

Characterization of the Physiological Stress Response in Lingcod

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Abstract.—The goal of this study was to describe the duration and magnitude of the physiological stress response in lingcod *Ophiodon elongatus* after exposure to brief handling and sublethal air stressors. The response to these stressors was determined during a 24-h recovery period by measuring concentrations of plasma cortisol, lactate, glucose, sodium, and potassium. Lingcod were subjected to brief handling followed by either a 15-min or a 45-min air stressor in the laboratory. After the 15-min stressor, an increase in cortisol or glucose could not be detected until after 5 min of recovery. Peak concentrations were measured after 30 min for cortisol and after 60 min for glucose and lactate. Glucose and lactate had returned to basal levels after 12 h, whereas cortisol did not return to basal levels until after 24 h of recovery. Immediately following a 45-min air stressor, all measured parameters were significantly elevated over levels in prestressor control fish. Cortisol concentrations tended to increase and reached a measured peak after 8 h of recovery, whereas glucose and lactate reached a measured peak after 1 h of recovery. Cortisol and lactate returned to basal levels within 24 h. Glucose, however, remained elevated even after 24 h of recovery. Plasma ions initially increased during the first hour of recovery, and the concentrations then declined to a level below that measured in control fish for the remainder of the 24-h recovery period. In addition, we evaluated the effect of fish size on the stress response. There was no significant difference between the stress response of smaller (41–49-cm [total length]) and larger (50–67-cm) lingcod after 45 min air exposure. In general, both the magnitude and duration of the primary and secondary stress responses in lingcod are comparable to those of salmonids.

For a variety of reasons, catches of both target and nontarget species are commonly discarded at sea (bycatch). Despite increased management efforts to reduce the proportion of discarded bycatch by implementation of modified fishing practices and gear (Kennelly 1999; Broadhurst 2000), the problem of

undetected mortality and the sublethal effects of stress resulting from capture and discard still remain (Davis 2002).

On the West Coasts of the United States and Canada, lingcod *Ophiodon elongatus* are large, carnivorous, rocky reef fish (Miller and Lea 1972; Miller and Geibel 1973) belonging to the species-depauperate family Hexagrammidae. These fishes are related to rockfishes (family Scorpaenidae) and sculpins (family Cottidae; Greenwood et al. 1966). Lingcod are caught by recreational fishers and in commercial trawl and

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hook-line fisheries, as well as being part of the bycatch for other fisheries. Reduced catch and a general decline in stock size led to concerns about the conservation of lingcod, culminating in the species being designated as overfished along the West Coast of the United States by the Pacific Fishery Management Council in 1999. Consequently, harvest limitations (minimum size and trip limits) were imposed on both the lingcod fishery and on fisheries that catch lingcod as bycatch. These actions, while limiting lingcod bycatch, have increased the proportion of smaller lingcod that are caught and returned to the ocean (Davis and Olla 2002). Despite the ecological and economical importance of lingcod as a component of both commercial and recreational fisheries, little is known about their physiology during capture and discard.

Mortality rates of fish that are hooked, landed, handled, and discarded can range from 0% to 88% (see Muoneke and Childress 1994 for review). Most studies have been carried out on freshwater fishes; to date, there have been few studies carried out to assess the effect of capture and release on lingcod survival and stress-related impacts. Recently, Davis and Olla (2002) showed that several practices involved in the capture and release of lingcod result in increased levels of stress indices and mortality. Furthermore, smaller fish tended to experience greater mortality than larger fish. Given the relative ease of taking physiological measures at sea, this may be a useful tool for determining the potential mortality associated with bycatch. However, the correlation between physiological measures of stress and behavioral impairment or delayed mortality is unclear, as studies have shown either a clear linkage (Schreck et al. 1997; Olla et al. 1998) or no link between stress and behavior or survival (Olla et al. 1992; Davis 2002).

It is well known that most teleosts studied to date show a classical physiological stress response after exposure to stressors, such as handling or crowding, and cortisol peaking within an hour or so, followed by secondary and tertiary stress responses (see Donaldson 1981; Schreck 1981; Wedemeyer and McLeay 1981). However, some teleosts have relatively slow cortisol responses to stress (Schreck 1981, 2000).

Given the unique phylogenetic status of lingcod and the fact that an understanding of its stress physiology could ultimately have management implications relative to understanding fitness of bycatch discard, we describe the physiological stress response of lingcod subjected to sublethal air stressors in the laboratory. In an effort to elucidate the potential link between stress physiology and stress-induced mortality in different-sized fish, we compare the physiological stress response of two size-classes of lingcod (smaller, 41–49

cm total length [TL]; larger, 50–67 cm TL) to an air stressor.

The primary response was assessed by measurement of plasma cortisol levels, while the secondary response was profiled by means of metabolic indicators, such as plasma glucose and lactate concentrations. Plasma sodium and potassium concentrations were also measured during the recovery period to provide an indication of ionoregulatory ability during the stress recovery.

Methods

Experiment 1. Stress Response and Recovery Time Course of Yearling Lingcod Following a 15-Min Air Stressor

Experimental fish.—Yearling lingcod (26.7 ± 0.33 cm TL, 133.7 ± 6.12 g) were obtained from National Oceanic and Atmospheric Administration (NOAA) Fisheries, Manchester, Washington, in January 2001. The fish were transferred to Hatfield Marine Science Center, Oregon State University, Newport, Oregon, and maintained for 63 d in 0.6-m-diameter, 1.0-m-deep tanks supplied with flow-through seawater (10 L/min; 7.0–8.0°C; 30–32 g/L salinity; $O_2 > 90\%$ saturation) at a density of 20 fish/tank. The fish were held under a photoperiod of 12 h light and 12 h dark in low-light conditions (daylight fluorescent, 5,000 K) at $0.5 \mu\text{M}$ photons $\cdot\text{m}^{-2}\cdot\text{s}^{-1}$. Fish were fed frozen Pacific herring *Clupea pallasii* ad libitum twice per week.

Experimental design.—In March 2001, yearling lingcod (21–30 cm TL) were transferred to six circular tanks (0.6 m diameter) and held at a density of seven fish/tank for 2 weeks before the experiment to allow for acclimation. Tanks were furnished with several short lengths of polyvinylchloride pipe to provide an opportunity for the lingcod to isolate themselves from one another in crevice-like locations. On the day of the experiment, one fish from each tank ($n = 6$) was rapidly (< 2 min) netted, and a mixed arteriovenous blood sample was taken (see below for method) to establish prestress (control) levels of the selected indices. Control fish were subsequently placed in a separate recovery tank. The remaining six fish from each tank were netted and placed in a plastic bucket covered with a perforated lid and held for 15 min in air at 14°C, conditions that in preliminary studies induced a nonlethal stress response. Fish were then returned to their original tank for recovery (six tanks, six fish/tank). Three fish from each tank were sampled for blood at one of six randomly assigned early-recovery time points (0, 5, 10, 20, 30, 40 min of recovery) and the remaining three fish from each tank were sampled at later-recovery time points (1, 2, 4, 8, 12, 24 h of recovery).

The assignment of each group of three fish to a sampling point was random. Total time from initial netting to blood sampling to being held on ice for all three fish was less than 5 min.

Experiment 2. Comparison of the Stress Response of Large and Small Lingcod to a 45-Min Air Stressor

Experimental fish.—Lingcod (41–67 cm TL) were captured in May 2001 by trawling with a commercial bottom trawl in the Pacific Ocean off Depoe Bay, Oregon (Davis and Olla 2002). Fish were transported back to the laboratory within 4 h. At this time, fish were separated based on two size-classes that were evident in fish captured by trawl: smaller (41–49 cm TL) and larger (50–67 cm TL), and 20 fish/tank were held in four 15,904-L circular tanks (4.5 m diameter, 1.0 m depth) supplied with flow-through seawater (20 L/min; 7.0–8.0°C; 30–32 g/L salinity; $O_2 > 90\%$ saturation). Fish were initially fed every other day with one live surf smelt *Hypomesus pretiosus* per fish until all fish had begun feeding and later fed frozen Pacific herring ad libitum twice per week. Feeding began within 8 d of capture and experiments were begun 14 d after fish had been brought into the laboratory. No significant mean growth was measured during the 2 weeks.

Experimental design.—In June 2001, five of the larger lingcod (50–67 cm TL) were removed from their holding tanks and a mixed arteriovenous blood sample was taken to establish prestress (control) levels of the selected indices. At this time, an additional 25 of the larger lingcod (50–67 cm TL) were transferred to rectangular tanks (90 × 60 × 30 cm) without water (five tanks, five fish/tank) and held for 45 min in air at 16.0°C, a time typical of sorting in trawling operations with larger catches (Davis and Olla 2002; Parker et al. 2003). Immediately after the air stressor, all five fish from one tank were sampled (recovery time = 0 h). The remaining fish were returned to three 140-L circular tanks (2.0 m diameter, 0.8 m depth; four tanks, 5 fish/tank) and all five fish from an individual tank were sampled after 1, 4, 8, or 24 h of recovery. The experiment was repeated on the same day for smaller lingcod (41–49 cm TL).

Sampling and analysis.—To obtain plasma, fish were rapidly netted from the tank, taking care to minimize disturbance to the netted fish or to those remaining in the tank. Fish were immediately immersed in an anesthetic dose (400 mg/L) of tricaine methanesulfonate (MS-222) buffered with sodium bicarbonate. Lingcod were completely anesthetized in 2.5–3.0 min. Fish were serially removed from the anesthetic solution and a mixed arteriovenous blood sample was obtained from the caudal peduncle with a heparinized Vacutainer and placed on ice. Total time

from initial netting to blood sampling to being held on ice was between 3 and 6 min for five fish. Plasma was separated by centrifugation and stored at –80°C until analysis for cortisol, glucose, lactate, and Na^+/K^+ (Na^+/K^+ for experiment 2 only). Sampled fish were returned to another tank for recovery.

Plasma cortisol was assayed by 3H radioimmunoassay as described by Foster and Dunn (1974) and modified by Redding et al. (1984). Plasma glucose levels were determined by colorimetric procedure according to Wedemeyer and Yasutake (1977). Plasma lactate levels were assayed by fluorometry (Passonneau 1974), and plasma sodium and potassium ion concentrations were determined with a Nova sodium–potassium analyzer (Nova Biomedical Corporation, Waltham, Massachusetts).

Statistical analysis.—No statistical analyses were carried out for experiment 1; the study was designed to be a preliminary range-finding experiment and hence is descriptive in nature, utilizing the number of fish available to us by maximizing the number of time points considered.

In experiment 2, data were transformed to logarithmic values when variances were heterogeneous (Bartlett's test). Two-way analysis of variance tests were performed to compare concentrations between time points and between sizes of fish. As there were no differences between the response of small and large fish, the data were combined for discussion in the text. A significance level of $P = 0.05$ was used for all statistical tests. All data are graphed as means ± 1 SE (untransformed data).

Results

Experiment 1. Stress Response and Recovery Time Course of Yearling Lingcod Following a 15-Min Air Stressor

No mortality resulted from the 15-min air exposure. Basal plasma cortisol levels in prestress control fish were less than 11.0 ng/mL in four of the six fish (Figure 1). However, two control fish had cortisol levels of ~185 ng/mL. Levels were not elevated immediately after the cessation of the stressor; however, after 5 min of recovery, plasma cortisol concentrations rose to 118.0 ± 20.6 ng/mL (mean ± 1 SE). Plasma cortisol levels continued to increase steadily, reaching a measured peak after 30 min of recovery (262.9 ± 13.1 ng/mL) and remained over 200 ng/mL for 2 h. Concentrations tended to decline steadily thereafter and were similar to prestress levels after 24 h of recovery.

Mean plasma glucose concentrations were not elevated over controls until 5 min of recovery (Figure 1), at which time they were approximately twice the

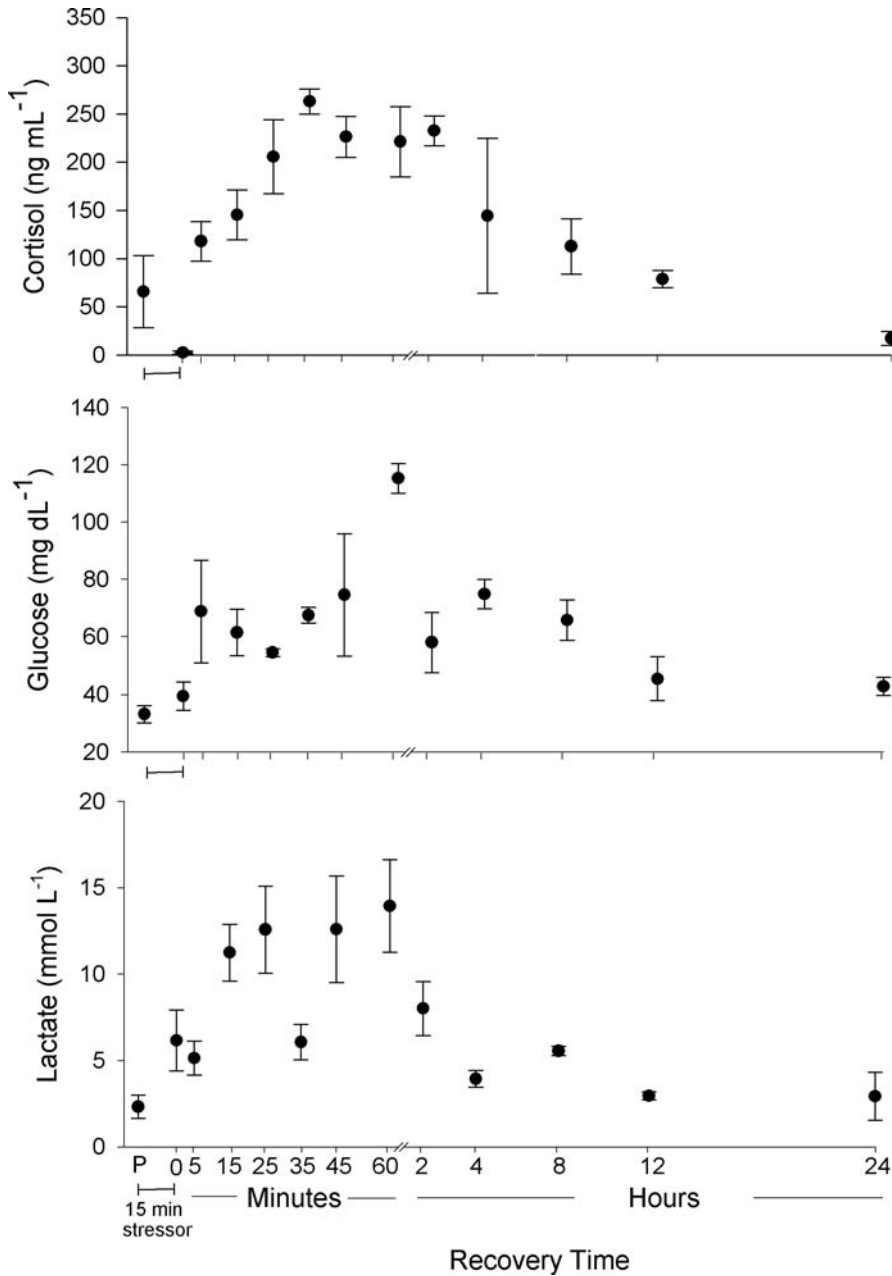


FIGURE 1.—Concentration of plasma components (cortisol, glucose, and lactate) for lingcod (21–30 cm TL). Fish were stressed in the laboratory for 15 min in air and allowed to recover for a period of 24 h (control: $n = 6$; recovery: $n = 3$). Values are means \pm SEs.

level of controls (68.9 ± 17.8 mg/dL). Glucose levels reached a measured peak (115.3 ± 5.2 mg/dL) after 1 h of recovery. Plasma glucose tended to decline thereafter, returning to basal levels after 12 h of recovery.

Immediately after air exposure, mean plasma lactate concentrations had doubled relative to those of pre-

stress control fish (6.1 ± 1.8 mmol/L; Figure 1). Plasma lactate tended to increase during the first hour of recovery, reaching a measured peak of 13.8 ± 2.7 mmol/L at 1 h and declining steadily thereafter, so that by 12 h recovery levels were similar to those in sampled prestress control fish.

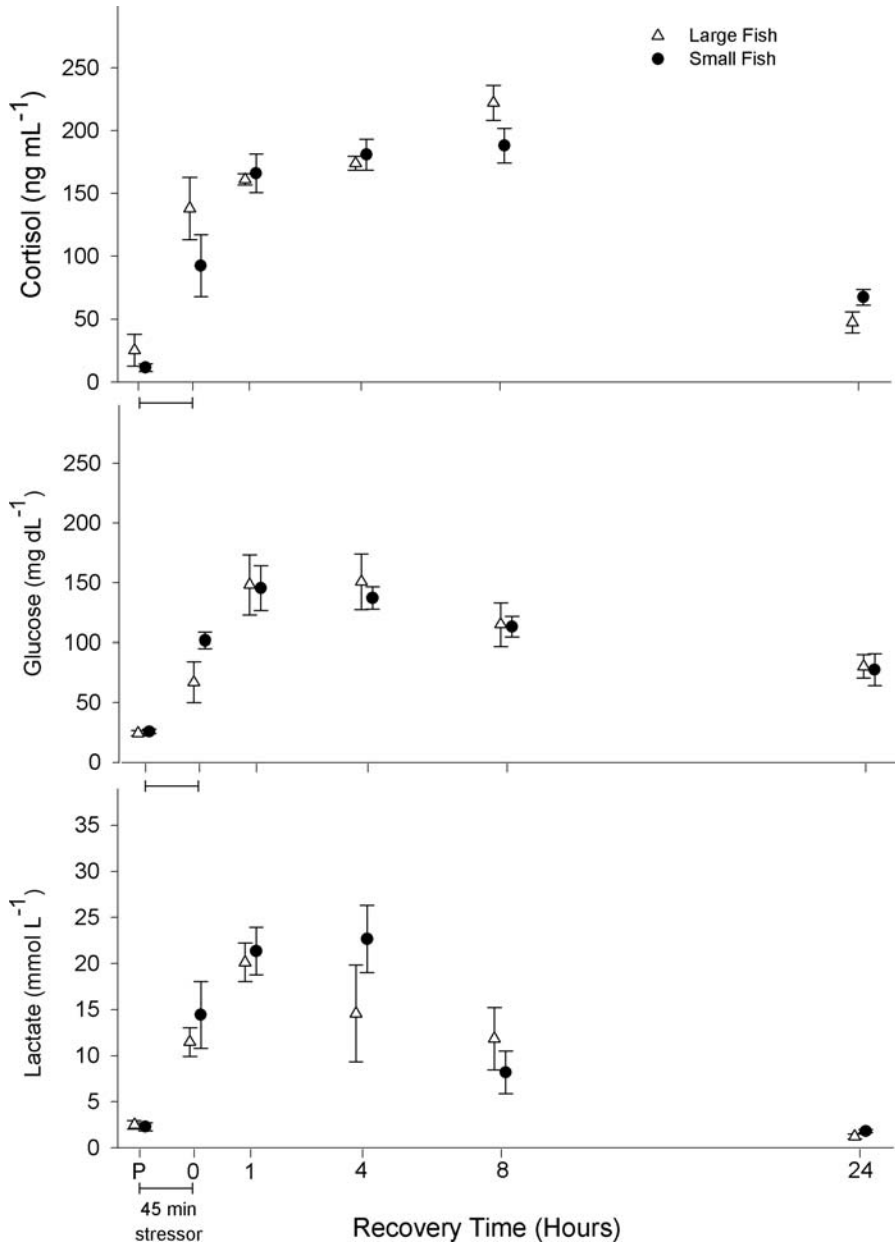


FIGURE 2.—Concentration of plasma components (cortisol, glucose, and lactate) for large (50–67 cm TL; $n = 5$) and small (41–49 cm TL; $n = 5$) lingcod. Fish were stressed in the laboratory for 45 min in air and allowed to recover for a period of 24 h. Values are means \pm SEs.

Experiment 2. Comparison of the Stress Response of Larger and Smaller Lingcod to a 45-Min Air Stressor

No mortality resulted from 45-min air exposure and there were no significant differences between the responses of larger and smaller fish for any of the parameters measured. Plasma parameters increased

rapidly after stress, reaching peak values and then decreasing by 24 h. Data are presented separately for smaller and larger fish (Figures 2, 3). Values and statistics are reported in the text for small and large fish combined.

Prestress levels of cortisol ranged between 1.0 and 58.2 ng/mL, with an overall mean concentration of

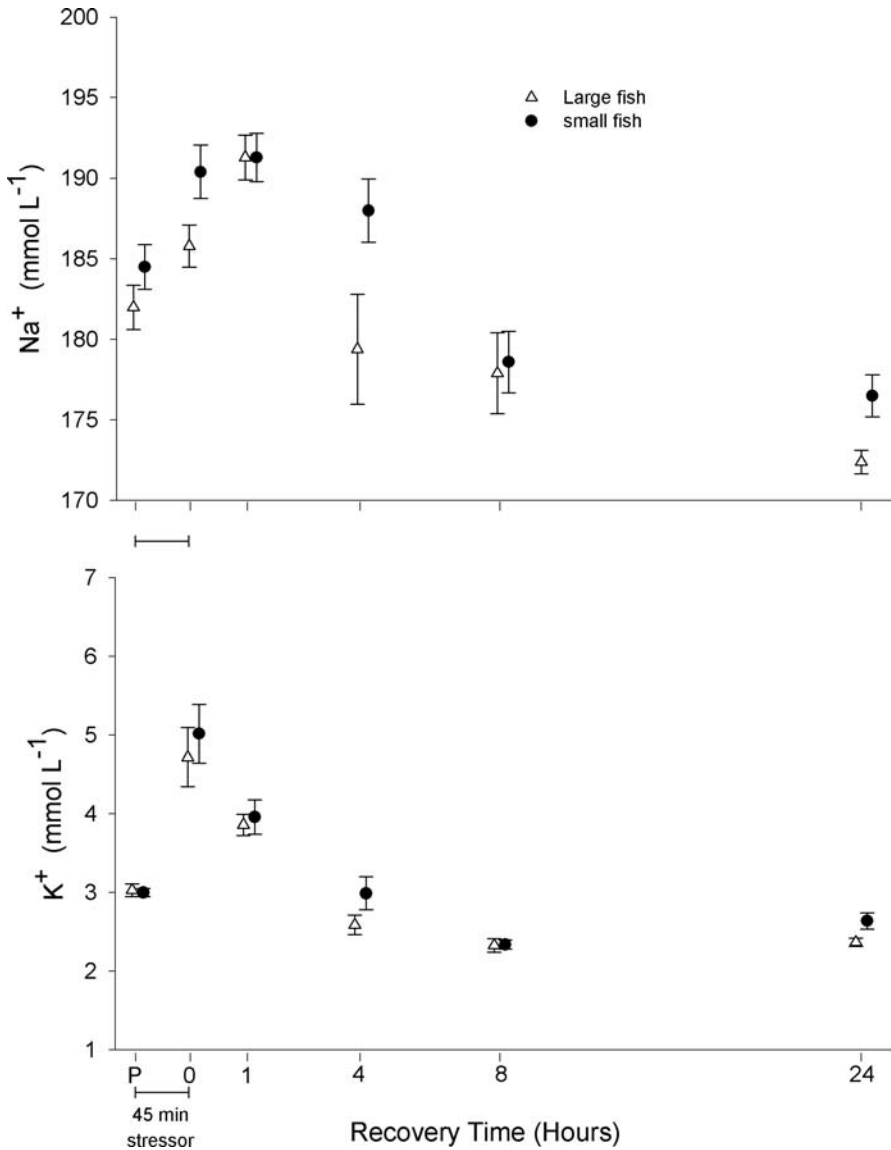


FIGURE 3.—Concentration of sodium and potassium ions for large (50–67 cm TL; $n = 5$) and small (41–49 cm TL; $n = 5$) lingcod. Fish were stressed in the laboratory for 45 min in air and allowed to recover for a period of 24 h. Values are means \pm SEs.

18.5 \pm 6.6 ng/mL (Figure 2). Mean plasma cortisol was significantly elevated immediately after air exposure (115.5 \pm 18.1 ng/mL; $P < 0.001$) compared with prestress controls. Concentrations tended to increase, reaching a measured peak of 205.3 \pm 10.9 ng/mL after 8 h of recovery. After 24 h of recovery, the mean plasma cortisol concentration was not significantly higher than levels in prestress fish.

Mean plasma glucose concentration immediately after air exposure was significantly elevated (84.6 \pm

10.4 mg/dL; $P < 0.01$) over that of controls (25.1 \pm 1.4 mg/dL; Figure 2) and reached a measured peak after 1 h of recovery (147.0 \pm 14.8 mg/dL). After 24 h of recovery, mean glucose concentration remained three times the level of prestress controls (79.1 \pm 7.8 mg/dL; $P < 0.01$).

The mean plasma lactate level immediately after air exposure was significantly elevated (12.8 \pm 1.9 mmol/L; $P < 0.01$) over that of controls (2.4 \pm 0.3 mmol/L; Figure 2) and reached a measured peak by 1 h of

recovery (20.5 ± 1.6 mmol/L). Plasma lactate concentration returned to prestress levels after 24 h of recovery.

The mean plasma sodium concentration after exposure to air initially increased significantly (183.2 ± 1.1 mmol/L; $P < 0.01$) over that of controls until reaching a measured peak by 1 h of recovery (191.3 ± 1.0 mmol/L; $P < 0.001$; Figure 3). Subsequently, mean levels fell below that measured on control fish and were significantly lower after 24 h recovery (174.5 ± 1.0 mmol/L; $P < 0.001$).

The mean plasma potassium concentration increased significantly over that of controls (3.03 ± 0.04 mmol/L) to a measured peak immediately after air exposure (4.9 ± 0.3 mmol/L; $P < 0.001$; Figure 3). The mean concentration then tended to decline to below prestress levels during the remainder of the recovery period.

Discussion

The physiological stress response that we have described for lingcod in this study generally follows the dynamics common for many fishes that have been studied thus far (Schreck et al. 1997). After handling and air exposure, the initiation of the response occurred within 0–5 min depending on the duration of the stressor, and peak levels of plasma cortisol, glucose, and lactate were reached within 1–2 h. Concentrations of these indices reached a plateau between 4 and 8 h and recovery was observed within 24 h (with the exception of glucose after the 45-min air exposure). Plasma ions (Na^+ , K^+) peaked within about 1 h, but then decreased and remained below basal levels even after 24 h of recovery.

In this study, we noted a substantial amount of variability in both basal levels and the magnitude of response and duration of recovery between individuals. The variable basal levels may be indicative of an inherent variability in the response of this species to the stress of holding conditions. The individual variability of response should be borne in mind if physiological parameters are to be used as an indicator of stress in lingcod in the laboratory or field. In December 2001, a further experiment was carried out in our laboratory designed to assess the combined effect of a 15-min air stressor and exposure to a pathogen of the *Vibrio* spp. on lingcod mortality (unpublished data). When fish from the same source as experiment 1 (with and without pathogen) were subjected to an identical 15-min air stressor ($n = 12$), similar concentrations and variability in both basal cortisol (19.6 ± 7.7 ng/mL) and poststressor cortisol (58.9 ± 17.8 ng/mL) were observed. No mortality was observed in any of the treatments up to 6 weeks poststress.

Plasma cortisol levels were not elevated until 5 min

after the cessation of the 15-min air stressor in experiment 1. This suggests that lingcod may have an extended latency of response when compared with many other species, including salmonids (see Donaldson 1981). After the air-stressor cortisol levels reached a maximum plateau of around 200 ng/mL, which is similar to that observed in many other marine and freshwater species, such as sea raven *Hemitripterus americanus* (Vijayan and Moon 1994), chinook salmon *Oncorhynchus tshawytscha* (Barton et al. 1986), hatchery-reared and wild rainbow trout *O. mykiss* (Barton et al. 1985; Clements et al. 2002), and sablefish *Anoplopoma fimbria* (Olla et al. 1998; Davis et al. 2001), but higher than those observed in wild winter flounder *Pseudopleuronectes americanus* (Barnett and Pankhurst 1998) and red porgy *Pagrus pagrus* (Rotllant et al. 2003) after single, relatively short-duration stressors. Similarly, Parker et al. (2003) observed that cortisol levels in lingcod that were held on deck between 35 and 300 min reached a maximum of approximately 200 ng/mL, regardless of the length of air exposure. It is also interesting to note that the magnitude of the cortisol response in this study was similar for fish from both experiments, but the metabolic response differed in intensity. This difference is probably related to the duration of the stressor. The production of cortisol during the stress response is thought to be, in part, attributable to the perception of the stressor by the fish, which may be similar whether the animal spends 15 or 45 min in air. However, our data suggest that a longer exposure to air results in a higher magnitude of the energetic response, as evidenced by the greater elevation in glucose and lactate after 45 min in air. Both plasma glucose and lactate tended to plateau at higher concentrations after 45 min in air when compared with 15 min in air. Both the magnitude and duration of the secondary stress response (lactate and glucose) of fish in the current study is generally within the range of values reported previously in studies carried out on salmonids (Black et al. 1962; Schreck et al. 1976; Jones and Randall 1978; Turner et al. 1983; Wood et al. 1983, 1990), striped bass *Morone saxatilis* held in both fresh and salt water (Cech et al. 1996), and sablefish (Davis et al. 2001), but again higher than values reported for starry flounder *Platichthys stellatus* (Milligan and Wood 1987; Pagnotta and Milligan 1991), the exception being the high levels of plasma lactate measured after the 45-min air exposure. Concentrations were almost double that in experiment 1 and are higher than those previously published for marine and freshwater fishes. This is probably due to an extended period of anaerobic metabolism during air exposure rather than as a result of increased activity associated with capture. The initial

behavioral response of the lingcod to capture was avoidance, which was characterized by highly intense burst swimming; however, very shortly after capture the fish lay motionless for the remaining duration of the air exposure. Similarly, in experiment 2, plasma glucose peaked at a higher level and remained elevated even after 24 h of recovery. This, again, is probably a result of the increased demand for energy substrate during the longer period of air exposure. The magnitude and duration of the hyperglycaemia can vary according to the nature and severity of the stressor and is also strongly dependent on the strain and nutritional status of the fish (Nakano and Tomlinson 1967; Wydoski et al. 1976), environmental temperature (Davis and Parker 1983), and size (Wydoski et al. 1976), although in the current study we saw no effect of size on the hyperglycaemia response. Longer recovery times have been reported previously by Pickering and Pottinger (1987), who showed a return to resting concentrations in brown trout *Salmo trutta* 72 h after an acute handling stressor. The increased mobilization of energy from muscle and liver into the blood required to maintain anaerobic metabolism may have long-term effects on fitness. This may be important when considering time on deck before discarding fish.

Plasma sodium and potassium ions were elevated over controls after 1 h of recovery following 45 min in air. Concentrations began to decline subsequently, but did not even out at prestress levels. Instead, the fish overcompensated, so that after 24 h, levels were below prestress concentrations. The majority of studies in which sodium and potassium are measured have been conducted on freshwater fishes, although it is well established that marine fishes generally respond to stress with an increase in the levels of plasma ions (Eddy 1981; Mauzeaud and Mauzeaud 1981; Redding and Schreck 1983; Van Anholt et al. 2004; Davis and Schreck 2005) similar to that observed in this study. Both cortisol and catecholamines affect ionoregulation at the gills, intestines, and urinary bladder. By acting on Na^+, K^+ -ATPase (3.6.1.36; IUBMB 1992) and, hence, the active transport of ions across chloride cells, catecholamines influence the net transport of ions and the permeability of the fish to water. Van Anholt et al. (2004) have also reported that plasma sodium tended to increase immediately after exposure to a stressor in gilthead seabream (also known as gilthead bream) *Sparus auratus* and, thereafter, decrease below resting levels for over 24 h. The authors consider this effect to be caused by an increase in activity of Na^+, K^+ -ATPase. In contrast, they found that levels of plasma potassium declined steadily after the stress and attributed this to an enhanced uptake of K^+ into the red

blood cells as a reaction to low oxygen. It is not clear why lingcod in this study overcompensated, but the combined effect of high plasma ions together with an extended period of low plasma osmotic pressure could have long-term deleterious effects on the fish.

We found no difference in the stress response of small fish and large fish in our experiment. Previously, Davis and Olla (2002) and Parker et al. (2003) reported that smaller lingcod tended to experience greater mortality than larger fish when subjected to several capture-related stressors. Although plasma concentrations of metabolic indices were the same for small and large fish in our study, the energetic cost would probably be significantly greater for the small fish, given they have fewer energy reserves and a higher metabolic rate.

This study clearly shows that short-term acute stressors cause a physiological stress response in lingcod that is similar in both magnitude and intensity to the classical response demonstrated for many species. However, we observed a high degree of variability between the responses of individual lingcod to the stressors. For this reason, we suggest that the use of plasma measures as a management tool to predict the fate of individual fish be employed with caution. Although the link between plasma indicators of stress and survival is poorly understood (Olla et al. 1992, 1998; Schreck et al. 1997; Davis 2002; Davis and Schreck 2005), previous research has shown that the cumulative effects of sublethal stressors lead to a reduced ability to cope with the subsequent stressors (Barton et al. 1986; Sigismondi and Weber 1988), reduced recruitment to successive life stages (Adams et al. 1985), or death, even though the response to a single stressor does not exceed the fish's physiological limits (Donaldson 1981; Carmichael 1984; Barton et al. 1986). Our data show that, in isolation, air exposure results in a physiological stress response that does not lead to mortality. However, the interaction between trawl capture, deck handling, and air exposure represents a series of acute stressors to the fish that may result in decreased survival (Davis and Olla 2002). With respect to the sublethal effects of capture on bycatch, it is well known that acute stressors have detrimental effects on a variety of processes, including behavior, growth, reproduction, and immunocompetence of adult fish and their progeny (Barton et al. 1987; Balm 1997; Pankhurst and Van der Kraak 1997, 2000; Schreck et al. 1997, 2001). Given that the response of lingcod to acute stressors appears similar to many species so far studied, the sublethal effects of stressors should be borne in mind when setting goals for bycatch handling.

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