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Immediate and delayed effects of gill-net capture on acid–base balance and intramuscular lactate concentration of gummy sharks, *Mustelus antarcticus*☆

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ABSTRACT

Many sharks are captured as untargeted by-catch during commercial fishing operations and are subsequently discarded. A reliable assessment of the proportion of discarded sharks that die post-release as a result of excessive physiological stress is important for fisheries management and conservation purposes, but a reliable physiological predictor of post-release mortality has not been identified. To investigate effects of gill-net capture on the acid–base balance of sharks, we exposed gummy sharks, *Mustelus antarcticus*, to 60 min of gill-net capture in a controlled setting, and obtained multiple blood and muscle tissue samples during a 72-h recovery period following the capture event. Overall mortality of gummy sharks was low (9%). Blood pH was significantly depressed immediately after the capture event due to a combination of respiratory and metabolic acidosis. Maximum concentrations of plasma lactate (9.9 ± 1.5 mmol L^{-1}) were measured 3 h after the capture event. Maximum intramuscular lactate concentrations (37.0 ± 4.6 μmol g^{-1}) were measured immediately after the capture event, and intramuscular lactate concentrations were substantially higher than plasma lactate concentrations at all times. Sharks in poor condition had low blood pH and high intramuscular lactate concentration, but blood pH does not appear to be a reliable predictor of survival. Suitability of intramuscular lactate concentration as predictor of delayed mortality deserves further investigation.

1. Introduction

Fishing capture has been shown to elicit profound physiological changes in elasmobranchs (e.g. Cliff and Thurman, 1984; Manire et al., 2001; Mandelman and Skomal, 2009; Frick et al., 2010a; Frick et al., 2010b), but information on the post-release fate of sharks that are incidentally captured during fishing operations and subsequently discarded alive is still scarce compared with the large amount of publications addressing delayed effects of capture stress in teleosts. Discard rates of sharks are high in global fisheries (see Stevens et al., 2000), and reliable estimates of discard mortality are essential to assess the overall impact of fishing pressure on shark populations. Identification of a suitable physiological predictor of delayed mortality could facilitate an accurate assessment of the proportion of sharks that are discarded alive, but die post-release as a direct consequence of physiological capture stress.

The problem of discard mortality will ultimately have to be addressed with data collected under natural conditions, but circumstances in the wild allow only one opportunity for blood sampling — immediately after the capture event and prior to the shark's release. Our recent study on the post-capture condition of gummy sharks, *Mustelus antarcticus*, found that sharks which died post-release displayed significantly higher blood lactate and potassium concentrations than surviving sharks, but these differences did not become apparent until hours after the capture event (Frick et al., 2010a). Blood lactate and potassium concentrations measured immediately after the capture event did not allow a prediction of a shark's probability of survival.

The physiological mechanisms underlying delayed mortality are still not fully understood. Sharks undergo metabolic and/or respiratory blood acidosis following capture, handling, and release (e.g. Cliff and Thurman, 1984; Mandelman and Farrington, 2007), and Mandelman and Skomal (2009) found a positive correlation between the degree of blood acid–base disturbance and at-vessel mortality of carcharhinid sharks. However, Wood et al. (1983) concluded that blood acidosis was unlikely to cause delayed mortality in fish, and suggested that the key toxic event leading to delayed mortality occurred in the intracellular compartment as a consequence of an intracellular build-up of metabolic protons. Richards et al. (2003) found that white muscle lactate production was correlated with metabolic acidosis in spiny dogfish, *Squalus acanthias*, following exhaustive exercise, but a possible connection between the extent of blood acid–base disturbance or intramuscular lactate concentration and post-capture condition of sharks has not been investigated.

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To examine if a connection between acid–base disturbance and the post-capture fate of a shark exists, we subjected gummy sharks to gill-net capture in a controlled setting and obtained blood and muscle tissue samples at various points in time during a 72-h recovery period following the capture event to monitor changes in blood acid–base balance and intramuscular lactate concentration. We hypothesized that intramuscular lactate concentration of gummy sharks would be significantly elevated and blood pH significantly depressed immediately after the capture event, and expected that the probability of survival would decrease with increasing intramuscular lactate concentrations. Our goal is to use this information to better understand the physiological and biochemical consequences of capture stress in this species in order to facilitate the development of better handling practices and reduce the negative effects of fisheries capture on shark by-catch.

2. Methods

2.1. Animal collection and experimental treatment

In August 2008, gummy sharks (n = 42; total length (TL) = 97 ± 2 cm (mean ± SE)) were collected by a commercial fisherman using longlines in coastal waters near Mallacoota (Victoria, Australia), and were transported to the laboratory facilities in Queenscliff (Victoria, Australia) in a 4000-liter truck-mounted fish transport tank containing aerated seawater. Holding facilities and husbandry protocols were as described in Frick et al. (2009; 2010a). Ambient water temperature ranged 12.1–14.1 °C; oxygen saturation was >95%. In September 2008, gummy sharks (n = 32; TL = 98 ± 2 cm) were individually subjected to 60 min of gill-net capture in captivity using the methods described in Frick et al. (2009). The degree of net entanglement during the capture event was categorized as light (not entangled around the gill region or only one mesh around gills), medium (gilled with multiple meshes around gills, but gill ventilation not obstructed), and heavy (completely restrained with multiple meshes around gills obstructing gill ventilation). General post-capture condition of sharks was qualitatively assessed from swimming behavior (voluntary movement vs. resting on the tank bottom), skin coloration (normal vs. pale), and degree of struggling during handling (vigorous struggling vs. little or no struggling).

2.2. Blood and tissue sample collection

Blood samples (1 ml) were collected via caudal venipuncture at 0, 3, 24, and 72 h post-capture as described in Frick et al. (2009). As a result of our tissue sampling regime described below, sample size of blood samples decreased over time. At each of these 4 sampling points, 8 sharks were quickly killed by decapitation and muscle tissue was collected from the epaxial muscle adjacent to the first dorsal fin. Tissue samples were immediately frozen in liquid nitrogen, and were stored at −80 °C until analyzed. No tissue was collected from one shark that died post-capture in the 72-h group, because the time of death was unknown. Blood and tissue samples were collected from an additional 10 gummy sharks (TL = 94 ± 4 cm) that were killed without prior treatment to establish reference values of minimally stressed sharks (baseline). Further gummy shark tissue samples were collected from the epaxial muscle adjacent to the first as well as the second dorsal fin of sharks (n = 9; TL = 90 ± 4 cm) that died during experiments for a different study in March 2008, and were kept frozen at -20 °C for later determination of muscle buffering capacity.

2.3. Blood analysis

Immediately after obtaining a sample, 3–4 drops of whole blood were used for pH and PCO_2 analysis in an i-Stat portable blood gas analyzer (CG4+ cartridge; Abbott Laboratories, Chicago, IL, USA). In addition, approximately 70 µl of whole blood was drawn into a microcapillary tube and centrifuged at 10000 rpm for 5 min for hematocrit determination. The remaining sample volume was centrifuged at 10000 rpm for 5 min, the plasma portion was removed and stored at −20 °C for later analysis. Plasma lactate and glucose concentrations were determined with a YSI 2300 StatPlus Lactate and Glucose Analyzer (YSI, Yellow Springs, OH, USA). Plasma potassium concentration was determined by an external pathology laboratory using a Beckman Synchron LX20PRO Analyzer (Beckman Coulter, Fullerton, California, USA).

2.4. Muscle tissue analysis

Frozen muscle tissue samples (0.5 g) were crushed to a fine powder using a stainless steel concussion mortar pre-cooled in liquid nitrogen. The powder was transferred into 5 ml of ice-cold 0.6 M perchloric acid and homogenized for 20 s. Samples were always placed on ice between processing steps. The homogenate was centrifuged for 5 min at 13000 rpm, and 3.6 ml of the supernatant was decanted into a small glass bottle containing 2 µl methyl orange. The solution was neutralized by adding approximately 0.2 ml of potassium bicarbonate, and was placed on ice for at least 2 h until precipitation of perchlorate ions stopped. The supernatant was transferred into Eppendorf tubes and stored frozen at −20 °C for later analysis. Lactate concentration of the extract was determined with a YSI 2300 StatPlus Lactate and Glucose Analyzer. Obtained values were then multiplied by the respective dilution factor resulting from the above procedure to derive the intramuscular lactate concentration. Results are presented as µmol g (wet tissue)$^{-1}$.

Muscle tissue buffering capacity was determined following Castellini and Somero (1981). In brief, 0.5 g of frozen muscle tissue was homogenized in 11 ml of 0.9% NaCl solution for 20 s, transferred into a plastic chamber with a magnetic stirrer and a pH probe (Hanna H1 8314 pH meter and Hanna H1 1230 combination pH electrode; Hanna Instruments, Woonsocket, RI, USA) and titrated from pH 6.0 to pH 7.0 by incremental addition of 10 µl of 0.2 M NaOH.

2.5. Conversions and calculations

The i-Stat portable blood gas analyzer is designed for use with mammalian blood and is therefore thermostatted to 37 °C. Hence, PCO_2 and pH results produced by the i-Stat were corrected for temperature to more closely reflect *in vivo* values (15 °C). Temperature conversions of pH and PCO_2 values were calculated using the respective equations from Mandelman and Skomal (2009):

$$pH_{TC} = pH_M - 0.011(T - 37) \quad (1)$$

$$PCO_{2TC} = PCO_{2M}\left(10^{-0.019\Delta T}\right) \quad (2)$$

with temperature $T = 15$, and $\Delta T = 37 - 15 = 12$.

It must be noted here that while relative changes of blood gas values determined with the i-Stat blood gas analyzer are reliable, caution should be exercised with temperature corrected absolute values (Gallagher et al., 2010).

Bicarbonate (HCO_3^-) concentrations were calculated using the Henderson–Hasselbalch equation with temperature corrected (15 °C) PCO_2 and pH values, and the constants pK' and αCO_2 experimentally determined for the nursehound, *Scyliorhinus stellaris,* by Randall et al. (1976). Blood metabolic acid load was estimated following Milligan and Wood (1986):

$$\Delta H_m^+ = [HCO_3^-]_1 - [HCO_3^-]_2 - \beta(pH_1 - pH_2) \quad (3)$$

Due to a lack of published values for gummy sharks, the whole blood non-bicarbonate buffer capacity β (−7.13 mmol pH unit^{-1} l^{-1}) for spiny dogfish, *S. acanthias*, was taken from Gilmour et al. (2002).

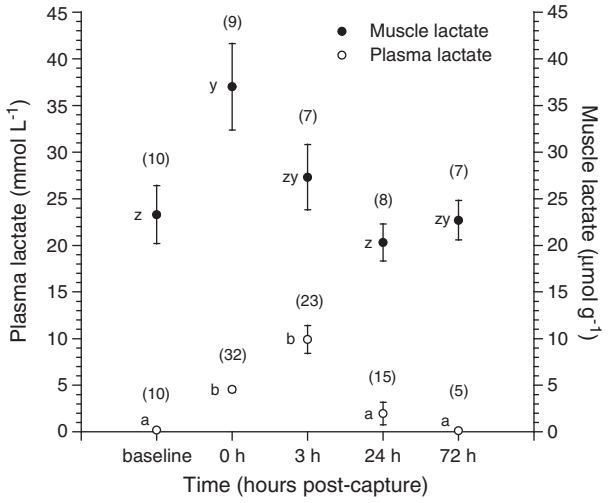

Fig. 1. Plasma and intramuscular lactate concentrations (mean ± SE) measured in minimally stressed (baseline) gummy sharks, *Mustelus antarcticus*, and at various points in time during a 72-h recovery period following 60 min of gill-net capture in a controlled setting. Numbers in parentheses represent underlying sample size. Differing alphabetic letters denote significant difference between individual data points (Tukey's post-hoc test, $P<0.05$).

2.6. Statistical analysis

Differences in blood variables at different points in time were analyzed using one-way Analysis of Variance (ANOVA) followed by Tukey's post-hoc test. Results for pH and PCO_2 were analyzed using nonparametric Kruskal–Wallis one-way ANOVA on Ranks followed by Tukey's post-hoc test. Changes in metabolic acid load were analyzed using nonparametric Wilcoxon Rank Sum test. Some variables were log-transformed prior to statistical analysis to meet assumptions of their respective tests, but all results presented here are non-transformed data. Statistical data analysis was performed using the R software package (R Development Core Team, 2006). Statistical significance was determined at $\alpha = 0.05$ and results are presented as mean ± SE unless stated otherwise.

3. Results

Overall gummy shark mortality was low. One shark died during the 60-min capture event and two sharks died post-capture. One shark was moribund at the end of the capture event and was killed for tissue sampling immediately after capture. The only gummy shark that died during the capture event had the highest intramuscular lactate concentration of all sharks (53.5 μmol g^{-1}). It was heavily entangled and its jaws were held shut by the net meshing. The second highest intramuscular lactate concentration (51.3 μmol g^{-1}) was measured in a shark that was also heavily entangled and was moribund at the end of the capture event. Intramuscular lactate concentration in one shark that died 25 min after the capture event was 43.0 μmol g^{-1}, but the tissue sample was not obtained until 80 min after the capture event. Sample size of dead sharks was insufficient to integrate these findings into the statistical analyses.

Both intramuscular and plasma lactate concentrations were significantly elevated immediately after the capture event (Tukey's post-hoc tests, $P<0.05$), but mean plasma lactate concentration did not peak until 3 h post-capture (9.9 ± 1.5 mmol l^{-1}), whereas mean maximum lactate concentration in muscle tissue (37.0 ± 4.6 μmol g^{-1}) was measured at 0 h, and was already decreasing at 3 h (Fig. 1). After 24 h, intramuscular and plasma lactate concentration were back to baseline levels. Mean plasma glucose concentration in minimally stressed sharks was 3.4 ± 4.6 mmol l^{-1}, and did not change significantly in response to the capture event. Mean plasma potassium concentration was significantly elevated at 3 h compared with baseline (Tukey's post-hoc tests, $P<0.05$; Fig. 2), and mean hematocrit was significantly lower at 24 h and 72 h compared with values measured at 0 h (Tukey's post-hoc tests, $P<0.05$; Fig. 3).

Statistical tests performed on both uncorrected (37 °C) and temperature corrected (15 °C) blood gas data yielded identical results. Consequently, significant events are only indicated for *in vivo* values (15 °C) in figures, and absolute values presented here refer to *in vivo* values unless stated otherwise. Mean blood CO_2 tension (PCO_2) at 0 h post-stress (5.6 ± 0.4 mm Hg) was significantly higher than baseline (2.8 ± 0.1 mmHg) (Tukey's post-hoc tests, $P<0.05$), and returned to levels close to baseline (3.5 ± 0.1 mmHg) at 3 h post-capture (Fig. 4). Similarly, mean blood bicarbonate (HCO_3^-) concentration at 0 h was higher (4.6 ± 0.6 mmol l^{-1}) than baseline (2.4 ± 0.1 mmol l^{-1}) (Fig. 5). This difference was not statistically significant due to *P*-value adjustments if values measured at 3, 24 and 72 h were included in the analysis (Tukey's post-hoc test: $t = 2.62$, adjusted $P = 0.07$), but was statistically significant in isolated analysis (Wilcoxon's rank sum test: $W = 90$, $P<0.05$). Bicarbonate concentrations measured at 3, 24, and 72 h were significantly lower than at 0 h (Tukey's post-hoc tests, $P<0.05$), and were not significantly different from baseline.

Mean blood pH of gummy sharks was significantly lower at 0 h (7.30 ± 0.03) and at 3 h (7.48 ± 0.04) post-capture compared with baseline (7.64 ± 0.01) (Tukey's post-hoc tests, $P<0.05$; Fig. 6). Immediately after capture, blood pH of sharks that died or were in very poor condition after the capture event, as indicated by pale skin coloration, little or no struggling during handling, and resting on the tank bottom upon release, was below the overall mean observed. Metabolic proton load ΔH^+_m increased significantly between baseline and 0 h, decreased significantly between 3 h and 24 h, and increased again between 24 h and 72 h post-capture (Wilcoxon rank sum tests, $P<0.05$; Fig. 7).

Gummy shark muscle buffering capacity of samples taken from muscle adjacent to the first dorsal fin was not significantly different from samples taken from muscle adjacent to the second dorsal fin (paired *t*-test; $df = 8$, $t = 0.49$, $P>0.05$). Overall buffering capacity was 58.4 ± 2.2 slykes (μmoles of base required to titrate the pH of 1 g wet weight of muscle by one pH unit, over the range of pH 6.0 to pH 7.0).

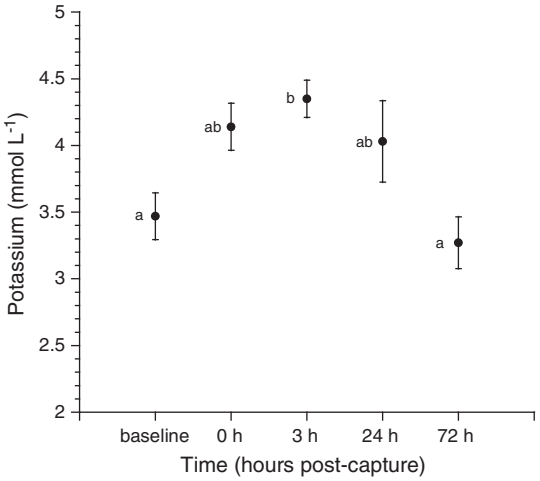

Fig. 2. Plasma potassium concentrations (mean ± SE) measured in minimally stressed (baseline) gummy sharks, *Mustelus antarcticus*, and at various points in time during a 72-h recovery period following 60 min of gill-net capture in a controlled setting. Underlying sample sizes are as for plasma lactate concentrations presented in Fig. 1. Other details are as in caption to Fig. 1.

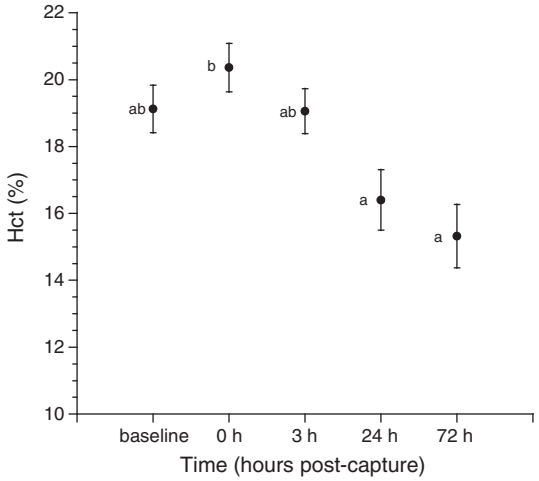

Fig. 3. Hematocrit (mean ± SE) measured in minimally stressed (baseline) gummy sharks, *Mustelus antarcticus*, and at various points in time during a 72-h recovery period following 60 min of gill-net capture in a controlled setting. Underlying sample sizes are as for plasma lactate concentrations presented in Fig. 1. Other details are as in caption to Fig. 1.

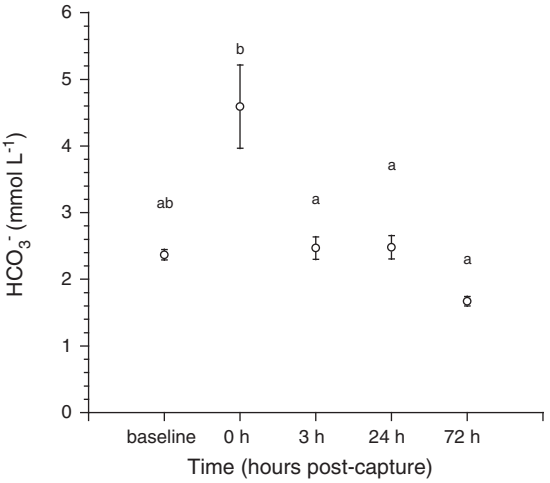

Fig. 5. Whole blood bicarbonate (HCO_3^-) concentrations derived from temperature corrected (15 °C) values for pH and PCO_2 measured in minimally stressed (baseline) gummy sharks, *Mustelus antarcticus*, and at various points in time during a 72-h recovery period following 60 min of gill-net capture in a controlled setting. Underlying sample sizes are as for plasma lactate concentrations presented in Fig. 1. Other details are as in caption to Fig. 1.

4. Discussion

Maximum plasma lactate concentrations of gummy sharks measured following a 60-min gill-net capture event in the present study were comparable to those measured in surviving gummy sharks after a 30-min and a 120-min gill-net capture event in an earlier study (Frick et al., 2010a). However, gummy shark mortality was much lower in the present study. The reasons for these differences are unclear. Water temperature during experiments for the present study was on average 2–3 °C lower than during experiments for the previous study. Increased water temperature can lead to an increase in capture-related mortality of teleosts (e.g. Olla et al., 1998; Davis and Olla, 2001) and elasmobranchs (e.g. Hueter and Manire, 1994; Morgan and Burgess, 2007), but temperature differences in those studies were generally >10 °C, and it is questionable if a temperature difference of only a few degrees could explain the rather distinct difference in mortality between the two studies. Animal collection protocol was identical in the present and our earlier study (Frick et al., 2010a), but it may be that sharks used in the present study were in better general condition, and a larger sample size may have eliminated such a possible confounding factor.

The highest intramuscular lactate concentrations of gummy sharks were measured immediately after capture. Maximum concentrations observed in our study (>50 μmol g^{-1}) exceeded values estimated for the nursehound (34–45 μmol g^{-1}), a shark of a size comparable to the gummy shark, following exhaustive exercise (Holeton and Heisler, 1983). It is conspicuous that gummy sharks with high intramuscular lactate concentrations were in poor condition. The small number of observations in our study does not allow any conclusive remarks, but a possible connection between intramuscular lactate concentration (as an indicator of intracellular metabolic proton generation) and capture-related immediate and delayed mortality of sharks undoubtedly deserves further investigation. If delayed mortality is in fact connected to excessive production of metabolic protons in muscle tissue, then sharks with a higher anaerobic capacity are expected to be more likely to succumb to the effects of a capture event, because they are able to generate a higher metabolic acid load in a short period of time than sharks with a lower potential for anaerobic metabolism.

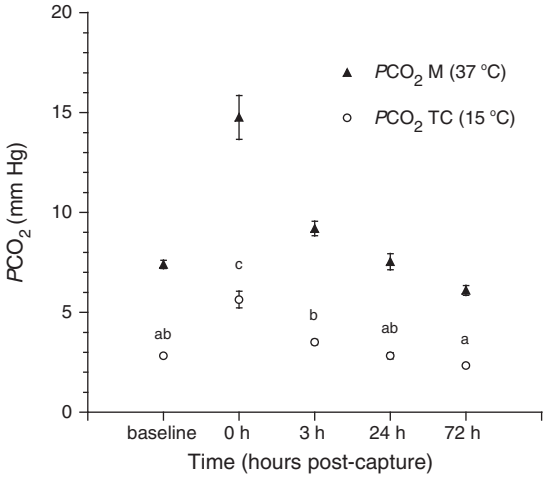

Fig. 4. Uncorrected (M) and temperature corrected (TC) whole blood CO_2 tensions (PCO_2) (mean ± SE) measured in minimally stressed (baseline) gummy sharks, *Mustelus antarcticus*, and at various points in time during a 72-h recovery period following 60 min of gill-net capture in a controlled setting. Underlying sample sizes are as for plasma lactate concentrations presented in Fig. 1. Other details are as in caption to Fig. 1.

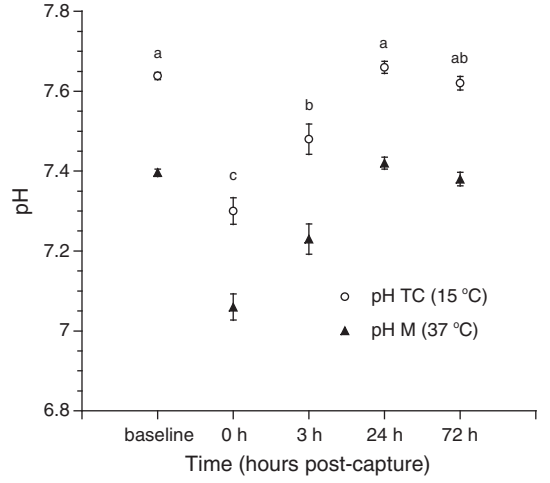

Fig. 6. Uncorrected (M) and temperature corrected (TC) whole blood pH (mean ± SE) measured in minimally stressed (baseline) gummy sharks, *Mustelus antarcticus*, and at various points in time during a 72-h recovery period following 60 min of gill-net capture in a controlled setting. Underlying sample sizes are as for plasma lactate concentrations presented in Fig. 1. Other details are as in caption to Fig. 1.

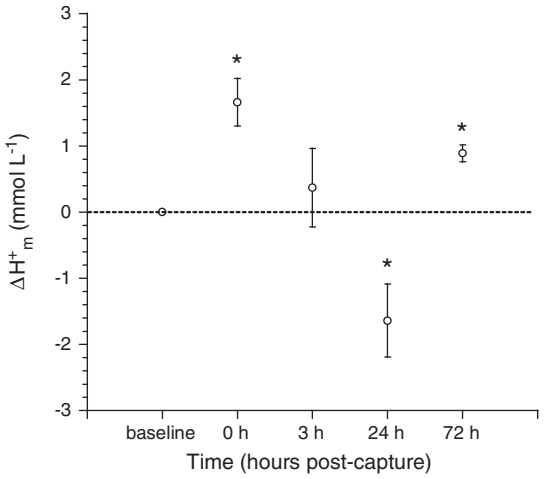

Fig. 7. Estimated blood metabolic acid load (ΔH^+_m) in gummy sharks, *Mustelus antarcticus*, at various points in time during a 72-h recovery period following 60 min of gill-net capture in a controlled setting. Underlying sample sizes are as for plasma lactate concentrations presented in Fig. 1. ΔH^+_m at rest (baseline) is, per definition, equal to 0. Asterisks denote statistically significant increase or decrease in metabolic acid load (Wilcoxon rank sum test; $P<0.05$).

Intramuscular lactate concentrations measured in minimally stressed sharks in the present study are considerably higher than other published values (Holeton and Heisler, 1983; Richards et al., 2003). In contrast to Richards et al. (2003), we did not anesthetize the sharks prior to terminal sampling, because anesthesia has been shown to affect stress-related blood variables (e.g. Frick et al., 2009). It is possible that brief periods of struggling during handling (20–30 s) prior to terminal sampling led to an increase in intramuscular lactate concentration, resulting in elevated "baseline" values in the present study. Alternatively, an increase in blood lactate following anesthesia (Frick et al., 2009) may be due to vasodilation induced by the anesthetic agent, resulting in increased lactate clearance out of muscle tissue during anesthesia, in which case resting values for intramuscular lactate obtained from anesthetized animals may be underestimates. Regardless, we applied the same sampling technique throughout the present study, and are confident that actual capture stress effects were detected at 0 h and 3 h post-stress, given that blood variable values returned to pre-stress levels after 24–72 h.

White muscle buffering capacity of gummy sharks determined in the present study (58.4 slykes) was at the upper end of reported values for elasmobranchs, similar to those measured in the gray smoothhound, *Mustelus californicus*, (52.5 slykes), the leopard shark, *Triakis semifasciata*, (54.3 slykes), and the highly active shortfin mako shark, *Isurus oxyrinchus* (60.7 slykes) (Dickson et al., 1993). This suggests that gummy sharks routinely produce large amounts of lactate in myotomal white muscle, because the capacity to anaerobically generate lactate (lactate dehydrogenase activity) is positively correlated with intracellular buffering capacity of white muscle (Castellini and Somero, 1981; Dickson et al., 1993).

Temperature-corrected baseline blood pH values determined for gummy sharks in the present study are in good accordance with presumed steady-state pH values published for the nursehound (Piiper et al., 1972; Holeton and Heisler, 1983), spiny dogfish, *S. acanthias*, and dusky smoothhound, *M. canis* (Mandelman and Skomal, 2009). Mean blood pH of gummy sharks was significantly depressed after the 60-min capture event in our study, and was lowest immediately after the capture event. Various other studies found a similar extent of blood pH depression (0.4–0.6 units) in elasmobranchs as a result of capture stress or exhaustive exercise (Piiper et al., 1972; Holeton and Heisler, 1983; Cliff and Thurman, 1984; Mandelman and Farrington, 2007), but in contrast to the present study, blood pH depression in those studies (with the exception of Mandelman and Farrington (2007)) was greatest 1–2 h after the stressful event. The aforementioned studies provide a much higher temporal resolution than ours, and it is possible that we did not detect the greatest extent of pH depression with the sampling regime used in the present study. Conversely, results of the aforementioned studies may have been confounded by repeated blood sampling at a high frequency, which has been shown to affect stress-related blood variables (Frick et al., 2009).

Blood pH values of gummy sharks that died or were in very poor post-capture condition were among the lowest observed immediately after capture in the present study, suggesting that blood pH may serve as a good indicator for the current condition of a shark. However, other sharks with similarly low (or lower) blood pH were able to recover and survived. These observations suggest that blood pH measured immediately after a capture event may not be a suitable predictor of post-release survival of this species.

Changes in bicarbonate levels observed in gummy sharks in the present study were relatively small compared with results of earlier publications (Piiper et al., 1972; Holeton and Heisler, 1983; Cliff and Thurman, 1984). In contrast to these previously published studies, which found depressed blood bicarbonate levels in nursehounds following exhaustive exercise (Holeton and Heisler, 1983) and in dusky sharks following rod and line capture (Cliff and Thurman, 1984), we measured significantly elevated bicarbonate concentrations immediately after the capture event. Mandelman and Skomal (2009) suggested that blood acidosis of dusky sharks following longline capture was of exclusively metabolic nature, and blood acidosis in nursehounds that were exercised to exhaustion in the study of Holeton and Heisler (1983) was also most likely caused by an accumulation of metabolic protons. In the present study, metabolic proton load was increased after the capture event, indicating that the observed blood acidosis was at least partially driven by a metabolic component. However, during metabolic acidosis bicarbonate ions take up excess metabolic protons, which leads to a decrease in blood bicarbonate concentration. Conversely, blood bicarbonate concentration temporarily increases during respiratory acidosis due to an acute CO_2 overload in the body of an animal caused by impeded or insufficient gas exchange. Hence, elevated blood bicarbonate levels observed at 0 h in the present study are indicative of a greater contribution of the respiratory component to blood acidosis in gummy sharks. The extent of respiratory acidosis experienced during and after a capture event may be influenced by the ability of a shark to maintain oxygen delivery during a stressful situation (Mandelman and Skomal, 2009), and forced hypoventilation caused by restraint and net entanglement around the gill region, as was the case with some gummy sharks in our study, will undoubtedly exacerbate respiratory acidosis.

Plasma potassium concentration of gummy sharks was significantly elevated following gill-net capture. Hyperkalemia is a well documented consequence of capture stress in sharks, and is presumably a result of potassium efflux out of muscle cells due to an excessive intracellular build-up of metabolic protons (e.g. Cliff and Thurman, 1984; Manire et al., 2001; Frick et al., 2010a). The possibility of hyperkalemia as proximate cause of death in sharks that die post-release was discussed in detail in Frick et al. (2010a). Mean hematocrit of gummy sharks was not significantly elevated after capture, but decreased continually during the 72-h recovery period. Declining hematocrit has previously been observed in serially bled elasmobranchs and teleosts, and is attributed to repeated blood extraction (Turner et al., 1983; Frick et al., 2010a).

5. Conclusions

The results of our study confirm the findings of previously published studies that fisheries capture can lead to severe disruption of the acid–base balance in some elasmobranchs. However, blood pH

measured immediately after a capture event does not appear to be a reliable predictor of post-release survival in gummy sharks, even though it may serve as a good indicator of the current condition of a shark immediately after capture. Using stress-related blood variables as predictors of post-release survival may not be the right path for field-based studies, where only one sample can be obtained immediately after the capture event, because changes in blood chemistry occur with a certain delay. Our data suggest that investigating changes in muscle biochemistry in response to capture stress is likely to yield more promising results. If severe intracellular acidosis as a result of excessive production of metabolic protons triggers a cascade of events that ultimately leads to delayed mortality, then sharks with a higher anaerobic capacity are expected to be more susceptible to the effects of capture stress. The possibility of such a correlation undoubtedly deserves further investigation.

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