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A study of delayed capture mortality syndrome in skipjack tuna, *Katsuwonus pelamis* (L).

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Abstract. This study was initiated to determine the cause(s) of delayed mortality in newly captured skipjack tuna, *Katsuwonus pelamis* (L.), being held at the National Marine Fisheries Service Kewalo Research Facility. Sixty-four per cent of 244 skipjack tuna delivered to the facility died, usually on the second or third day after capture. The capture history, morphological data, serum chemistry (21 standard parameters), haematology, and histological samples of major organs, were obtained from 30 fish sampled at sea immediately after capture, or after approximately 4, 9, 24, 48 or 500+ h in captivity. The cause(s) of death in these fish could not be attributed to anoxia, disseminated intravascular coagulation, lactic acidosis, capture myopathy or infection. Post-capture haemodilution is hypothesized as a major factor of delayed capture mortality syndrome in skipjack tuna.

Introduction

The capture and maintenance of large marine fish is necessary for public aquaria display, aquaculture or scientific study. The capture and transport of a large fish is a difficult and expensive task which too often results in the death of the fish. The stress of capture and adaptation to captivity initiates complex physiological responses by the fish which appear to lead to dysfunction in organ systems resulting in death (Love 1970; Mazeaud, Mazeaud & Donaldson 1977). The initial stress of capture has been reported to cause almost immediate death in wild mammals from simple trauma, shock (i.e. low blood pressure) and subsequent pooling of blood (Soma, Burrows & Marshal 1974). Capture-related deaths in fish have been attributed to anoxia (Hall 1930; Pawson & Lockwood 1980) or lactic acidosis (Black 1958; Beamish 1966; Wardle 1978). Delayed mortalities following capture in fish have been attributed to disseminated intravascular coagulation (Smith 1980), loss of osmotic homeostasis (Stevens 1972; Pic, Mayer-Gustan & Maetz 1974, 1975; Eddy 1981; Pawson & Lockwood 1980; Sleet & Weber 1982), muscle myopathy and renal tubular necrosis (Roberts, McQueen, Shearer & Young 1973; Harthoorn & Young 1974), or secondary bacterial or viral infection (Mazeaud *et al.* 1977). Studies by Beggs, Holeton & Crossman

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(1980) and Wood, Turner & Graham (1983) indicate that the initial osmotic imbalance and high serum lactate levels are not major contributors to delayed mortality in stressed fish. Wood *et al.* (1983) hypothesized that the cause of delayed mortality in exercised trout is the intracellular build up of some unknown anion (probably an organic acid).

Skipjack tuna, *Katsuwonus pelamis* (L.), have been studied in captivity since 1960 at the National Marine Fisheries Service (NMFS) Kewalo Research Facility in Honolulu, Hawaii, USA. Mortalities within hours after capture and transportation of skipjack tuna to the facility were ascribed to skin lesions (trauma) incurred during handling (Nakamura 1972). Although improved handling techniques reduced mortality and live specimens have been routinely kept at the facility (Nakamura 1972), the overall mortality rate for newly captured fish has remained high (>60%). The purpose of this study was to describe this delayed capture mortality syndrome (DCMS) and to determine its cause(s) in recently captured skipjack tuna.

Methods

Capture

The NMFS Kewalo Research Laboratory procedures for capture, transport and maintenance of live tuna were described by Nakamura (1972), and Chang, Brill & Yoshida (1983). The fish were caught aboard commercial 18-m fishing boats using live bait and barbless hooks. Each tuna was pulled free of the water and dropped directly into a 6000-l baitwell with a seawater exchange system. Normally, 10–20 fish were transported for as long as 6 h in the baitwells to the NMFS dock where they were dip-netted into a 2700-l transfer tank. This tank was lifted by a mobile crane and immersed directly in a 7·3-m diameter (42 000 l) holding tank into which the fish swam freely.

Epizootiology

Data on mortalities of 244 skipjack tuna delivered to the Kewalo Research Facility were compiled from records kept by NMFS personnel. The data included date of capture, date of death, length, weight and sex of each fish.

Experimental animals

A total of 30 fish were used in this study from several loads of fish and were sampled immediately after capture (n=6), when the fishing boat arrived at the NMFS dock (1–6 h after capture, n=7), or from the holding tank at approximately 9 (n=6), 24 (n=3), 48 (n=6) and >500 h (n=2) after capture. Due to a general paucity of information concerning 'normal' serum chemistry and haematological values from skipjack tuna, data from the six fish sampled at sea are termed 'basal' as opposed to 'normal'. Likewise, data from the two fish which were in captivity for greater than 500 h are termed 'capture adapted.' Fish were removed from the holding tank using a dip net. The six fish sampled at sea were picked up from the deck of the fishing boat within 5 s of capture.

Clinical observations

Data recorded for each fish included time of capture, arrival time at the Kewalo Research Facility, boat name, total number of fish caught and general condition at the time of sampling. In addition, swimming speed and pattern were observed during each day post-capture.

Haematology and clinical chemistry

Each fish was held firmly in a net and blood was withdrawn from the ventral aorta into a 10 cc syringe with an 18 gauge needle within 10 s of netting. Blood was transferred to three evacuated collecting tubes; containing anticoagulant (EDTA) for haematology, 2 ml chilled 8% perchloric acid, and a plain tube to obtain serum from clotted blood for serum chemistry analyses. The fish was then killed by a blow to the head.

Packed cell volume (PCV), red blood cell count (RBC) and mean red blood cell volume (MCV) were determined by standard methods, with the exception that Herrick's stain (Natt & Herrick 1952) was used for the RBC count. Lactic acid was measured from the protein-free supernatant extracted from the perchloric acid tube as described in *Sigma Technical Bulletin 826 UV*. Serum was extracted from the tube of clotted blood by centrifugation within 24 h of collection and was stored in sealed vials at −10°C. Serum was collected from only three of the six fish examined at sea. Osmolarity was determined by freezing point depression of 0·25 ml of serum using a Fiske* osmometer. All other serum chemistry values (total protein, albumin, globulin, bilirubin, alkaline phosphatase, SGPT, SGOT, LDH, CPK, creatinine, blood urea nitrogen, glucose, cholesterol, triglycerides, uric acid, calcium, iron, sodium, potassium and chloride) were determined by a commercial laboratory using a Technicon Sequential Multiple Analyzer Computer (SMAC) (Alexander & Ingram 1980; Smith & Ramos 1980).

Necropsy

All necropsies were completed within 45 min and most within 15 min of death. Fork length (mm), weight (±3 g) and corrected for blood sample loss, were recorded and compared to a standard weight-length curve (Wt(g)=L(mm)**3·36836/48457) developed for Pacific skipjack tuna (Nakamura & Uchiyama 1966). The presence of any external lesions or parasites were noted. Tissue samples of major organs were preserved in 10% neutral (buffered) formalin. Tissues were processed for routine histology and stained with haematoxylin and eosin.

Results

Epizootiology

Of 244 skipjack tuna delivered to the Kewalo Research Facility, 156 (64%) died, usually on the second and third days after capture (Fig. 1).

*Reference to trade names does not imply endorsement by the National Marine Fisheries Service, NOAA.

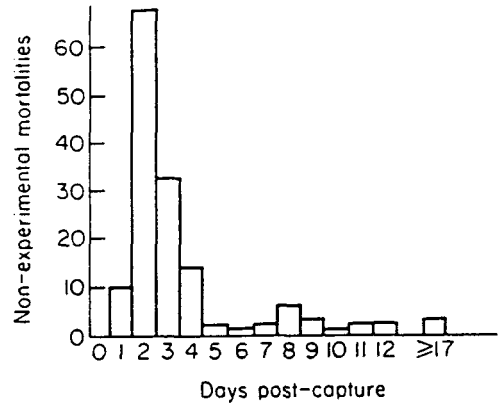

Figure 1. Incidence of mortalities of 244 live-caught skipjack tuna after arrival at the Kewalo Research Facility.

Clinical observations

After transfer to the 24-ft holding tanks the fish usually, but not always, formed a loosely organized school. On the first day after capture, swimming speed, judged by following the schooling fish around the tank perimeter, was greater than that of capture-adapted tuna and the tuna made frequent sharp turns. When not swimming as a school in a circular pattern around the perimeter of the tank, a common swimming pattern was a 'figure eight'. Tuna swimming in a figure eight pattern would do so along 6 m of tank wall shaded from the slanting rays of the sun. The radius of turn at the ends of the figure eight was approximately 1 m. This turn was sharp enough that the fish often lost forward momentum at the apex, necessitating a burst of tail beats to regain cruising speed. Within 2 days, tuna of questionable health were seen swimming very close to the circumferential tank walls, often scraping the sides of their bodies against the vinyl liner of the tank. These fish lacked a visual response to objects in or above the water, tended not to school with other fish in the tank, and were usually found dead within a day of showing the 'wall scraping' behaviour.

By contrast, tuna that adapted to captivity had a more relaxed random swimming pattern and formed a tight, well-coordinated school within the tank. Adapted tuna were constantly changing the frequency of their tail beat by alternating a series of moderate tail beats with short periods of coasting or gliding when tail movement was minimal.

Therefore, the clinical signs associated with postcapture mortality included: (1) increased swimming speed, (2) absence or reduced use of coast and glide swimming behaviour, (3) 'figure eight' swimming patterns, (4) scraping along the sides of the tank, and (5) collapse and death, usually on the second or third day after capture.

Gross lesions

The weight–length ratios of the 4 h (n=7) and 500+ h (n=2) (capture-adapted) fish, were close to the normal weight described by the weight–length equation for skipjack tuna (Nakamura & Uchiyama 1966). The weight–length ratio of fish 24 and 49 h after capture

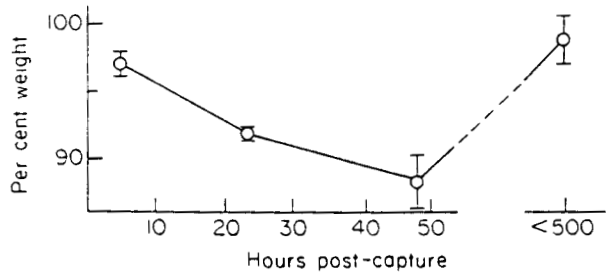

Figure 2. Weight of fish expressed as percentage of 'normal' weight from length–weight regression (mean ± sem).

averaged 92 and 89% of normal indicating a weight loss of approximately 10% (180 g) after being delivered to the holding tanks (Fig. 2).

Gross lesions on fish found dead in the tanks included scrape marks along the flanks and corneal opacity of the eyes. These lesions were probably a result of the 'wall scraping' behaviour described above. Occasionally a tuna was observed with hook injury involving

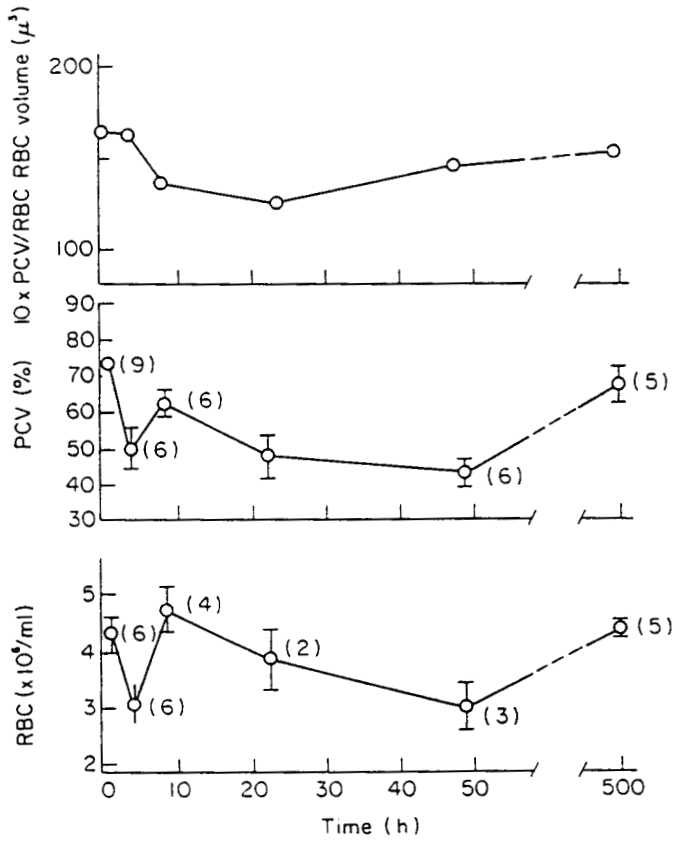

Figure 3. Haematology. Changes in (a) mean red blood cell volume (10×PCV/RBC), (b) packed cell volume (PCV%), and (c) mean red blood cell count (RBC×10⁶/ml), up to 500 h post-capture (all values ± sem).

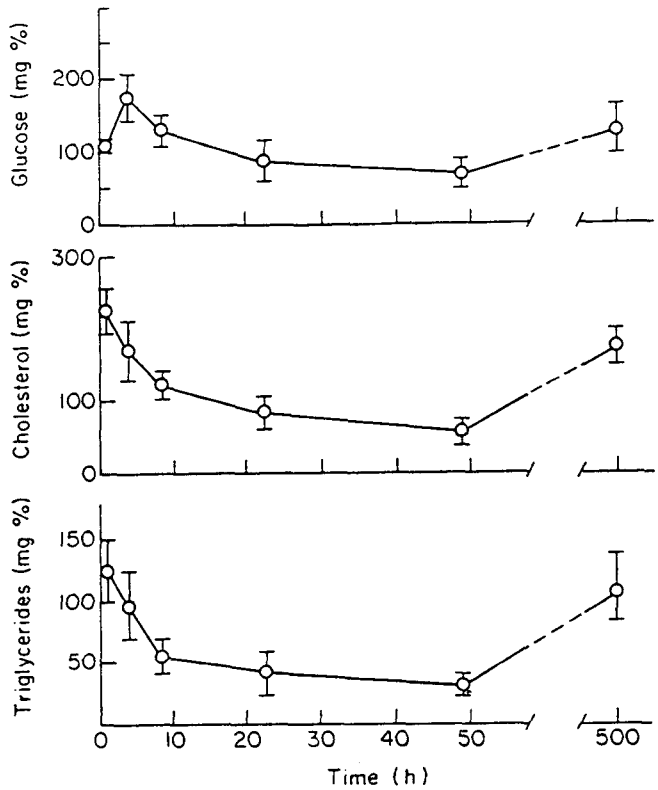

Figure 4. Changes in serum glucose (mg %), cholesterol (mg %), and triglyceride levels (mg %) in fish up to 500 h post-capture (mean ± sem).

tearing or dislocation of bones in the upper jaw; however, the barbless hook usually caused only minor injuries which were difficult to find during postmortem examination.

The upper intestine and lower portions of the stomach of all tuna examined were infected with digenean and nematode parasites.

Haematology

Packed cell volume averaged 74% in tuna sampled at sea, but decreased sharply within 4 h to 46% and returned to near basal levels (63%) at 9 h. Red blood cell count followed the same pattern as PCV but did not decrease proportionally as much as did the PCV, thereby causing an apparent decrease in the mean red blood cell volume (PCV*10/RBC) (Fig. 3). Although not specifically measured, it was noted that the time required for clot formation within the plain blood tubes varied widely between individual fish.

Serum chemistry

Cholesterol, glucose, triglycerides, proteins, calcium, iron and lactic acid were generally lower in fish killed during the first 48 h after capture but were near basal levels in capture-

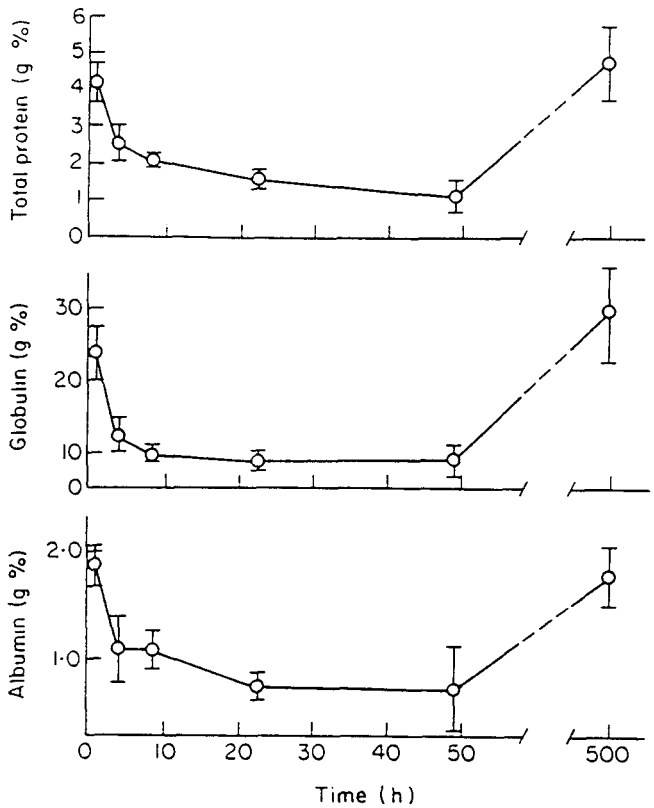

Figure 5. Serum proteins: total protein (g %), globulin (g %), and albumin (g %) in tuna up to 500 h post-capture (mean ± sem).

Table 1. Summary of changes (± sem) in variables associated with delayed capture mortality syndrome (DCMS) in captive skipjack tuna. Basal values are from wild-caught fish at sea. The DCMS values are from fish in captivity for 20–60 h. Capture adapted values are from two fish (except as noted) in captivity for at least 500 h

Parameter	Basal	DCMS	Capture adapted
Weight (% normal) (n=5)	0·970±0·011	0·916±0·010	0·99±0·024
Packed cell value (%) (n=5)	71·9±3·4	44·8±3·3	62±4·5
Red blood cell count (×10⁶/ml)	173±33	135±14	177±4
Total protein (g %)	4·2±0·55	1·7±0·25	3·8, 5·8
Albumin (g %)	1·87±0·18	0·76±0·15	1·5, 2·1
Cholesterol (mg %)	221±35	68±12	150, 201
Triglycerides (mg %)	124±27	34±5·4	79, 139
Osmolarity (mOs)	447±13	360±18	377, 424
Calcium (mg %)	19·8±2·1	12·1±1·4	16·8, 20·3
Iron (mg %)	140±24	30·5±9·6	131, 122
Chloride (meg/l)	171±1·7	185±3·8	160, 165
Lactate (mg %)	117±6	69±15	31, 46
Glucose (mg %)	109±3·3	75±15	164, 97

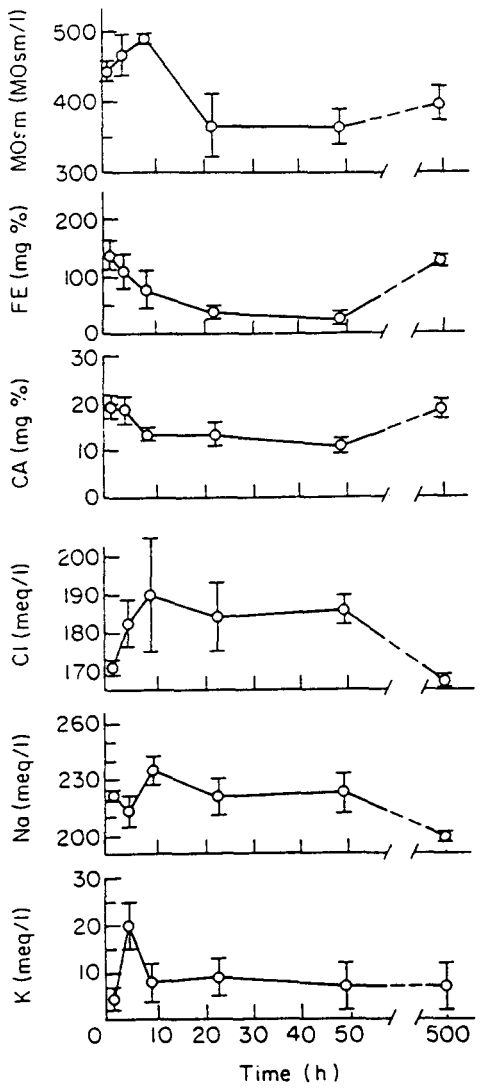

Figure 6. Measure of (a) total serum osmolarity (MOsm/l) and the major serum electrolytes, (b) iron (FE mg %), (c) calcium (CA mg %), (d) chloride (meq/l), (e) sodium (NA meq/l), and potassium (K meq/l) in the serum of tuna up to 500 h post-capture (mean ± sem).

adapted (500+ h) tuna (Figs 4–6 & Table 1). Serum enzymes decreased from initially high levels and remained low in capture-adapted animals (Fig. 7). Only uric acid and plasma chloride levels increased significantly during the first 48 h after capture (Fig. 8 & Table 1). The BUN, creatinine, alkaline phosphatase and bilirubin did not show significant changes (Figs 5–8 & Table 1).

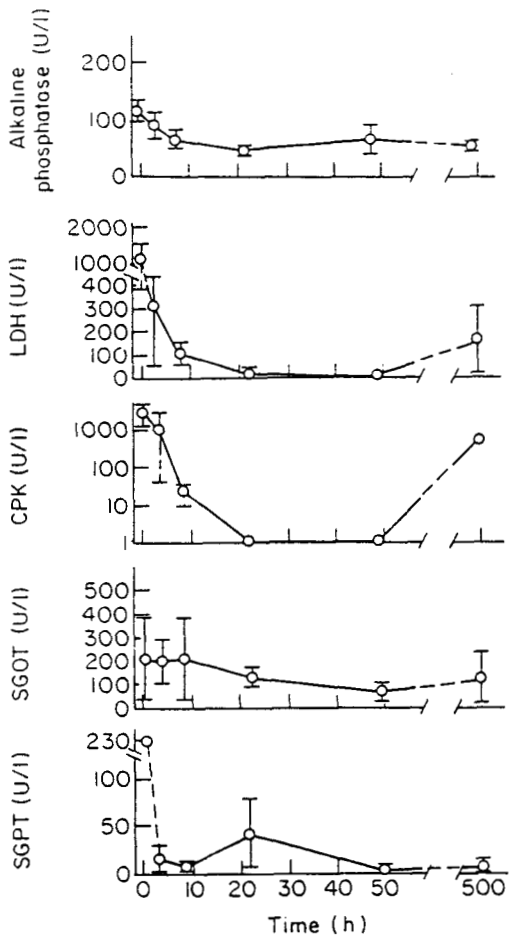

Figure 7. Serum enzyme levels: alkaline phosphatase (U/l), LDH (U/l), CPK (U/l), SGOT (U/l), and SGPT (U/l) (mean ± sem).

Histology

Mild congestion of caudal kidney, liver and other visceral organs was observed in DCMS tuna as compared to basal or capture-adapted fish. Focal acute degeneration of muscle cells was seen in three, two, and one muscle fibres, respectively, in three of six DCMS fish sampled from the 48 h group. No other microscopic morpho-anatomical differences were found between DCMS tuna and basal or capture-adapted tunas.

Discussion

The primary response exhibited by the skipjack tuna to capture and handling was similar to the stress response described for other marine fish. There were transient increases in blood

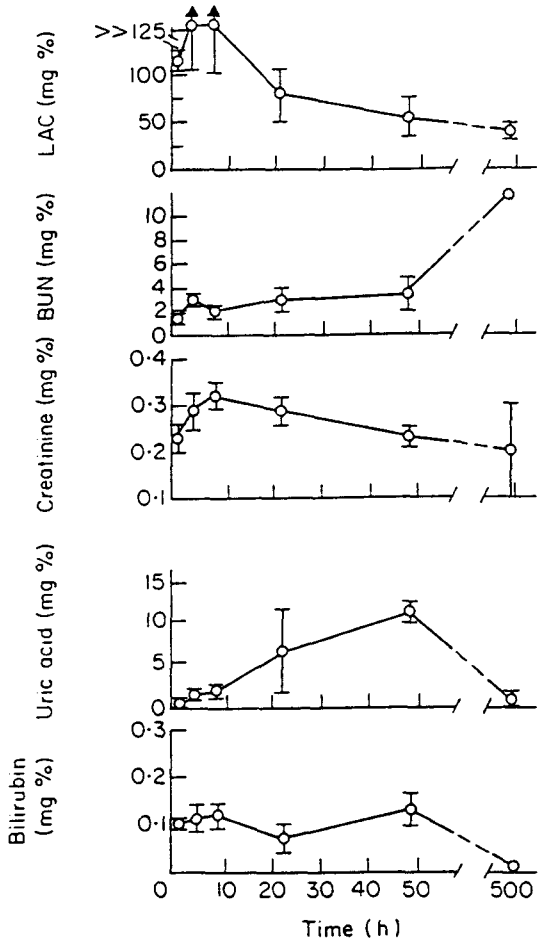

Figure 8. Metabolic waste products: (a) Serum lactate (LAC mg %), (b) blood urea nitrogen (BUN mg %), (c) creatinine (mg %), (d) uric acid (mg %), (d) bilirubin (mg %), were measured up to 500 h post-capture (mean ± sem).

glucose and lactate levels after capture. There was a transient increase in serum osmolarity due primarily to an increase in the major blood ions, chloride, lactate and potassium. There was an approximate 10% weight loss from fish held in captivity for 9–49 h (Fig. 3). The majority of mortalities occurred 24–48 h after capture.

It is unlikely that the observed mortalities were caused by simple trauma. There were no obvious scrape marks or hook wounds on the fish used in this study which would have debilitated the fish. Observations by R. W. Brill (Southwest Fisheries Center Honolulu Laboratory, NMFS, Honolulu) and R. Kearney (South Pacific Tuna Commission, Noumea, New Caledonia, personal communication) at the Kewalo Research Facility indicate that skipjack tuna adapted to captivity have a very low mortality rate as a result of recapture by hook, tagging and release back into their holding tank.

The evidence does not support the hypothesis that death was due to either anoxia, or bacterial or viral infection. The high incidence of mortalities after 24–48 h in captivity occurred too late to be caused by anoxia and too soon to be caused by infection. Histological examination failed to find evidence supporting infection as a cause of death. Similarly, death caused by lactic acidosis cannot be established. Even though the blood levels of lactate reached extremely high concentrations approximately 4–8 h after capture, the deaths did not occur until 48 h after capture, at which time the blood lactate levels were significantly reduced.

Signs usually associated with capture myopathy, including weakness, impaired or noncognizant vision, incoordination, and death following severe physical stress (Harthoorn & Young 1974), were all apparent in captured skipjack tuna. However, clinical pathologic abnormalities, such as an elevation in circulating enzymes (SGOT, CPK, LAD, SGPT; Fig. 7) indicative of muscle sarcomere damage and kidney lesions often associated with capture myopathy in mammals, were not found in these fish during the period of mortality. Therefore, the data do not support the hypothesis that the fish died from capture myopathy.

Also, disseminated intravascular coagulation (DIC) does not appear to be the cause of death. Although some pooling or intravascular coagulation of blood may have occurred (as evidenced by the 'congestion' noted during histological examination), there was no evidence of haemorrhage, blood clots, or the presence of distorted or damaged red blood cells which are indicative of DIC. Further, although DIC has been suggested as a possible cause of death (Smith 1980), it has never been demonstrated in fish.

Osmotic dysfunction did occur during the first 8 h after capture. Sodium, chloride, potassium, iron and calcium normally constitute the major contributors to serum osmolarity (MOsm/l), but other molecules such as serum proteins or lactate ions may contribute significantly if they occur in abnormally high concentrations. The transient increase in serum osmolarity (Fig. 6) appears to be due primarily to the increase in chloride, lactate and potassium. Increased corticosteroid levels effect active ion transport across the gills and decrease water uptake through the intestinal epithelium (Pic *et al.* 1975; Eddy 1981), possibly causing an increased serum ion level accompanied by dehydration. An increased drinking rate and subsequent absorption of ions and water across the gastroesophageal membrane, as described by Sleet & Weber (1982) for buffalo sculpin, would presumably lead to increased serum ion levels accompanied by hydration. The apparent 10% weight loss in fish from the 23- and 49-h groups suggests that the tuna were losing water and gaining salts across the gills without adequate replenishment by swallowing water. The high serum osmolarity and ion levels were only transitory. Concentrations at or below basal levels were observed within 24 h of capture, a full 24 h before the majority of mortalities occurred.

Haemodilution could explain the almost equivalent drop in total protein, globulin, albumin, triglycerides and cholesterol, all of which decreased 58–63% from basal levels within 24 h of capture. Calcium decreased 30% and iron 71% in the same period. The parallel decreases in these values make it likely that the cause was dilution. The PCV, often used as an indicator of haemodilution in mammals, decreased 'only' 36% during the first 24 h. However, release of erythrocytes from the spleen and kidneys could account for the discrepancy between the percentage dilution suggested by PCV decrease (36%) and the decrease suggested by other serum chemistry values (60%).

The blood volume of fish may not be as stable as that of land vertebrates. Alexander &

Ingram (1980) noted that the PCV of an immobilized albacore decreased during the first 12 h but forwarded no hypothesis as to the reason for this decrease. Harbell, Hodgins & Schiewe (1979) noted a decrease in PCV, total protein, albumin, globulin, chloride, sodium, total osmolarity and alkaline phosphatase levels, and the presence of a distended gut filled with fluid in juvenile freshwater salmon experimentally infected with vibriosis. The infected fish gained weight and the control fish actually lost weight during the experiment. They ascribed the low plasma protein and osmolarity to kidney failure. Osmolarity changes and haemodilution similar to those found in this study have been observed in anaesthesized buffalo sculpin (Sleet & Weber 1982). These authors attribute the serum dilution to factors other than fluid uptake across the upper gastrointestinal tract.

The source of the fluid that increases the blood volume is unknown. It is tempting to attribute the rapid rise in blood osmolarity and increased volume to the fish swallowing and absorbing water and salts from the gut faster than the ion-exchange mechanism in the gills can excrete the added salt load. Assuming an average normal blood volume of 6% in a 1·8 kg skipjack tuna, approximately 130 ml of diluting fluid would be required to dilute the blood by 60%. However, the addition of only 1·5 ml of sea water ([Cl]=1900 mg/dl) would be adequate to account for the observed 11% increase in the serum chloride concentration, but considering the apparent weight loss of 10% during the first 48 h in captivity and the results of Sleet & Weber, it seems more plausible that the diluting fluid came at least partially from the more isotonic inter- or intracellular tissue fluids and was then lost to the surrounding sea water by osmosis.

Although the initial function of haemodilution could be to maintain blood pressure and adequate blood flow, thereby keeping the fish alive, excessive haemodilution could cause death due to several factors including: (1) inability to control acid-base balance due to low serum proteins, (2) decrease in the oxygen and metabolite carrying capacity of the blood, or (3) impairment of metabolite transfer through tissue membranes. The authors hypothesize that delayed capture mortality syndrome (DCMS) is caused by the combined effects of several factors including haemodilution, tissue dehydration and excessive fluid weight loss. Further experiments are needed to test this hypothesis; for instance, measurement of the internal water and electrolyte balances in the different body compartments and in the environment.

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