gills and decrease water uptake that in turn can increase serum ion levels (Bourke et al. 1987). When a strong acid, such as lactic acid, is added to the blood, protons are released that are subsequently buffered by the bicarbonate anion. Despite this physiological change the fish maintains neutrality; in this case the addition of the lactate anion replaces the bicarbonate and the total charge between cations and anions remains the same (Abelow 1998). Either the continued addition of protons or

removal of lactate through active white muscle uptake results in a deficiency of ions which can then be replaced through $\text{HCO}_3^-/\text{Cl}^-$ and H^+/Na^+ branchial exchange systems (Piiper et al. 1972; Shuttleworth 1988; Manire et al. 2001). The major electrolytes will be discussed in relation to this study and comparisons with current literature will also be presented. As the majority of literature focuses on freshwater teleosts, differences in ionic balance with elasmobranchs would be expected as they have different osmoconformatory strategies. In light of this, comparisons will mainly focus on the large pelagic teleosts and limited elasmobranch data.

Serum Electrolytes

Serum concentrations of chloride did not differ significantly with stress, levels remained in the region of 250 mEq/L for both size classes (Figures 9 and 10). Chloride is one of the major anions. A lack of change in chloride concentration in stressed fish indicates that the lactic acidosis occurs in normochloremic conditions; i.e. the level of chloride in the blood remains the same. The anion gap or ion balance is again maintained by an increase in lactate (Abelow 1998). This finding concurs with some researchers (Wydoski et al 1976; Wood et al. 1983; Cliff and Thurman 1984) but contradicts results found by others (Bourke et al. 1987; Cooper and Morris 1998). The various results obtained are either caused by interspecific differences or differing experimental procedures. The overall range for chloride concentration in fish is difficult to assess based on sampling techniques and values reported such as whole blood, plasma or serum levels. Fish subjected to exhaustive exercise have chloride concentrations averaging 140, 156, and 173 mEq/L for rainbow trout, skipjack and yellowfin tuna

(*Thunnus albacares*), respectively (Wood et al. 1983; Wells et al. 1986). Elasmobranch values on the average are higher and range between 202-310 mEq/L for various sharks (Cliff and Thurman 1984; Wells et al. 1986; Stoskopf 1993; Cooper and Morris 1998; Manire et al. 2001).

Sodium is the major cation and is used to maintain osmotic pressure and normal distribution of water within the body (Tietz 1976). The value of sodium ions did not change significantly over the recovery time and remained within the same region as the chloride values for the neonates (250 mEq/L), which is consistent with other studies (Cliff and Thurman 1984). The juvenile sharks, however, did experience a significant rise in sodium concentration to 280 mEq/L immediately after stress that continued through 3 hours and was fully recovered by 6 hours. Again the results from this study both concur and disagree with previous work. Cliff and Thurman (1984) found no changes in sodium concentration after exhaustive exercise while Wood (1991) cites marked electrolyte fluxes and shifts into the intracellular compartment. The total ion flux can be disturbed if there is a large enough rise in the lactate anion, this disturbance can be corrected by an increase in the cation concentration to balance the ionic charge (Abelow 1998). As sodium is the principle cation, concentrations can increase either to maintain electroneutrality or Na^+ can be actively exchanged for H^+ ions to reduce the effects of intercellular acidosis (Shuttleworth 1988). Fish subjected to exhaustive exercise have sodium concentrations averaging 160, 217, and 233 mEq/L for rainbow trout, skipjack and yellowfin tuna, respectively (Wood et al. 1983; Wells et al. 1986). Elasmobranch values range between 250-359 mEq/L for various sharks (Cliff and

Thurman 1984; Wells et al. 1986; Stoskopf 1993; Cooper and Morris 1998; Manire et al. 2001).

As bicarbonate levels in the plasma fall, a gradient is created that causes potassium to shift out of the cells and into the plasma to maintain electroneutrality; this shift will cause the plasma potassium values to rise until neutrality has been established (Abelow 1998). The levels of potassium rose slightly after stress from 3 mEq/L and the values had returned to the baseline or control values for this study by 3 hours for the neonates and 6 hours for the juveniles. The range for the stressed neonatal and juvenile samples did not increase beyond 4 mEq/L and 6 mEq/L, respectively. The fish experienced a mild hyperkalemia while the potassium levels were elevated. The recovery of control levels by 6 hours implies that while immediate normality for these fish might have been impaired, no long-term effects were experienced. The results found in this study are similar to the other studies mentioned. Both teleosts and elasmobranchs experience at least a small rise in potassium ions after exposure to capture stress (Cliff and Thurman 1984; Bourke et al. 1987; Wood 1991).

As previously stated, potassium levels of 7 mEq/L or greater can cause bradycardia, disrupt myocardial function and potentially increase lactic acid production by extending anaerobic conditions (Cliff and Thurman 1984). Increased and continued levels of plasma potassium can reduce the threshold potential required to stimulate muscle cells and can result in constant stimulation which causes tetany, or rigidity of the dorsal musculature (Smith 1992). Two neonate sharks that were caught on the first trip in 1999 died in the tanks overnight. Their blood was sampled in order to investigate the cause of their deaths. The most prominent features of their blood chemistry were the

low pH (6.821 and 6.166) and the elevated levels of potassium (14.6 and 21.1 mEq/L). Fish subjected to the stress associated with capture and exercise have potassium concentrations averaging 4, 15, and 20 mEq/L for rainbow trout, skipjack and yellowfin tuna respectively (Wood et al. 1983; Wells et al. 1986). Elasmobranch values range between 3-9 mEq/L, with the mako shark exhibiting the highest value in all the sharks examined (Cliff and Thurman 1984; Wells et al. 1986; Stoskopf 1993; Cooper and Morris 1998; Manire et al. 2001). The mammalian system differs slightly from fish muscle in their response to acidosis, mammals tend to gain sodium, chloride and water and unlike fish, lose potassium (Wood 1991).

The calcium ion showed a significant increase immediately after stress (around 16 mg/dl or 4 mM) and remained elevated through 3 hours for both size classes. One of the reasons for an increase in electrolytes such as Ca^{2+} and Mg^{2+} is leakage from the cells if the cellular membrane is compromised, or from increasing corticosteroid levels (Bourke et al. 1987). Increases in serum calcium have been found for the dusky shark (Cliff and Thurman 1984) pelagic gamefish (Wells et al. 1986) and for the Northern pike (*Esox lucius*) (Soivio and Oikari 1976) after capture, however, not for the perch (*Perca fluviatilis*) (Haux and Sjobeck 1985). Similar effects as those caused through hyperkalemia can occur in fish with extreme calcium elevation: impairment of muscular contraction and neuromuscular nerve transmission (Cliff and Thurman 1984). As the calcium levels in this study had stabilized and were decreasing within 3 hours, it is proposed that significant muscular damage did not occur within the 10 minute angling event. Yellowfin tuna and wild perch subjected to exhaustive exercise experience calcium concentrations ranging 2-6.4 mM (Haux and Sjobeck 1985; Wells et al. 1986).

Elasmobranch values range between 3-4.8 mM for various sharks (Cliff and Thurman 1984; Wells et al. 1986; Stoskopf 1993; Manire et al. 2001).

Serum magnesium is one of the most important electrolytes and functions as an activating complex for some important enzymatic reactions. The enzyme systems include hexokinase, creatine kinase, alkaline phosphatase and any reaction that involves ATP (Tietz 1976). Although serum magnesium levels followed a similar pattern as the calcium, the serum magnesium levels remained elevated throughout the 24 hours for the stressed neonate sharks, with a slight difference between the groups from 2-2.5 mEq/L. The juveniles did not show any significant differences, but there was a rise in the concentration of the stressed group that peaked at 6-hours at 3.5 mEq/L, but recovery was established by 24 hours. Elevated magnesium levels will have similar effects on the body as elevated levels of calcium (Cliff and Thurman 1984). Soivio and Oikari (1976) found an increase in magnesium in the Northern pike that lasted for 4 hours after handling, while Haux and Sjobeck (1985) found magnesium levels in perch disturbed for 3 days after capture. Magnesium concentrations in elasmobranchs range between 1 and 4 mM (Cliff and Thurman 1984; Stoskopf 1993; Cooper and Morris 1998).

Enzymes and Metabolites

The levels of creatine kinase (CK) increased by 143%, (150 U/L), compared to the resting values for the neonate sharks, although no statistical significance ($p = 0.253$) was observed. The juvenile sharks also experienced a rise in CK levels (700%), within 3 hours the stressed group for both size classes had recovered to below control levels (Figures 11 and 12). Creatine kinase catalyzes the reaction that uses creatine phosphate

to produce ATP. ATP is rapidly used up and when the concentration of ADP increases equilibrium restores the balance by reducing creatine phosphate stores, generating creatine thereby forming the necessary ATP (Moyes and West 1995). The immediate need for energy in the form of ATP is solved through the breakdown of glycogen into lactate. For every mole of glycogen converted to lactate, 3 moles of ATP are formed (Driedzic and Hochachka 1978). Overall stress in fish produces a decrease in ATP and creatine phosphate stores to levels well below those seen in higher vertebrates following exhaustion (Wood 1991). When significant muscular damage occurs, there is a leakage of ions and enzymes into the blood. In this study, the lack of continued CK elevation might indicate a lack of significant muscular damage.

Ranges for the enzyme creatine kinase are from 1000-1426 U/L for skipjack and yellowfin tuna and 380-494 U/L for black (*Makaira indica*) and blue marlin (*Makaira nigricans*) (Wells et al. 1986). Creatine kinase concentrations vary dramatically for elasmobranchs. Cliff and Thurman (1984) found a maximal level of 100 U/L at 6 hours while values from Murru (1984) range between 172-1610 U/L. The high value was found for the brown (or sandbar) shark and is 10 times greater than the maximal level obtained in this study. Different methodology and capture sites were used for Murru's (1984) work but that should not make a substantial difference, no other reference to CK levels in sandbar sharks can be found to validate these numbers. The sandbar shark, from this study, is consistent with the observations obtained by Cliff and Thurman (1984) on the dusky shark; the maximal CK level obtained in the dusky shark are the same as my values.

Serum levels of the enzyme gamma-glutamyl transferase (GGT) decreased immediately after stress for the neonates, but were considered recovered from 3 hours onwards. No differences in GGT were observed for the juvenile sharks. Surplus amino acids can be used as metabolic fuels for the body in the form of fatty acids, ketone bodies and glucose; the enzyme GGT is the most important in the class of enzymes that converts amino acids into keto-acids (Stryer 1975). An increase in GGT can indicate disturbed hepatic function, but decreases are not commonly seen (Tietz 1976). A common reaction catalyzed by GGT is the production of glutamine to glutamate the conversion of which removes an ammonium ion (NH_4^+) the principle component of urea (Abelow 1998). Elasmobranchs are osmoconformers, they use a combination of solutes to maintain their osmolarity close to that of the environment, and urea is the principle solute contributing 40% of the osmolarity (Ballantyne 1997). As previously mentioned, urea concentrations are not considered to be acid-base dependent (Shuttleworth 1988). The only other exercise physiology study that attempted to observe this enzyme, did not report any activity within the plasma of large pelagic gamefish (Wells et al. 1986).

Raised alkaline phosphatase (AP) values are also another indicator of liver malfunction. Commonly AP reacts in the same direction as GGT but not as rapidly (Tietz 1976). This, however, was not seen in this study. The levels of alkaline phosphatase found in this study were highly variable, from 10-30 U/L, the neonates and juveniles followed opposing patterns through 6 hours; by 24 hours both stress and control samples for both size classes were the same. Raised serum levels of AP have been noted for fish that appear to be experiencing stress (Wells et al. 1986). However, Bourke et al. (1987) did not find any significant changes in AP levels for skipjack tuna.

The range of alkaline phosphatase is from 62-100 U/L for skipjack and yellowfin tuna (Wells et al. 1986). Elasmobranchs fall in the range of 2.3-28.2 U/L (Murru 1984; Manire et al. 2001). The use of the AP levels obtained in this study to explain post-exercise physiology would be speculative.

The juvenile sharks also showed significant changes with phosphorus and creatinine. The phosphorus increased immediately after exercise, the stress values then fell below the baseline levels and steadily declined through 24 hours. The control levels were elevated between 3 and 6 hours; these particular juveniles experienced the roughest capture from the tanks which might explain the difference between the control and stress levels. The pattern for phosphorus is similar to the pattern for alkaline phosphatase in juvenile sharks. Phosphorus is usually present in the body in the bones or in the cells as inorganic phosphate. It is involved in the intermediary metabolism of carbohydrates and is a component of physiologically important compounds such as nucleic acids (DNA) and nucleotides (ATP) (Tietz 1976). Inorganic phosphorus along with proteins such as albumin account for intracellular buffering in the white muscle of fish and changes in circulating levels of phosphorus could be due to leakage from damaged cells (Manire 2001). Phosphorus ranges from 3.2-21.7 mg/dl have been found for various sharks (Murru 1984; Stoskopf 1993).

Creatinine levels increased immediately following stress, the stress values then fell below the control levels for the remainder of the analysis. Creatinine is the resultant product from the spontaneous breakdown of creatine and creatine kinase (Tietz 1976). Increased creatinine can be indicative of decreased glomerular filtration rate (Wells et al. 1986). However renal failure did not appear to occur in this study. Creatinine values

average 0.03 and 0.02 mM for skipjack and yellowfin tuna respectively (Wells et al. 1986); and elasmobranchs fall between the range of 0-0.9 mM (Murru 1984; Stoskopf 1993; Manire 2001).

Environmental variables and stress response

Many environmental factors have been examined and almost all can play a role in evoking the stress response. Discrepancies between studies may be related to changes in variables such as temperature, salinity and water quality to name a few (Barton 1997). When trying to compare data it is important to consider the effects of these independent variables and to attempt to standardize the work. Two well-known environmental variables that can detrimentally affect the metabolic status of fish in conjunction with exercise are temperature and air exposure. Warmer water has lower dissolved oxygen content and so the fish can experience hypoxia while still in the water; removal of the fish out of the water obviously poses a threat as they are immediately subjected to hypoxic conditions.

Early studies with freshwater fish found significant variations in delayed mortality results ranging between zero and forty percent. In regard to this discrepancy, further studies were undertaken to determine the cause of these inter and intraspecific differences. Wilkie et al. (1996) concluded that post-angling mortality is temperature dependent and suggested that anglers minimize playing time and air exposure during the midsummer months. Ferguson and Tufts (1992) subjected rainbow trout to various levels of exercise and air exposure. Their results indicate that the brief amount of air exposure associated with non-consumptive angling could add a significant amount of

stress to the fish and inhibit survival. Kieffer et al. (1994) found that the lactacidosis experienced in rainbow trout was much larger in fish acclimated to 18°C than 5°C. They also found, however, that recovery rates did not significantly vary for either temperature. They concluded that while acclimation temperature does not play a part in recovery time it could function to explain the discrepancies in the literature of the lactacidosis dynamics after exhaustive exercise.

As previously mentioned, there were no significant differences in temperature or salinity for either the capture sites or the holding tanks so these two variables did not play a substantial role in explaining the physiological changes due to catch and release angling in this study. Air exposure can detrimentally affect the stress response and potential recovery of fish (Ferguson and Tufts 1992). It was impossible to avoid air exposure during either phase of the study; however, the length of time the sharks were out of the water was minimized to facilitate eventual recovery. The sandbar sharks used in both phases of the study were exposed to less than two minutes of direct air exposure. Two minutes can have a dramatic effect on the acid-base status of these fish, however blood was removed usually within 30 seconds so the physiological consequences were not as pronounced. As all the sharks utilized had similar times out of water and the experimental protocol was the same for all samples removed, air exposure might have elevated the levels of lactate beyond their “exercise-stress” only values but the same addition was held throughout and so the experimental error is reduced. The baseline sharks were sampled with the same experimental protocol and lactate values of 1.0 mM were found. This is considered the minimal level for fish so this shows the added air exposure did not significantly alter the blood chemistry data.

Additional stressors can include the site (cardiac, dorsal, caudal areas), or type (cannulation or puncture) of blood removal (Wood et al. 1983). The use of caudal puncture, although previously disagreed upon, was the most effective method of obtaining rapid and reliable samples from each shark in this study. Cooper and Morris (1998) in fact have stated that the use of caudal puncture is an effective method of obtaining baseline samples from undisturbed fish. Another stressor that needs to be considered is the effect of confinement in holding tanks for 2-5 days without food. Food was not given to the sharks for two reasons. It is very difficult to make sharks start eating in captivity without the addition of already feeding captive sharks (Wardle 1981). As each shark captured is physiologically different, short-term starvation ensured the samples would be more consistent. Haux and Sjobeck (1985) pointed out that a short period of fasting is commonly used in mammals to stabilize the biochemical and physiological parameters before experimentation began. The control sharks in the tank study had similar values to the baseline levels obtained during the field study. This indicates that chronic tank stress was not a factor in the results, in fact, table one uses both field and tank recovery samples to provide a list of baseline blood chemistry data for the reader.

A number of sharks were caught and left in the tanks during the duration of the study to assess the potential effects of chronic stress. Two sharks were kept during the two years for 92 and 95 hours and both had blood chemistry values well within the range of the baseline sharks. The neonate kept for 95 hours in 1999 also had serum values within range of the baseline sharks and had a normal pH value of 7.426 upon release.

Post-Release Survivorship and possible Implications for Management

This study was one of the first to obtain baseline levels for sandbar sharks in the wild (Table 1). The findings indicate that physiological recovery from metabolic disturbances after exhaustive exercise occurs rapidly. The comparison to the dusky shark indicates that two similar species from the same family can have markedly different post-exercise physiological disturbances (Cliff and Thurman 1984). The results from the tank study indicate that recovery easily occurs during the 24-hour post-exercise confinement (Table 16). The greatest implication from a rapid recovery time is the increased chance for survival as the sharks attain full recovery within a matter of hours, and can continue their normal behavior such as feeding, evading predators and eventually reproducing.

Once recovery time was established, the more interesting question, do these sharks survive once released into the wild either after angling or confinement, was answered. The fate of all sharks released over the two years will never be known, but in light of the recapture data that was received by the Apex Predators Program, Narragansett, RI, we know that a percentage can and do survive the stress of recreational capture and release. A recapture rate of at least 8.6% was reported from this study (see Table 17). Previous work in DE Bay with gillnets and longline gear has found a recapture rate of 6.4% for neonate and juvenile sandbar sharks (Merson and Pratt 2001).

Four sharks from the rod and reel study were recaptured, only one of these with a fight time of 10.22 minutes was a graphical outlier for lactate, glucose and uric acid (Figure 1). The pH, blood gas and calcium levels were all normal. This shark had a comparable time out of water and sampling regime; however, when it was released it

wallowed near the boat for 5-6 minutes (release = 2). This shark was on the smaller end of the size ranges and possibly experienced anaerobic activity for a longer period. Two recaptured sharks that bear special notation were at either end of the time-at-large data for the recaptures. One of the 10-minute stressed neonates that was released immediately after angling in the inlet (pH = 7.154) was recaptured one hour and forty five minutes later attempting to take a bait from one of the anglers on the beach. Indeed, pH recovery had occurred by 1.5 hours, although immediate physiological disturbances resulted, the shark was recovered enough to continue its normal feeding behavior within two hours. Another recapture, with a time at large of 367 days, was a neonate shark used in the 1999 rod and reel field study with a fight time of 56 seconds. During the second year, as a juvenile, it was used as a control shark and was released with a pH of 7.413. This shark was not only subjected to the stress associated with normal rod and reel angling, but also experienced the additional blood sampling and muscle puncture and was still able to fully recover, over-winter and return to Delaware bay after one full year. Together this data coupled with the other recapture information indicates that post-release survivorship is not compromised in neonatal and juvenile sandbar sharks after release from exhaustive exercise.

It is important to understand the post-release survivorship for this species and other sharks because mortality after release is an important concern for fisheries managers. In order to protect and promote stock viability and abundance, managers must implement programs regarding minimum sizes, quotas, bag limits and catch and release fishing. Fishery managers must take into account both facets of the fishery, commercial and recreational capture, coupled with removal and possible release of fish.

The addition of the prohibited species group within the FMP for highly migratory species is an attempt to curb further depletion and promote revival of these stocks (FMP 2000). The K selected life history strategies for large coastal and pelagic sharks as discussed by Branstetter (1990) leave these sharks highly susceptible to overfishing and make them challenging to manage effectively. As the sandbar shark, one of the most commercially important sharks, shares similar behaviors, environments, and life history strategies with other sharks on the prohibited species list it was important to determine if they were capable of handling the stress associated with catch and release angling. Now that the physiological effects of stress on sandbar sharks have been determined and their post-release survivorship is known to be high, fisheries managers can use this information when determining policy. The results of this study will hopefully aid fisheries managers in effectively regulating the sandbar shark, and other large coastal carcharhinids.

Conclusions

The study quantified the physiological changes that occur in sandbar sharks during exhaustive exercise and also followed the sharks through their metabolic recovery. Independent variables were examined for significance, and fight time and size contributed important results. The results support the hypothesis that there is a direct relationship between the length of capture and the physiological response. Sharks were not fought for longer than 20 minutes, but any juvenile sandbar shark that is caught recreationally will usually be brought in before 20 minutes (Skomal, Personal Communication 2000). The use of 10 minutes for the fight time in the recovery study

was long enough to exhaust both the neonate and juvenile sharks. The results from the field study and tank recovery study were consistent. The values obtained during the field study for sharks fought for 10 minutes closely match those from the recovery study, as do the baseline field and control tank samples. These findings indicate that the angling method employed during the recovery study duplicated the stressful event in the field and the captive fish did not experience significant chronic stress.

Unlike the results obtained by Cliff and Thurman (1984) the sandbar shark requires significantly less time than 24 hours to reach full metabolic recovery. In fact, recovery occurs within 1.5 hours “post catch” in both neonates and juveniles. This finding is significant, most fish are not able to recover from exhaustive exercise this rapidly. Tufts et al. (1991) and Booth et al. (1995) have reported a recovery time of 12-24 hours in salmonids. A list of recovery times for blood chemistry parameters for various species are listed in table 16. Further work is required with the sandbar to examine the metabolic changes that are occurring not only in the blood, but also in the white muscle during exercise. The results of this study have helped show how two similar sharks, the sandbar and dusky, experience physiological disturbances and recovery differently.

Table 1. Baseline serological parameters for the sandbar shark (*Carcharhinus plumbeus*). Baseline values discussed here include two rod and reel samples that were fought for 18 seconds, three control neonates and two control juveniles from the tank study. Tank study fish were captured using longline gear, acclimated to holding tanks for two days, sampled and released. The values are from the current study and that of Stoskopf (1993).

	This Study (1999)				Stoskopf (1993)		Population
	N	Minimum	Maximum	Mean	N	Mean	
Hematocrit	7	52	77	17.5	20	19.8	Captive
Glucose (mg/dl)	7	14	20	65.6	33	63	Wild
pH	7	7.314	7.522	7.417			
Lactate (mM)	7	0.91	2.71	1.3			
pCO ₂ (mmHg)	7	6.1	13	9.4			
HCO ₃ (mM)	7	5	6.5	6.0			
BUN (mg/dl)	7	609	978	837	34	848	Wild
Creatinine (mg/dl)	7	0	0	0.0	33	0.45	Wild
Phosphorus (mg/dl)	7	3.7	6	4.9	30	5.4	Wild
Calcium (mg/dl)	7	1.6	13.8	11.8	33	13.6	Wild
Total Protein (g/dl)	7	0.5	3.3	1.9	33	2.2	Wild
Albumin (g/dl)	7	0.4	1.1	0.7	33	0.5	Wild
Globulin (g/dl)	7	0.5	2.5	1.3	33	1.7	Wild
Ratio A:G	7	0.3	0.6	0.5			
Sodium (meq/l)	7	165	273	235	27	284	Wild
Chloride (meq/l)	7	147	275	223	25	236	Wild
Potassium (meq/l)	7	2.5	3.6	3.1	32	3.1	Wild
tCO ₂ (meq/l)	7	0	7	3.7			
AGAP	7	0	24	13.0			
Tbilirubin (mg/dl)	7	0	0.11	0.0			
Dbilirubin (mg/dl)	7	0	0.02	0.0			
Ibilirubin (mg/dl)	7	0	0.1	0.0			
Alkaline Phosphatase (U/L)	7	11	22	14.1			
GGT (U/L)	7	0	10	3.9			
ALT (U/L)	7	1	21	6.0	37	19	Wild
AST (U/L)	7	1	12	5.0	35	22	Wild
LDH (U/L)	7	0	3	0.4	28	100	Wild
CK (U/L)	7	0	56	29.7			
Cholesterol (mg/dl)	7	25	79	43.4	32	54	Wild
Triglycerides (mg/dl)	7	29	73	43.0	9	36	Wild
AMY (U/L)	7	0	3	0.9			
Magnesium (meq/l)	7	1.9	2.4	2.2			
Uric acid (mg/dl)	7	0	0.1	0.0	33	0.45	Wild
NA/K	7	53	99	75.1			

Table 2. Blood chemistry and size data for the field phase of the study. This data includes both 1999 and 2000 samples. The field study was conducted during 1999-2000 on juvenile sandbar sharks (*Carcharhinus plumbeus*). Sharks were captured using rod and reel gear in Delaware Bay, DE.

	N	Minimum	Maximum	Mean	SEM
Weight (kg)	19	1.2	3.3	1.7	0.2
FL (cm)	22	49.0	68.0	53.3	1.2
TL (cm)	22	56.0	78.0	61.5	1.4
Hematocrit	22	15.0	22.5	19.5	0.4
Glucose (mM)	19	55.0	94.0	71.2	2.2
pH	22	7.06	7.43	7.27	0.02
Lactate (mM)	22	1.1	4.0	2.1	0.2
pCO ₂ (mmHg)	22	8.6	22.3	13.6	0.7
HCO ₃ (mM)	22	5.1	6.7	6.0	0.1
BUN (mg/dl)	18	70.0	957	825	51.8
Creatinine (mg/dl)	18	0.0	0.1	0.0	0.0
Phosphorus (mg/dl)	18	4.4	7.8	6.0	0.2
Calcium (mg/dl)	18	13.0	14.9	14.0	0.1
Total Protein (g/dl)	18	1.5	2.7	1.8	0.1
Albumin (g/dl)	18	0.5	1.4	0.6	0.0
Globulin (g/dl)	18	0.3	2.0	1.2	0.1
Ratio A:G	18	0.3	4.7	0.7	0.2
Sodium (meq/l)	18	165	288	250	8.0
Chloride (meq/l)	18	147	280	232	7.9
Potassium (meq/l)	18	2.1	4.5	3.4	0.1
tCO ₂ (meq/l)	18	4.0	7.0	5.1	0.2
AGAP	18	0.0	19.0	13.7	1.2
Tbilirubin (mg/dl)	18	0.0	0.1	0.0	0.0
Dbilirubin (mg/dl)	18	0.0	0.0	0.0	0.0
Ibilirubin (mg/dl)	15	0.0	0.1	0.0	0.0
Alkaline Phosphatase (U/L)	18	7.0	18.0	12.2	0.7
GGT (U/L)	18	0.0	6.0	2.3	0.4
ALT (U/L)	18	0.0	24.0	2.9	1.3
AST (U/L)	18	0.0	26.0	3.8	1.6
LDH (U/L)	4	0.0	2.0	0.5	0.5
CK (U/L)	18	3.0	35.0	15.8	1.7
Cholesterol (mg/dl)	18	26.0	62.0	45.2	2.4
Triglycerides (mg/dl)	18	21.0	73.0	45.8	3.2
AMY (U/L)	18	0.0	4.0	0.7	0.3
Magnesium (meq/l)	18	2.1	2.7	2.4	0.0
Uric acid (mg/dl)	18	0.0	0.3	0.1	0.0
NA/K	18	53.0	90.0	75.0	2.3

Table 3. Significant linear regression analysis versus independent variables for the field study data. The field study was conducted in Delaware bay during 1999-2000 on juvenile sandbar sharks (*Carcharhinus plumbeus*). Only significant results are presented with $p < 0.05$ a, (N = 20).

	Fight Time (min)	Weight (kg)	Fork Length (cm)	Total Length (cm)
pH	$R^2=.755$ **			
pCO ₂ (mmHg)	$R^2=.764$ **			
pO ₂ (mmHg)				
HCO ₃ ⁻ (mM)				
Lactate (mM)	$R^2=.482$ **			
hematocrit				
glucose (mg/dl)	$R^2=.286$ *			
BUN (mg/dl)				
Creatinine (mg/dl)				
Phosphorus (mg/dl)				
Total Protein (g/dl)				
Albumin (g/dl)		$R^2=.654$ **	$R^2=.991$ **	$R^2=.988$ **
Globulin (g/dl)			$R^2=.980$ **	$R^2=.980$ **
Ratio A:G		$R^2=.564$ **	$R^2=.995$ **	$R^2=.997$ **
Calcium (mg/dl)	$R^2=.327$ *			
Chloride (meq/l)	$R^2=.211$ *			
Sodium (meq/l)				
Potassium (meq/l)				
tCO ₂ (meq/l)	$R^2 = 0.568$ **			
AGAP				
Alk Phos (U/L)				
GGT (U/L)				
ALT (U/L)		$R^2=.479$ **	$R^2=.944$ *	$R^2=.949$ *
AST (U/L)				
LDH (U/L)				
CK (U/L)				
Cholesterol (mg/dl)				
Triglycerides (mg/dl)				
AMY (U/L)				
Magnesium (meq/l)				
Uric acid (mg/dl)	$R^2=.308$ *			
NA/K				

a * $p < 0.05$, ** $p < 0.01$

Table 4. A principle component analysis (PCA) with a varimax rotation for the field study data. The field study was conducted in Delaware bay during 1999-2000 on the sandbar shark. Significance is indicated by (*) with $p < 0.05$.

	1	2 *	3	4	5	6	7	8
ALT U/L	0.971							
RATIO	0.958							
AST U/L	0.935							
albumin g/dl	0.916							
globulin g/dl	-0.695						0.516	
pH		-0.965						
PCo ₂ (mmHg)		0.893						
CO ₂ meq/l		-0.762				0.326	0.305	
Lactate (mM)		0.727				0.363		
URIC mg/dl		0.707	-0.401			0.366		
calcium mg/dl	-0.4	0.642	0.316				0.398	
BUN mg/dl			0.931					
AGAP			0.877					
CK U/L	-0.393		-0.66					
chloride meq/l				0.977				
sodium meq/l				0.947				
Potassium meq/l				0.659	-0.596			
ALP U/l	0.475			-0.549				0.528
NA/K					0.847			
AMY U/L					0.788			
HCO ₃ (mM)					-0.698			
TRIG mg/dl						0.939		
GGT IU/L	0.488				0.432	-0.511	0.319	
CHOL mg/dl	0.377				0.452	0.457		0.35
MAG mEq/L							0.848	
Tprotein g/dl			0.406				0.574	0.37
glucose (mg/dl)	-0.352	0.396				0.428	-0.51	
HCT	0.318							0.773
phosphorus mg/dl	-0.493							0.611

* Rotation converged in 11 iterations.

(*) Component Significant ($p = 0.029$)

Eigenvalue	5.605	4.334	3.118	2.93	2.914	2.331	2.137	1.813
% of Variance	19.329	14.944	10.753	10.102	10.049	8.038	7.368	6.253
Cumulative %	19.329	34.273	45.026	55.128	65.177	73.215	80.583	86.836

Table 5. Descriptive blood chemistry data for the neonate tank recovery study. Data was collected in July of 1999 in Delaware Bay, DE. Both groups are presented for comparison.

	Control					Stress				
	N	inimu	Maximu	Mean	SEM	N	inimu	aximu	Mean	SEM
Hematocrit	12	11	18	15.9	1.2	12	13	21	16	1.9
Glucose	12	58.0	89.0	72.2	2.9	12	46.0	148.0	90.0	8.9
pH	12	7.4	7.5	7.5	0.0	12	7.0	7.6	7.4	0.1
Lactate (mM)	12	0.6	1.2	0.9	0.1	12	0.4	7.1	2.9	0.6
pCO ₂ (mmHg)	12	6.5	9.7	8.1	0.3	12	5.9	19.3	9.7	1.3
HCO ₃ (mM)	12	4.9	6.8	5.7	0.1	12	4.3	5.6	5.0	0.1
BUN (mg/dl)	12	286	999	815	74.7	12	750	1017	916	26.4
Creatinine (mg/dl)	12	0.0	0.0	0.0	0.0	12	0.0	2.0	0.2	0.2
Phosphorus (mg/dl)	12	3.4	5.7	4.3	0.2	12	3.3	8.6	5.6	0.4
Calcium (mg/dl)	12	1.6	15.8	12.5	1.0	12	12.6	16.6	14.4	0.4
Total Protein (g/dl)	12	0.5	3.3	1.8	0.2	12	1.5	2.2	1.8	0.1
Albumin (g/dl)	12	0.5	1.1	0.7	0.1	12	0.4	0.7	0.5	0.0
Globulin (g/dl)	12	0.5	2.5	1.2	0.1	12	1.0	1.6	1.3	0.0
Ratio A:G	12	0.3	0.9	0.5	0.0	12	0.3	0.5	0.4	0.0
Sodium (meq/l)	12	186	273	254	7.1	12	216	312	266	7.6
Chloride (meq/l)	12	165	252	233	7.2	12	192	279	246	7.4
Potassium (meq/l)	12	2.1	3.9	3.1	0.1	12	3.0	3.9	3.4	0.1
tCO ₂ (meq/l)	12	0.0	6.0	3.3	0.7	12	0.0	5.0	3.2	0.6
AGAP	12	9.0	24.0	17.4	1.3	12	10.0	32.0	19.4	1.7
Tbilirubin (mg/dl)	12	0.0	0.1	0.0	0.0	12	0.0	0.1	0.0	0.0
Dbilirubin (mg/dl)	12	0.0	0.0	0.0	0.0	12	0.0	0.0	0.0	0.0
Ibilirubin (mg/dl)	12	0.0	0.1	0.0	0.0	12	0.0	0.1	0.0	0.0
Alk Phos (U/L)	12	6.0	22.0	12.1	1.3	12	9.0	24.0	14.8	1.5
GGT (U/L)	12	1.0	6.0	2.9	0.4	12	1.0	4.0	2.3	0.3
ALT (U/L)	12	0.0	21.0	3.6	1.7	12	0.0	15.0	3.8	1.3
AST (U/L)	12	0.0	12.0	2.8	1.0	12	0.0	35.0	4.4	2.8
LDH (U/L)	12	0.0	5.0	0.8	0.5	12	0.0	1.0	0.1	0.1
CK (U/L)	12	3.0	66.0	25.8	6.4	12	7.0	241.0	44.0	19.2
Cholesterol (mg/dl)	12	25.0	69.0	45.3	4.0	12	21.0	83.0	46.6	5.1
Triglycerides (mg/dl)	12	33.0	85.0	56.3	4.5	12	28.0	91.0	56.9	5.4
AMY (U/L)	12	0.0	3.0	0.8	0.3	12	0.0	2.0	0.7	0.2
Magnesium (meq/l)	12	1.8	2.4	2.0	0.1	12	2.0	2.9	2.3	0.1
Uric acid (mg/dl)	12	0.0	0.1	0.0	0.0	12	0.0	0.3	0.1	0.0
NA/K	12	62.0	129	83.1	4.7	12	55.0	98.0	78.9	3.3

Table 6. Descriptive blood chemistry data for the juvenile tank recovery study. Data was collected in July of 1999 in Delaware Bay, DE. Both groups are presented for comparison.

	Control					Stress				
	N	Minimu	Maximum	Mean	SEM	N	Minimum	Maximum	Mean	SEM
Hematocrit	10	14	25	20.9	1.73	12	9	25	20.7	1.87
Glucose (mM)	10	31	91	60.6	5.16	12	54	175	94.25	11.85
pH	10	7.331	7.535	7.462	0.02	12	7.137	7.529	7.39	0.04
Lactate (mM)	10	0.653	7.57	2.157	0.67	12	0.61	12.86	5.639	1.34
pCO ₂ (mmHg)	10	5.8	11.3	7.67	0.50	12	5	17.5	8.317	1.11
HCO ₃ (mM)	10	4.9	6	5.33	0.11	12	3.4	5.9	4.65	0.19
BUN (mg/dl)	10	689	1014	808	39.64	12	677	1005	857	39.96
Creatinine (mg/dl)	10	0	0.2	0.05	0.03	12	0	0.5	0.2	0.05
Phosphorus (mg/dl)	10	0.5	8	4.67	0.62	12	2	21.4	8.03	1.50
Calcium (mg/dl)	10	0.4	15	12.79	1.39	12	1.8	16.6	14.02	1.20
Total Protein (g/dl)	10	0.8	3.5	2.56	0.25	12	2.4	3.5	3.058	0.09
Albumin (g/dl)	10	0.3	0.8	0.64	0.06	12	0.6	0.8	0.7	0.02
Globulin (g/dl)	10	0.5	2.7	1.92	0.20	12	1.6	2.8	2.358	0.09
Ratio A:G	10	0.3	0.6	0.35	0.03	12	0.2	0.5	0.308	0.02
Sodium (meq/l)	6	231	273	255.2	6.11	10	249	297	273	4.23
Chloride (meq/l)	6	204	275	248.7	10.37	10	228	301	263	7.55
Potassium (meq/l)	6	2.5	3.3	2.9	0.14	10	2.6	5.4	3.68	0.26
tCO ₂ (meq/l)	10	3	8	4.8	0.49	12	2	6	3.85	0.36
AGAP	3	8	23	13.67	4.70	6	12	31	23.83	3.14
Tbilirubin (mg/dl)	10	0	0.11	0.048	0.01	12	0.01	0.09	0.063	0.01
Dbilirubin (mg/dl)	10	0	0.01	0.007	0.00	12	0	0.02	0.0067	0.00
Ibilirubin (mg/dl)	10	0	0.1	0.041	0.01	12	0	0.08	0.055	0.01
Alkaline Phos (U/L)	10	0	28	16.3	2.70	12	0	38	26.67	2.87
GGT (U/L)	10	1	12	6.4	1.31	12	0	12	7	0.12
ALT (U/L)	10	0	29	6.8	2.94	12	1	6	3.5	0.44
AST (U/L)	10	0	27	7.4	3.09	12	3	29	10.5	2.02
LDH (U/L)	10	0	2	0.2	0.20	12	0	4	0.33	0.33
CK (U/L)	10	0	48	15.3	5.00	12	5	703	121.2	56.90
Cholesterol (mg/dl)	10	6	79	58.2	6.44	12	31	82	65.08	3.57
Triglycerides (mg/dl)	10	2	55	33.4	4.62	12	18	50	35.08	3.29
AMY (U/L)	10	0	3	0.5	0.31	12	0	2	0.667	0.23
Magnesium (meq/l)	10	0	3.2	2.03	0.32	12	0	4	2.84	0.29
Uric acid (mg/dl)	10	0	0.2	0.04	0.02	12	0	0.3	0.062	0.03
NA/K	6	80	99	88.5	2.88	10	55	102	76.8	4.48

Table 7. Independent Sample T-Test on the variation between sandbar shark size classes for the field study. Data were collected in Delaware bay during 1999-2000. Significant values are presented with $p < 0.05^a$

Levene's Test for Equality of Variances		T-Test for Equality of Means			
		F	Sig.	t	Sig. (2-tailed) ($p < 0.050$)
Control Data:					
HCO ₃ T=1.5	Equal variances assumed	0.0645161	0.812018	-3.3588	0.028
PCO ₂ T=6	Equal variances assumed	9.3677419	0.054964	10.0559	0.002
Stress Data:					
Lactate T=1.5	Equal variances assumed	1.488157	0.289507	-3.0582	0.038
Lactate T=3	Equal variances assumed	5.522907	0.040635	-3.2212	0.009
	Equal variances not assumed			-3.2212	0.013
PO ₂ T=24	Equal variances assumed	1.6004605	0.274518	-2.795	0.049
Glucose T=0.17	Equal variances assumed	1	0.373901	7.12039	0.002
Glucose T=3	Equal variances assumed	4.4125705	0.103582	3.38327	0.028

^a Only Significant values are presented.

Table 8. Principle component analysis with varimax rotation for the tank recovery study data. The recovery study was conducted in Delaware bay on sandbar sharks during the field seasons of 1999-2000. Significance is indicated by (*) with $p < 0.05$.

	1 *	2	3	4 *	5	6	7	8	9	10
HCT		0.434	0.372			-0.532				
glucose (mg/dl)			0.696	-0.348						
pH	-0.934									
Lactate (mM)	0.526		0.57		0.304					
PCO ₂ (mmHg)	0.959									
HCO ₃ (mM)				0.430						0.696
BUN mg/dl		-0.599			0.559					
Creatinine mg/dl			0.92							
phosphorus mg/dl			0.411	-0.460				0.378		
calcium mg/dl		0.516		-0.366			0.468	0.346		
Tprotein g/dl		0.954								
albumin g/dl				0.653				-0.319	0.334	
globulin g/dl		0.954								
RATIO		-0.357		0.730						0.308
sodium meq/L					0.898					
chloride meq/L					0.939					
potassium meq/L	0.570	-0.642			0.394					
CO ₂ meq/L	-0.340							-0.883		
AGAP								0.784		
TBILI mg/dl						0.935				
DBILI mg/dl			0.609					0.541		
IBILI mg/dl						0.934				
ALP U/l			0.327						0.754	
GGT IU/L				0.64		0.374	0.345		0.358	
ALT U/L	0.453								0.772	
AST U/L	0.759				-0.37				0.372	
LDH U/L										0.826
CK U/L	0.884									
CHOL mg/dl							0.931			
TRIG mg/dl							0.926			
AMY U/L						0.315			0.56	
MAG mEq/L				0.454						
URIC mg/dl			0.857	-0.767						
NA/K	-0.526	0.715								

a Rotation converged in 21 iterations.

(*) Component Significant ($p < 0.05$)

Total Eigenvalue	4.888	3.947	3.495	3.077	2.979	2.871	2.728	2.576	2.128	1.805
% of Variance	14.377	11.609	10.281	9.051	8.763	8.443	8.023	7.576	6.258	5.308
Cumulative %	14.377	25.986	36.267	45.318	54.081	62.524	70.547	78.123	84.381	89.689

Table 9. Two-Way Analysis of Variance for the 1999 neonate serum data and 2000 blood chemistry data for the tank recovery study. Data were collected in Delaware bay during 1999-2000. Significant results are indicated with $p < 0.05$ ($N = 24$).

	df	F	Sig. ($P < 0.05$)		df	F	Sig. ($P < 0.05$)		df	F	Sig. ($P < 0.05$)
Glucose mg/dl				BUN mg/dl				TBILI mg/dl			
GROUP	1	1.652	0.289	GROUP	1	0.708	0.462	GROUP	1	1.788	0.273
TIME	3	1.174	0.449	TIME	3	0.543	0.686	TIME	3	0.645	0.636
GROUP*TIME	3	4.570	0.017	GROUP*TIME	3	3.168	0.053	GROUP*TIME	3	0.705	0.563
pH				Creatinine mg/dl				GGT IU/L			
GROUP	1	0.952	0.401	GROUP	1	1.434	0.317	GROUP	1	1.846	0.267
TIME	3	1.875	0.309	TIME	3	1.000	0.500	TIME	3	2.192	0.268
GROUP*TIME	3	19.06	0.000	GROUP*TIME	3	0.922	0.452	GROUP*TIME	3	1.195	0.343
Lactate mM				CA mg/dl				ALT U/L			
GROUP	1	6.734	0.081	GROUP	1	2.023	0.250	GROUP	1	0.011	0.923
TIME	3	1.178	0.448	TIME	3	0.469	0.725	TIME	3	3.472	0.167
GROUP*TIME	3	2.164	0.132	GROUP*TIME	3	1.568	0.236	GROUP*TIME	3	0.609	0.619
PCO₂				Tprotein g/dl				AST U/L			
GROUP	1	0.704	0.463	GROUP	1	0.019	0.900	GROUP	1	0.866	0.421
TIME	3	1.948	0.299	TIME	3	0.623	0.646	TIME	3	6.122	0.085
GROUP*TIME	3	13.06	0.000	GROUP*TIME	3	1.684	0.210	GROUP*TIME	3	0.342	0.795
HCO₃⁻				ALB g/dl				LDH U/L			
GROUP	1	23.09	0.170	GROUP	1	4.765	0.117	GROUP	1	4.119	0.135
TIME	3	1.503	0.373	TIME	3	1.000	0.500	TIME	3	0.593	0.661
GROUP*TIME	3	0.395	0.758	GROUP*TIME	3	1.659	0.216	GROUP*TIME	3	0.532	0.667
PHOS mg/dl				GLOB g/dl				CK U/L			
GROUP	1	15.82	0.028	GROUP	1	0.017	0.906	GROUP	1	0.893	0.414
TIME	3	2.476	0.238	TIME	3	0.848	0.552	TIME	3	3.843	0.149
GROUP*TIME	3	0.566	0.645	GROUP*TIME	3	2.102	0.140	GROUP*TIME	3	1.648	0.218
Alkaline Phos U/L				Ratio A:G				CHOL mg/dl			
GROUP	1	0.656	0.477	GROUP	1	7.000	0.077	GROUP	1	0.143	0.730
TIME	3	0.254	0.855	TIME	3	3.524	0.164	TIME	3	8.264	0.058
GROUP*TIME	3	3.959	0.027	GROUP*TIME	3	0.875	0.475	GROUP*TIME	3	0.282	0.837
Amylase U/L				NA mEq/L				TRIG mg/dl			
GROUP	1	0.028	0.878	GROUP	1	1.943	0.258	GROUP	1	0.021	0.893
TIME	3	0.252	0.856	TIME	3	1.429	0.388	TIME	3	5.844	0.091
GROUP*TIME	3	3.242	0.050	GROUP*TIME	3	0.687	0.573	GROUP*TIME	3	0.495	0.691
Mag mEq/L				CL mEq/L				URIC mg/dl			
GROUP	1	31.35	0.011	GROUP	1	2.482	0.213	GROUP	1	3.429	0.161
TIME	3	2.405	0.245	TIME	3	0.792	0.574	TIME	3	0.429	0.748
GROUP*TIME	3	0.257	0.855	GROUP*TIME	3	0.598	0.625	GROUP*TIME	3	1.556	0.239
Hematocrit				K mEq/L				NA/K			
GROUP	1	0.001	0.973	GROUP	1	4.313	0.129	GROUP	1	0.714	0.460
TIME	3	0.054	0.981	TIME	3	5.478	0.098	TIME	3	4.056	0.140
GROUP*TIME	3	3.007	0.061	GROUP*TIME	3	1.072	0.389	GROUP*TIME	3	1.107	0.375
AGAP				TCO2 mEq/L							
GROUP	1	24.00	0.016	GROUP	1	0.030	0.873				
TIME	3	126.94	0.001	TIME	3	3.182	0.184				
GROUP*TIME	3	0.066	0.977	GROUP*TIME	3	2.031	0.150				

Table 10. Independent samples T-Test of significance over recovery time for neonate blood chemistry data for the tank recovery study. The data were collected in Delaware bay during the field seasons of 1999-2000. Significance is indicated by $p < 0.05$.

Time (hours)	pH			pCO ₂ (mmHg)			HCO ₃ (mM)			Lactate (mM)		
	Control	Stress	T value	Control	Stress	T value	Control	Stress	T value	Control	Stress	T value
0	7.415			9.433			6.017			0.950		
0.17		7.101	7.852 **		16.42	-7.568 **		5.433	2.342*		3.360	-5.299 **
1.5	7.367	7.327	0.938	9.933	11.50	-1.410	5.567	5.967	-0.862	2.156	3.180	-0.762
3	7.417	7.408	0.308	9.35	8.950	0.469	5.980	5.520	1.355	0.969	3.523	-3.329 **
6	7.480	7.514	-0.823	7.067	6.367	1.878	5.200	5.100	0.306	0.831	2.067	-1.091
10	7.377	7.428	-3.378 *	9.833	7.467	2.804 *	5.667	4.833	2.210	0.783	1.051	-2.774
24	7.468	7.442	1.680	8.067	7.567	0.098	5.867	5.067	1.648	0.880	1.048	-0.607

[* = $p < 0.05$, ** = $p < 0.01$]

Time (hours)	Glucose (mM)			pO ₂ (mmHg)			Hematocrit		
	Control	Stress	T value	Control	Stress	T value	Control	Stress	T value
0	3.830			27.95			15.58		
0.17		3.830	0.000		20.20	3.652 *		18.33	-3.884**
1.5	----	----	----	32.13	30.87	0.173	14.00	17.83	-2.091
3	4.108	6.365	-3.238*	27.47	28.87	-0.381	15.50	16.17	-0.534
6	4.034	6.384	-2.291	29.30	29.17	0.052	16.83	15.50	0.918
10	----	----	----	27.47	33.27	-0.813	15.00	13.33	2.500
24	4.052	3.405	0.971	31.43	26.60	1.094	17.17	14.83	4.95**

Table 11. Independent samples T-Test of significance over recovery time for neonate electrolyte and enzyme data for the tank recovery study. The data were collected in Delaware bay during the field season of 1999. N = 24 sharks for this analysis. Significance is indicated by $p < 0.05$.

Time (hours)	NA mEq/L			CL mEq/L			K mEq/L			CA mg/dl		
	Control	Stress	T value	Control	Stress	T value	Control	Stress	T value	Control	Stress	T value
0	241.0			219.0			5.973			13.467		
0.17		254.0	-0.383		242.0	-0.616		6.623	6.280**		15.0333	-4.364*
3	290.0	256.0	2.062	264.0	234.0	2.165	5.713	5.843	-0.126	15.00	13.10	1.442
6	266.0	255.0	0.988	243.0	233.0	0.867	5.593	5.387	-0.823	14.00	14.50	-0.719
24	262.0	253.0	0.568	245.0	236.0	0.575	5.553	5.807	0.232	13.07	13.63	-0.921

[* = $p < 0.05$, ** = $p < 0.01$]

Time (hours)	Mg mEq/L			CK U/L			GGT IU/L			Alkaline Phosphatase U/L		
	Control	Stress	T value	Control	Stress	T value	Control	Stress	T value	Control	Stress	T value
0	1.967			53.33			3.333			17.33		
0.17		2.300	-2.774*		129.0	-1.334		1.333	4.243*		12.00	1.387
3	2.233	1.933	1.521	20.33	26.00	-0.295	3.000	3.333	-0.500	10.33	19.33	-2.359
6	2.367	2.200	0.693	11.67	15.00	-0.867	2.000	1.333	1.000	9.333	16.33	-2.970*
24	1.967	2.400	-1.554	8.667	15.00	-1.845	3.333	3.000	0.229	5.033	1.155	0.000

Table 12. Repeated measures analysis for the juvenile tank recovery study data. Data collected in Delaware Bay during 1999-2000. N = 9 sharks for this analysis. Significant values are presented with $p < 0.05$. Variables were tested at T = 0.17, 3 hours.

	df	Mean Square	F	Sig. (P< 0.05)
pH				
TIME	1	0.04	8.19	0.024
TIME * GROUP	1	0.107	21.84	0.002
GROUP	1	0.064	16.12	0.005
Lactate (mM)				
TIME	1	86.09	20.38	0.003
TIME * GROUP	1	3.757	0.890	0.377
GROUP	1	40.60	4.540	0.071
PCO2 (mmHg)				
TIME	1	27.91	2.909	0.132
TIME * GROUP	1	75.40	7.858	0.026
GROUP	1	14.57	1.349	0.284
HCO3 (mM)				
TIME	1	0.000	0.0006	0.981
TIME * GROUP	1	0.047	0.10067	0.760
GROUP	1	3.803	4.80677	0.064
PO2 (mmHg)				
TIME	1	1244	1.46623	0.265
TIME * GROUP	1	79.80	0.09408	0.768
GROUP	1	0.028	0.000	0.996
Hematocrit				
TIME	1	2.250	0.16711	0.695
TIME * GROUP	1	20.25	1.50398	0.260
GROUP	1	0.000	0.000	1.000

Table 13. Independent Sample T-Test of the difference between the control and stressed juvenile serum data for the tank recovery study. The data were collected in Delaware bay during 1999-2000. Significant values are $p < 0.05$.

Dependent Variable:	t-test for Equality of Means		Sig (2-tail)
	t	df	
BUN MG/DL	-0.90	20	0.378
Creatin mg/dl	-2.52	20	<i>0.020</i>
PHOS mg/dl	-1.93	20	0.068
CA mg/dl	-0.67	20	0.511
TPROTEINg/dl	-2.00	20	0.059
ALBg/dl	-1.04	20	0.311
GLOBg/dl	-1.99	12	0.069
RATIO	1.11	20	0.281
NA mEq/L	-2.47	14	<i>0.027</i>
CL mEq/L	-1.15	14	0.269
K mEq/L	-2.19	14	<i>0.046</i>
CO2 mEq/L	1.59	20	0.128
AGAP	-1.84	7	0.108
TBILI mg/dl	-1.29	20	0.212
DBILI mg/dl	0.13	20	0.895
IBILI mg/dl	-1.30	20	0.207
ALP U/l	-2.59	20	<i>0.017</i>
GGT IU/L	-0.35	20	0.729
ALT U/L	1.22	20	0.238
AST U/L	-0.86	20	0.397
LDH U/L	-0.33	20	0.748
CK U/L	-1.69	20	0.107
CHOL mg/dl	-0.98	20	0.340
TRIG mg/dl	-0.30	20	0.764
AMY U/L	-0.45	20	0.660
MAG mEq/L	-1.88	20	0.074
URIC mg/dl	-1.48	20	0.155
NA/K	1.87	14	0.083

Table 14. Independent samples T-Test of significance over recovery time for juvenile blood chemistry data for the tank recovery study. The data were collected in Delaware bay during the field seasons of 1999-2000. Significance is indicated by $p < 0.05$.

Time (hours)	pH			pCO ₂ (mmHg)			HCO ₃ (mM)			Lactate (mM)		
	Control	Stress	T value	Control	Stress	T value	Control	Stress	T value	Control	Stress	T value
0	7.467			9.567			6.500			0.917		
0.17		7.156	9.634**		15.817	-3.812 **		5.417	2.612 *		3.133	-3.494 *
1.5	7.347	7.336	0.331	12.63	10.93	1.337	6.833	5.767	2.213	3.500	6.607	-2.011
3	7.403	7.419	-0.373	11.267	8.833	1.126	6.400	5.533	1.282	2.831	8.742	-3.150*
6	7.464	7.461	0.066	7.433	6.267	1.038	5.200	4.400	2.376	4.617	7.497	-0.937
14	7.390	7.439	-0.654	10.33	8.800	1.02	6.067	5.933	0.496	1.249	1.001	0.584
24	7.478	7.449	0.959	7.25	6.133	2.091	5.25	4.233	2.067	1.100	0.960	0.607

[* = $p < 0.05$, ** = $p < 0.010$]

Time (hours)	Glucose (mM)			pO ₂ (mmHg)			Hematocrit		
	Control	Stress	T value	Control	Stress	T value	Control	Stress	T value
0	2.998			35.07			19.60		
0.17		3.109	-1.014		30.68	0.414		23.25	-2.822*
1.5	----	----	----	35.467	83.80	-1.225	24.00	22.33	1.250
3	2.646	4.367	-3.246**	48.23	52.78	-0.151	21.58	20.25	0.508
6	4.330	8.160	-3.975**	15.00	28.83	-1.470	21.17	20.33	0.332
14	----	----	----	32.60	38.87	-0.726	19.50	19.50	0.000
24	3.358	5.292	-1.102	26.05	44.97	-3.223 *	19.67	18.33	0.649

Table 15. Independent samples T-Test of significance over recovery time for juvenile electrolyte and enzyme data for the tank recovery study.
The data were collected in Delaware bay during the field season of 1999. N = 6 sharks for this analysis. Significance is indicated by $p < 0.05$.

Time (hours)	NA mEq/L			CL mEq/L			K mEq/L			CA mg/dl			Mg mEq/L		
	Control	Stress	T value	Control	Stress	T value	Control	Stress	T value	Control	Stress	T value	Control	Stress	T value
0	252.0			263.5			2.850			13.75			2.200		
0.17		274.3	-3.795*		285.7	-1.324		3.533	-1.973		16.10	-6.040**		2.667	-2.711
3	264.0	291.0	-2.496	247.5	258.0	-0.868	3.150	4.500	-1.480	14.53	16.23	-8.273**	2.533	3.100	-2.292
6	268.0	265.7	0.132	266.0	251.7	0.461	2.900	3.933	1.485	10.07	13.67	-0.714	1.233	3.667	-2.411
24	231.0	264.0	----	204.0	252.0	-1.540	2.500	2.700	1.155	13.30	10.07	0.602	2.300	1.933	0.284

[* = $p < 0.05$, ** = $p < 0.01$]

Time (hours)	CK U/L			Alkaline Phosphatase U/L			Creatinine mg/dl			Phosphorus mg/dl		
	Control	Stress	T value	Control	Stress	T value	Control	Stress	T value	Control	Stress	T value
0	9.500			11.00			0.000			4.400		
0.17		166.3	-3.312*		23.33	-3.974*		0.067	-1.549		6.033	-3.368*
3	24.67	7.667	1.444	16.67	27.67	-2.952	0.033	0.267	-4.950*	4.867	8.733	-3.639*
6	11.00	48.00	-1.212	15.67	31.67	-1.891	0.133	0.367	-1.565	4.733	13.900	-1.904
24	13.50	262.7	-0.872	22.00	24.00	-0.124	0.000	0.100	-1.342	4.700	3.467	1.133

Table 16. Comparison of standard acid-base variable recovery times for the sandbar shark in this study with data from other species commonly referenced in the literature. Species comparisons range from freshwater teleosts to marine teleosts and elasmobranchs.

Species	Metabolic Component	Recovery Time Range	Reference
Rainbow Trout (<i>Oncorhynchus mykiss</i>)	pH	4-8 hours	Ferguson and Tufts 1992; Kieffer et al. 1994; Milligan 1996
	pCO ₂ (mmHg)	4-8 hours	Ferguson and Tufts 1992; Kieffer et al. 1994
	HCO ₃ ⁻ (mM)	4-8 hours	Ferguson and Tufts 1992; Kieffer et al. 1994
	Lactate (mM)	8-12 hours	Wood et al. 1983; Kieffer et al. 1994; Milligan 1996
	Glucose (mg/dl)	up to 24 hours	Wydoski et al. 1976
	Na (mEq/L)	12 hours	Wood et al. 1983
	Cl (mEq/L)	8-12 hours	Wydoski et al. 1976; Wood et al. 1983
	K (mEq/L)	12 hours	Wood et al. 1983
Atlantic Salmon (<i>Salmo salar</i>)	pH	4 hours	Wilkie et al. 1997
Striped Bass (<i>Morone saxatilis</i>)	pH	2-4 hours	Cech et al. 1996
	Lactate	2-4 hours	
Northern Pike (<i>Esox lucius</i>)	K (mEq/L)	48 hours	Soirio and Oikari 1976
	Ca (mEq/L)	48 hours	
	Mg (mEq/L)	48 hours	
Skipjack tuna (<i>Katsuwonus pelamis</i>)	pH	20 mins	Perry et al. 1985
	pCO ₂ (mmHg)	20 mins	
	HCO ₃ ⁻ (mM)	30 mins	
Dogfish (<i>Scyliorhinus stellaris</i>)	pH	6-8 hours	Püper et al. 1972
	pCO ₂ (mmHg)	1 hour	
	HCO ₃ ⁻ (mM)	6-8 hours	
	Lactate (mM)	24 hours	
Smooth Dogfish (<i>Mustelus canis</i>)	Hematocrit	24 hours	Barham and Schwartz 1992
Port Jackson shark (<i>Heterodontus portusjacksoni</i>)	Hematocrit	12 hours	Cooper and Morris 1998
Dusky shark (<i>Carcharhinus obscurus</i>)	pH	24 hours	Cliff and Thurman 1984
	pCO ₂ (mmHg)	24 hours	
	HCO ₃ ⁻ (mM)	24 hours	
	Lactate (mM)	24 hours	
	Na (mEq/L)	24 hours	
	Cl (mEq/L)	24 hours	
	K (mEq/L)	24 hours	
	CK (U/L)	24 hours	
	Glucose (mg/dl)	> 24 hours	
	Mg (mEq/L)	> 24 hours	
Sandbar shark (<i>Carcharhinus plumbeus</i>)	pH	< 3 hours	This study
	pCO ₂ (mmHg)	< 3 hours	
	HCO ₃ ⁻ (mM)	< 3 hours	
	Lactate (mM)	10-14 hours	
	Na (mEq/L)	3 hours	
	Cl (mEq/L)	3 hours	
	K (mEq/L)	3 hours	
	CK (U/L)	3 hours	
	Glucose (mg/dl)	< 24 hours	
	Mg (mEq/L)	3 hours	

Table 17. Original capture and recapture information from tag recapture information provided to the Apex Predators Program, NMFS, Narragansett, RI. Sharks from both years (1999-2000) have been recaptured either in DE Bay or along the Atlantic seaboard. Blank data indicates either unknown value or an unreliable estimate. All sharks were returned to the water with the same tags to aid in future recaptures. RR = rod and reel (field) phase of the study, R+R is rod and reel gear used to recapture the sharks.

Original Capture Information							Recapture Information				
Sample #	Tag #	FL (cm)	TL (cm)	Weight (kg)	Sex	Participation in study	Release Condition	Gear	Time at Liberty	Distance Traveled (miles)	FL (cm)
SB9906	BR4613	52	61	1.5	F	RR: 0.56 sec	1	Longline	367 days	8 South East	62
SB9909	BR4616	51	59	1.3	M	RR: 10.13 min	2	R+R	13 days	1.3 South East	59
SB9918	BR4627	51	58	1.6	M	RR: 8.07 min	2	R+R	29 days	1 South East	
SB9927	BR4636	48	54	1.3	M	stress T = 0	2	R+R	74 days	20 South East	53
SB9929	BR4638	52	60	1.7	F	stress T = 0	1	R+R	1.45 hours	0.01 South East	52
SB0043	BR1541	51	59	1.6	F	stress T = 3	2	Gillnet	92 days	219 South	52
SB0050	BR1547	53	61	1.5	M	control T = 10	2	R+R	12 days	4 North	
SB0051	BR1544	50	57	1.4	M	stress T = 10	2	R+R	12 days	14 North West	
SB0062	BR1556	50	58		M	RR: 20.12 min		R+R	34 days	2 North West

Figure 1. Physiological changes in blood parameters over increasing fight time (minutes) for sandbar sharks in Delaware bay (1999-2000). All graphs shown are significant with $p < 0.05$. Fish that have been recaptured are noted by a plus (+) symbol.

Figure 2. Physiological changes in blood parameters over increasing weight (kg) for sandbar sharks in Delaware bay (1999-2000). All graphs shown are significant with $p < 0.05$. There are three juvenile sharks driving the relationship, 2.8kg, 3.3kg and 3.3kg. None of these larger fish have been recaptured.

Figure 3. Physiological changes in blood parameters over increasing fork length (cm) for sandbar sharks in Delaware bay (1999-2000). All graphs shown are significant with $p < 0.05$. There are three juvenile sharks driving the relationship, 64cm, 67cm and 68cm. None of these larger fish have been recaptured.

Figure 4. Physiological changes in blood parameters over increasing total length (cm) for sandbar sharks in Delaware bay (1999-2000). All graphs shown are significant with $p < 0.05$. There are three juvenile sharks driving the relationship, 74cm, 78cm and 78cm. None of these larger fish have been recaptured.

Figure 5. Physiological changes in blood gases over recovery time for neonatal sandbar sharks in Delaware bay (1999-2000). All bars are mean values with 95% confidence intervals. The open bar indicates the period of exercise, with R indicating the resting value. The resting value was taken from control fish at T=0, they were sampled and released. The solid bars are control and hatched are stress values. The samples were taken at 0, 1.5, 3, 6, 10, and 24 hours after the 10-minute angling event. Significance is noted with an asterisk (*) and the associated p-value is given.

Figure 6. Physiological changes in blood gases over recovery time for juvenile sandbar sharks in Delaware bay (1999-2000). All bars are mean values with 95% confidence intervals. The open bar indicates the period of exercise, with R indicating the resting value. The resting value was taken from control fish at T=0, they were sampled and released. The solid squares are control and triangles are stress values. The samples were taken at 0, 1.5, 3, 6, 14, and 24 hours after the 10-minute angling event. Significance is noted with an asterisk (*) and the associated p-value is given.

Figure 7. Physiological changes in hematocrit, lactate and glucose over recovery time for neonate sandbar sharks in Delaware bay (1999-2000). All other notations are the same as Figure 5

Figure 8. Physiological changes in hematocrit, lactate and glucose over recovery time for juvenile sandbar sharks in Delaware bay (1999-2000). All other notations are the same as Figure 6

* (p=0.012)

* (p=0.05)

Figure 9. Physiological changes in electrolytes over recovery time for neonatal sandbar sharks in Delaware bay (1999-2000). All other notations are the same as figure 5.

Figure 10. Physiological changes in electrolytes over recovery time for juvenile sandbar sharks in Delaware bay (1999-2000). All other notations are the same as figure 6.

Figure 11. Physiological changes in enzymes over recovery time for neonatal sandbar sharks in Delaware bay (1999-2000). All other notations are the same as figure 5.

Figure 12 - Physiological changes in enzymes and metabolites over recovery time for juvenile sandbar sharks in Delaware bay (1999-2000). All other notations are the same as figure 6.

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