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Responses by Pacific Halibut to Air Exposure: Lack of Correspondence among Plasma Constituents and Mortality

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[Note]

Responses by Pacific Halibut to Air Exposure: Lack of Correspondence among Plasma Constituents and Mortality

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Abstract.—Age-1 and age-2 Pacific halibut *Hippoglossus stenolepis* were exposed to a range of times in air (0–60 min) and air temperatures (10°C or 16°C) that simulated conditions on deck after capture to test for correspondence among responses in plasma constituents and mortality. Pacific halibut mortality generally did not correspond with cortisol, glucose, sodium, and potassium since the maximum observed plasma concentrations were reached after exposure to 30 min in air, while significant mortality occurred only after exposure to 40 min in air for age-1 fish and 60 min in air for age-2 fish. Predicting mortality in discarded Pacific halibut using these plasma constituents does not appear to be feasible. Lactate concentrations corresponded with mortality in age-1 fish exposed to 16°C and may be useful predictors of discard mortality under a limited set of fishing conditions.

Pacific halibut *Hippoglossus stenolepis* form the basis for a major fishery using longline gear along the West Coast of the United States and Canada and throughout Alaska waters. Pacific halibut are also caught incidentally in trawl and longline fisheries directed towards other demersal species. Fishing regulations require that all Pacific halibut caught incidentally in other fisheries or that are of sublegal size (<81.3 cm total length [TL]) be discarded back into the ocean. In 2003 15% of Pacific halibut caught were discarded (i.e., 6,488 metric tons was discarded from a total fishery of 44,171 metric tons [Gilroy 2004]). These discards have the potential to limit other directed fisheries if their amount exceeds management targets. Similar constraints by discards on fishing activity and stock–recruitment exist in many other fisheries worldwide in which sensitive species are discarded (Hall et al. 2000).

Fishers or observers can visually estimate the amounts of nontarget fish (bycatch) and their rates of immediate mortality prior to discarding, but the mortality rates of discards are generally unknown. Since delayed discard mortality often cannot be observed directly, the development of substitute measures for delayed mortality in fisheries would reduce uncertainty in estimates of fishing mortality and improve management. These measures may also be useful in assessing mortality associated with other anthropogenic stressors (e.g., thermal or chemical pollution, dam passage, fish transport, and caging). There are few methods for estimating mortality rates after fish are discarded. Correlating observed wounding and delayed mortality in discarded Pacific halibut with tag and recapture methods has been used under a limited set of fishing conditions by the International Pacific Halibut Commission (IPHC) to model and calculate discard mortality rates (Williams et al. 1989; Trumble et al. 2000). Measurement of plasma constituents in fish stressed by capture or handling has been suggested as a method for estimating delayed mortality (Schreck 1981; Wood et al. 1983; Schreck et al. 1989; Olla et al. 1998). A preliminary effort was made to extend the scope of the discard mortality model for IPHC Pacific halibut by using plasma constituents as predictive measures for potential mortality in a study of trawl capture at two towing times (30 and 120 min) and deck handling from 1 to 20 min (Oddsson et al. 1994). Results of their plasma study suggested that glucose and sodium may reflect the effects of towing time on mortality, while potassium may reflect the effects of handling time. These results showed that no single variable would be associated with Pacific halibut discard mortality and that plasma constit-

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uents reacted differently to different types of stressors.

The goal of this study was to investigate changes in plasma constituents and mortality associated with handling in air. We tested the hypothesis that exposure to air and elevated temperature (similar to conditions experienced during handling of trawl catches) would result in responses of plasma constituents that corresponded with observed mortality. The role of fish age in affecting the extent of stress and mortality experienced was also examined, as younger fish are often more sensitive to stressors than older fish.

Methods

Age-0 Pacific halibut were collected with a beam trawl from Chiniak Bay, Kodiak Island, Alaska (57°40′N, 152°30′W) in the summers of 2002 and 2003. Fish were air-shipped to the laboratory in Newport, Oregon, where they were reared for up to 20 months prior to the experiments. They were initially maintained in circular 159-L tanks with a thin layer of sand on the bottom and flow-through seawater at 8–10°C, and were fed to satiation three times a week on chopped pink shrimp *Pandalus jordani* and squid *Loligo opalescens*. When fish reached 10 cm total length (TL) they were transferred to circular 15,904-L tanks with a thin layer of sand on the bottom and flow-through seawater at 6–7°C, and fed to satiation three times a week on gel food consisting of salmon feed pellets, squid, Pacific herring *Clupea pallasii*, krill *Euphausia superba*, amino acid supplements, vitamins, and gelatin. Pacific halibut were used in air stressor experiments after they reached age 1 (16.7–31.0 cm TL; held in the laboratory for 8 months) and age-2 (39.5–50.2 cm TL; held in the laboratory for 20 months).

Time course for plasma constituents.—The short-term (4-h) time course for induction of changes in plasma concentrations of cortisol, lactate, glucose, sodium, and potassium associated with air exposure was determined using age-2 Pacific halibut. Baseline plasma constituents in fish were determined by quickly capturing eight Pacific halibut by net from holding tanks, giving them a lethal dose of tricaine methanesulfonate (400 mg/L), and then sampling their blood by needle from the caudal vein into heparinized Vacutainers stored on ice. An additional 24 Pacific halibut were then captured by net and four groups of six fish were placed in four rectangular tanks (90 × 60 × 30 cm) and exposed to air in a controlled temperature room. Fish were held in 16°C air for 30 min and allowed to move freely. Fish ceased movement after no longer than 1 min in air and no attempt was made to account for variation in activity levels among treatments. This protocol simulated a typical time for trawl-captured Pacific halibut to be exposed to air during sorting on the deck of a fishing vessel, but did not include the effects of towing in a net which would be expected to decrease tolerance of air exposure (Hoag 1975; Trumble et al. 1995; Davis and Olla 2001). After 30-min air exposure one group of six fish were given a lethal dose of tricaine methanesulfonate and blood was sampled immediately. The other three groups of six fish were placed in three, circular 3,140-L tanks supplied with flow-through seawater at 6.0°C for recovery. Then at 1, 2, and 4 h after air exposure, fish were quickly captured by net, given a lethal dose of tricaine methanesulfonate, and blood was sampled. No moribund fish were sampled during these experiments. The plasma was separated from other components by centrifugation (3 min at 2,500 × gravity). Plasma was collected and frozen at −80°C until analysis. Cortisol was determined by ^{3}H radioimmunoassay as described by Foster and Dunn (1974), modified by Redding et al. (1984), and validated by C. B. Schreck (Corvallis laboratory) for Pacific halibut. Lactate was determined by fluorometry (Passonneau 1974). Glucose was determined by the orthotoluidine colorimetric procedure according to Wedemeyer and Yasutake (1977). Sodium and potassium ion concentrations were determined with ion detection electrode using a NOVA sodium–potassium analyzer (Nova Biomedical, Newton, Massachusetts).

Plasma constituents after air exposure.—The variation in plasma constituent concentrations associated with the effects of air exposure time and temperature was determined in age-1 and age-2 Pacific halibut. Initially, six age-1 fish were netted, given a lethal dose of tricaine methanesulfonate, and sampled to determine baseline concentrations of plasma cortisol, lactate, glucose, sodium, and potassium. Then an additional 36 fish were netted and six groups, each containing six fish, were placed in six rectangular tanks. Groups of six fish in a tank were exposed to air for 20, 30, or 40 min at 10.0°C or 16.0°C. Fish were allowed to move freely and ceased movement after no later than 1 min in air and no attempt was made to account for variation in activity levels among treatments. After air exposure, fish were given a lethal dose of tricaine methanesulfonate and sampled immediately. The experiment was repeated in the same way with

TABLE 1.—Pacific halibut plasma concentrations of cortisol (ng/mL), lactate, glucose, potassium, and sodium (all mmol/L) in baseline fish and fish that had recovered (0–4 h) in seawater after exposure to air (30 min at 16°C). Values for plasma constituents are means ± SEs. Different lowercase letters indicate statistically different values based on multiple comparisons from a one-way analysis of variance.

Plasma constituent	Baseline	Recovery (h)			
		0	1	2	4
Cortisol	15 (7.4) y	137 (9.1) z	175 (12.2) z	154 (17) z	112 (24) z
Lactate	0.1 (0.0) x	0.4 (0.0) yz	0.5 (0.1) z	0.4 (0.1) yz	0.2 (0.1) xy
Glucose	1.9 (0.1) y	4.7 (0.3) z	4.1 (0.4) z	4.8 (0.8) z	3.8 (0.2) z
Sodium	176 (1.0) x	188 (1.6) yz	193 (1.9) z	188 (2.7) yz	183 (1.7) xy
Potassium	3.9 (0.2) z	4.4 (0.3) z	4.1 (0.2) z	4.0 (0.1) z	3.6 (0.1) z

age-2 fish after completion of the first experiment. No moribund fish were sampled during these experiments.

Mortality after air exposure.—The variation in mortality associated with air exposure and temperature was determined in age-1 and age-2 Pacific halibut. Sixty age-1 fish were netted from holding tanks and 10 groups of six fish each were placed in 10 rectangular tanks and exposed to air for 20, 30, 40, 50, or 60 min at 10.0°C or 16.0°C. Fish were then returned to flowing seawater and kept together in treatment groups in 20 circular 3,140-L tanks for 36 d. Mortality was observed 1 h after air exposures and then daily for 36 d. Fish were fed chopped squid twice per week during the recovery period. The experiment was repeated in the same way with age-2 fish after completion of the first experiment.

Statistical analysis.—The variation in plasma constituent concentrations associated with recovery time in seawater (baseline, 0–4 h) after exposure to air for 30 min was tested by one-way analysis of variance (ANOVA). Variation in plasma constituents associated with air exposure, air temperature, and fish age were tested by three-way ANOVA with interactions. Results were considered statistically significant at $\alpha < 0.050$, and multiple comparisons were made using the Tukey method for all pairwise comparisons (either for recovery time in the time course experiment, or for time in air × air temperature × fish age in the air exposure experiment). In the mortality experiment, each treatment group had six fish. Statistically significant mortality within a treatment group was observed when five fish out of six died in a group (one-tailed sign test: $P = 0.031$). The one-tailed sign test was used based on previous mortality results. Significant mortality occurred in treatments in which 83–100% mortality was observed. Correspondence between plasma constituents and mortality was determined by comparing treatments in which significant mortality and maximum plasma constituent concentrations were reached. All calculations were performed using the Statistix 8.0 program.

Results

Time Course for Plasma Constituents

Immediately after Pacific halibut were exposed to 16°C air for 30 min, plasma cortisol, lactate, glucose, and sodium concentrations were higher than baseline (Table 1; $P = 0.001$), while potassium was unchanged ($P = 0.079$). Through 4 h after fish exposure to air, there were no further significant changes in concentrations of cortisol, glucose, and potassium. In contrast, lactate and sodium were lower than maximum values observed and were recovered by 4 h after fish exposure to air.

Plasma Constituents after Air Exposure

Exposure of Pacific halibut to air resulted in higher concentrations of cortisol, lactate, glucose, sodium, and potassium than in baseline fish (Figure 1; Table 2; $P = 0.001$). Lactate in age-1 fish exposed to 16°C air was the only plasma constituent that showed significantly higher concentrations (multiple comparisons) as exposure time increased from 20 to 40 min. All other plasma constituent concentrations were similar among air exposure times (20–40 min) when compared within air temperature and fish age classifications. Exposure of fish to 16°C air resulted in a higher mean concentration of lactate and potassium ($P = 0.003$) and a lower mean concentration of cortisol ($P = 0.035$) and no apparent changes for glucose and sodium ($P > 0.065$) compared with fish at 10°C. Younger fish had higher mean lactate and potassium levels than older fish ($P = 0.003$), while older fish had higher cortisol, glucose, and sodium levels than younger fish ($P < 0.010$). There were significant interactions between the effects of time in

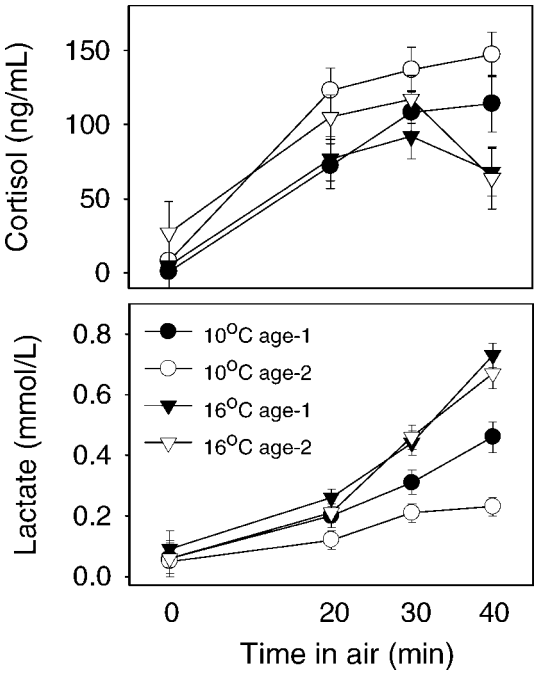

FIGURE 1.—Pacific halibut plasma cortisol and lactate concentrations associated with air exposure (0–40 min), air temperature (10°C or 16°C), and fish age (age 1 or age 2). Fish with no air exposure (0 min) were netted from holding tanks (6–7°C) and sampled immediately. Values for plasma constituents are mean ± SE.

air and air temperature for cortisol ($P = 0.040$), and lactate and potassium ($P = 0.001$). There were significant interactions between the effects of time in air and fish age for glucose ($P = 0.014$) and sodium ($P = 0.004$). No three-way interactions were observed.

Mortality after Air Exposure

No significant mortality occurred within 1 h of exposure to 10°C air for up to 60 min in age-1 and age-2 Pacific halibut (Table 2). When exposed to 16°C air, age-1 fish showed significant mortality within 1 h after exposure to 40 min in air (83.3%) and 50 and 60 min in air (100%); while age-2 fish showed significant mortality within 1 h after exposure to 60 min in air (100%). Through the course of these experiments, no further mortality was observed after initial observations made 1 h after air exposure.

Discussion

Rapid induction of higher plasma constituent concentrations has often been noted in fish subjected to many types of acute stressors (Donaldson 1981; Wedemeyer 1996; Schreck et al. 1997). When Pacific halibut were exposed to 16°C air which simulated handling of trawl catches, fish showed maximum observed plasma concentrations of cortisol, lactate, glucose, sodium, and potassium by 30 min from the start of air exposure and no further increases up to 4 h later in recovery seawater tanks. These results suggested that sampling 30 min after the start of exposure to these particular stressors probably captured maximum observed values for plasma cortisol, lactate, glucose, sodium, and potassium in Pacific halibut. By 4 h after air exposure, plasma lactate and sodium concentrations had recovered to baseline levels, while cortisol and glucose remained elevated. It is possible that plasma constituents in Pacific halibut would take longer to reach maximum levels or to return to baseline levels after exposure to colder air temperatures because of slower metabolic rates, but we did not test for this effect. Resolution of temperature effects on the balance between rates for secretion and clearance of these constituents is an interesting question that was well beyond the scope of this study.

Plasma responses in Pacific halibut associated with air exposure and increased temperature were similar in magnitude and timing to those observed in other flatfishes exposed to capture-related stressors (e.g., towing in nets, swimming, crowding, air exposure, or increased temperature; Wardle 1972; Fletcher 1975; Wardle 1978; Jørgensen and Mustafa 1980; Turner et al. 1983; Fletcher 1984; White and Fletcher 1986; Milligan and Wood 1987; White and Fletcher 1989; Girard and Milligan 1992; Waring et al. 1992; Oddsson et al. 1994; Waring et al. 1996; Via et al. 1997; Barnett and Pankhurst 1998; Plante et al. 2003). In these past studies maximum (and baseline) observed plasma concentrations for cortisol ranged from 70 to 255 (5–100) ng/mL; for lactate, from 0.4 to 20.0 (0.1–2.2) mmol/L; for glucose, from 1.1 to 10.5 (0.4–3.0) mmol/L; for potassium, from 3.1 to 6.0 (2.3–3.8) mmol/L; and for sodium, from 155 to 252 (136–230) mmol/L. Maximum concentrations observed for these compounds were generally reached within 1 h from the start of exposure to an acute stressor.

Patterns of response in plasma constituents can vary with stressor type. In an earlier study of trawl-caught Pacific halibut, higher levels of plasma potassium were observed as handling time increased from 1 to 20 min, while higher levels of glucose and sodium reflected increased towing time from 30 to 120 min (Oddsson et al. 1994). In the present

TABLE 2.—Pacific halibut plasma glucose, sodium, and potassium concentrations (mmol/L) and mortality (%) associated with exposure to air for various periods of time and at two different temperatures, by age of fish (1 or 2). Fish with no air exposure (0 min) were netted from holding tanks (6–7°C) and sampled immediately for plasma or transferred into recovery tanks for mortality observations. Values for plasma constituents are means ± SEs. Asterisks indicate significant mortality (sign test; $n = 6$).

Temperature (°C)	Age (years)	Exposure time (min)					
		0	20	30	40	50	60
		Glucose					
10	1	1.9 (0.6)	3.3 (0.4)	3.1 (0.4)	3.7 (0.5)		
10	2	1.9 (0.5)	3.4 (0.3)	4.9 (0.3)	4.7 (0.3)		
16	1	2.1 (0.6)	3.4 (0.3)	3.6 (0.4)	3.9 (0.4)		
16	2	1.7 (0.5)	4.4 (0.3)	5.0 (0.3)	6.0 (0.3)		
		Sodium					
10	1	170 (2.5)	180 (1.6)	186 (1.6)	192 (2.0)		
10	2	181 (2.0)	184 (1.4)	185 (1.4)	187 (1.4)		
16	1	174 (2.5)	184 (1.4)	184 (1.6)	188 (1.6)		
16	2	179 (2.0)	187 (1.4)	188 (1.7)	188 (2.0)		
		Potassium					
10	1	3.9 (0.6)	6.2 (0.4)	6.0 (0.4)	6.5 (0.5)		
10	2	3.8 (0.5)	4.7 (0.4)	4.3 (0.4)	4.0 (0.4)		
16	1	6.0 (0.6)	4.9 (0.4)	6.4 (0.4)	8.1 (0.4)		
16	2	4.4 (0.5)	4.5 (0.4)	4.6 (0.5)	6.3 (0.5)		
		Mortality					
10	1	0	0	0	16.7	0	16.7
10	2	0	0	0	0	0	0
16	1	0	0	0	83.3*	100*	100*
16	2	0	16.7	0	16.7	33.3	100*

study, similar variation in response patterns was evident. Lactate and potassium were higher in 16°C than in 10°C air, while temperature had no effect on cortisol, glucose, and sodium. Lactate and potassium were higher in younger fish, while cortisol, glucose, and sodium were higher in older fish. Because age-1 Pacific halibut were reared for 8 months and age-2 Pacific halibut were reared for 20 months in the laboratory prior to experimentation, these results may be attributable to age differences or to differences in fish condition associated with laboratory rearing. We were not able to discriminate between these alternatives. Smaller (younger) fish are widely considered to be more sensitive to capture-related stressors than larger (older) fish (Davis 2002). When sablefish *Anoplopoma fimbria* were reared in the laboratory for up to 20 months and then exposed to air for up to 60 min, younger fish had higher mortality than older fish (Davis and Parker 2004).

Pacific halibut mortality in the present study did not correspond with plasma cortisol, glucose, sodium, and potassium concentrations. A similar lack of correspondence between these plasma constituents and mortality has been noted in captured sablefish and lingcod *Ophiodon elongatus* (Davis et al. 2001; Davis 2002; Parker et al. 2003). Lactate corresponded to mortality in sablefish and Pacific halibut that were exposed to elevated temperature (Olla et al. 1998; this study), but did not correspond to sablefish mortality under other combinations of towing, hooking, or increased seawater temperature (Davis et al. 2001). While lactate may be an exception under some stressor conditions, plasma constituents generally cannot be used to predict mortality in captured Pacific halibut and other marine species since these constituents do not show consistent responses to different types of stressors and could not be matched to levels of mortality (Davis 2002).

The task of predicting stress and mortality in captured fish is made more difficult by interactions among stressors in the fishing process. Mortality of fish that are handled and discarded from fishing operations is a function of a complex suite of potential stressors including capture, wounding, catch constituents, fish size and species, seawater temperature, light conditions, air exposure and temperature, handling, infection, and behavior deficits associated with increased predation (Davis 2002, 2005). In the present study for fish not towed in a net, Pacific halibut showed mortality after 40–60 min in air. The interaction of trawl capture and air exposure decreased the time in air that resulted

in significant mortality, as trawl-caught Pacific halibut showed mortality after 20–40 min in air on deck (Hoag 1975; Oddsson et al. 1994; Trumble et al. 1995). Similar interactions between the effects of towing in a net and air exposure have been observed in lingcod, with mortality after 45 min in air for towed fish and 60 min in air for fish that were not towed (Davis and Olla 2002).

Stress is a normal adaptive response by fish to changes in internal or external conditions. When physiological tolerance limits are exceeded, mortality may result. Changes in concentrations of fish plasma constituents are generally related to acute and chronic stressor intensity and may be useful for measuring the level of sublethal stress induced by fishing and aquaculture practices (Schreck 1981; Wood et al. 1983; Wedemeyer 1996). However, these patterns of response have not been widely investigated under conditions in which stressors transition from sublethal to lethal conditions that are often present in fishing operations and other anthropogenic sources for fish injury (e.g., thermal or chemical pollution, dam passage, fish transport, and caging). Development of alternative substitute measures for potential mortality in fish exposed to anthropogenic stressors should consider a wide range of fitness measures that integrate animal condition in an ecologically meaningful manner and that can be measured in field settings (Olla et al. 1980; Schreck et al. 1997; Cooke et al. 2002; Davis 2005). Examples of possible measures include injury, loss of orientation, reflex actions, swimming activity, and rates of predation on stressed fish.

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References

Barnett, C. W., and N. W. Pankhurst. 1998. The effects of common laboratory and husbandry practices on the stress response of greenback flounder *Rhombosolea tapirina* (Günther, 1862). Aquaculture 162: 313–329.

Cooke, S. J., J. F. Schreer, K. M. Dunmall, and D. P. Phillip. 2002. Strategies for quantifying sublethal effects of marine catch-and-release angling: insights from novel freshwater applications. Pages 121–134 *in* J. A. Lucy and A. L. Studholme, editors. Catch and release in marine recreational fisheries. American Fisheries Society, Symposium 30, Bethesda, Maryland.

Davis, M. W. 2002. Key principles for understanding fish bycatch discard mortality. Canadian Journal of Fisheries and Aquatic Sciences 59:1834–1843.

Davis, M. W. 2005. Behaviour impairment in captured and released sablefish: ecological consequences and possible substitute measures for delayed discard mortality. Journal of Fish Biology 66:254–265.

Davis, M. W., and B. L. Olla. 2001. Stress and delayed mortality induced in Pacific halibut by exposure to hooking, net towing, elevated seawater temperature and air: implications for management of bycatch. North American Journal of Fisheries Management 21:725–732.

Davis, M. W., and B. L. Olla. 2002. Mortality of lingcod towed in a net as related to fish length, seawater temperature, and air exposure: a laboratory bycatch study. North American Journal of Fisheries Management 22:1095–1104.

Davis, M. W., and S. J. Parker. 2004. Fish size and exposure to air: potential effects on behavioral impairment and mortality rates in discarded sablefish. North American Journal of Fisheries Management 24:518–524.

Davis, M. W., B. L. Olla, and C. B. Schreck. 2001. Stress induced by hooking, net towing, elevated sea water temperature, and air in sablefish: lack of concordance between mortality and physiological measures of stress. Journal of Fish Biology 58:1–15.

Donaldson, E. M. 1981. The pituitary–interrenal axis as an indicator of stress in fish. Pages 11–47 *in* A. D. Pickering, editor. Stress and fish. Academic Press, London.

Fletcher, D. J. 1984. Plasma glucose and plasma fatty acid levels of *Limanda limanda* (L.) in relation to season, stress, glucose loads, and nutritional state. Journal of Fish Biology 25:629–648.

Fletcher, G. L. 1975. The effects of capture, ''stress,'' and storage of whole blood on the red blood cells, plasma proteins, glucose, and electrolytes of the winter flounder (*Pseudopleuronectes americanus*). Canadian Journal of Zoology 53:197–206.

Foster, L. B., and R. T. Dunn. 1974. Single-antibody technique for radioimmunoassay of cortisol in unextracted serum or plasma. Clinical Chemistry 20: 365–368.

Gilroy, H. L. 2004. The Pacific halibut fishery, 2003. International Pacific Halibut Commission. Available: http://www.iphc.washington.edu/halcom/pubs/ammeet/2004/bluebook/fishery.pdf. (September 2004).

Girard, S. S., and C. L. Milligan. 1992. The metabolic fate of blood-borne lactate in winter flounder (*Pseu-*

dopleuronectes americanus) during recovery from strenuous exercise. Physiological Zoology 65: 1114–1134.

Hall, M. A., D. L. Alverson, and K. I. Metuzals. 2000. By-catch: problems and solutions. Marine Pollution Bulletin 41:204–219.

Hoag, S. H. 1975. Survival of halibut released after capture by trawls. International Pacific Halibut Commission, Scientific Report 57, Seattle.

Jørgensen, J. B., and T. Mustafa. 1980. The effect of hypoxia on carbohydrate metabolism in flounder (*Platichthys flesus* L.), I. Utilization of glycogen and accumulation of glycolytic end products in various tissues. Comparative Biochemistry and Physiology 67B:243–248.

Milligan, C. L., and C. M. Wood. 1987. Muscle and liver intracellular acid–base and metabolite status after strenuous activity in the inactive, benthic starry flounder *Platichthys stellatus*. Physiological Zoology 60:54–68.

Oddsson, G., E. K. Pikitch, W. Dickhoff, and D. L. Erickson. 1994. Effects of towing, sorting, and caging on physiological stress indicators and survival in trawl caught and discarded Pacific halibut. Pages 437–442 *in* D. D. MacKinley, editor. High-performance fish: proceedings of an international fish physiology symposium. Fish Physiology Association, Vancouver.

Olla, B. L., M. W. Davis, and C. B. Schreck. 1998. Temperature-magnified postcapture mortality in adult sablefish after simulated trawling. Journal of Fish Biology 53:743–751.

Olla, B. L., W. H. Pearson, and A. L. Studholme. 1980. Applicability of behavioral measures in environmental stress assessment. Rapports et Procès-verbaux des Réunions, Conseil International pour l'Exploration de la Mer 179:162–173.

Parker, S. J., P. S. Rankin, R. W. Hannah, and C. B. Schreck. 2003. Discard mortality of trawl-caught lingcod in relation to tow duration and time on deck. North American Journal of Fisheries Management 23:530–542.

Passonneau, J. V. 1974. Fluorimetric method. Pages 1468–1472 *in* H. U. Bergmeyer, editor. Methods of enzymatic analysis. Academic Press, New York.

Plante, S., C. Audet, Y. Lambert, and J. de la Noüe. 2003. Comparison of stress responses in wild and captive winter flounder (*Pseudopleuronectes americanus* Walbaum) broodstock. Aquaculture Research 34:803–812.

Redding, J. M., C. B. Schreck, E. K. Birks, and R. D. Ewing. 1984. Cortisol and its effects on plasma thyroid hormone and electrolyte concentrations in freshwater and during seawater acclimation in yearling coho salmon, *Oncorhynchus kisutch*. General and Comparative Endocrinology 56:146–155.

Schreck, C. B. 1981. Stress and compensation in teleostean fishes: response to social and physical factors. Pages 295–321 *in* A. D. Pickering, editor. Stress and fish. Academic Press, London.

Schreck, C. B., M. F. Solazzi, S. L. Johnson, and T. E. Nickelson. 1989. Transportation stress affects performance of coho salmon, *Oncorhynchus kisutch*. Aquaculture 82:15–20.

Schreck, C. B., B. L. Olla, and M. W. Davis. 1997. Behavioral responses to stress. Pages 145–170 *in* G. K. Iwama, A. D. Pickering, J. P. Sumpter, and C. B. Schreck, editors. Fish stress and health in aquaculture. Cambridge University Press, New York.

Trumble, R. J., S. M. Kaimmer, and G. H. Williams. 2000. Estimation of discard mortality rates for Pacific halibut bycatch in groundfish longline fisheries. North American Journal of Fisheries Management 20:931–939.

Trumble, R. J., G. H. Williams, and S. E. Hughes. 1995. Methods to improve survival of Pacific halibut bycatch discarded from a factory trawler. Pages 591–610 *in* Proceedings of the International Symposium on North Pacific Flatfish. Alaska Sea Grant College Program, Fairbanks.

Turner, J. D., C. M. Wood, and H. Hobe. 1983. Physiological consequences of severe exercise in the inactive benthic flathead sole (*Hippoglossoides elassodon*): a comparison with the active pelagic rainbow trout (*Salmo gairdneri*). Journal of Experimental Biology 104:269–288.

Via, J. D., G. van den Thillart, O. Cattani, and P. Cortesi. 1997. Environmental versus functional hypoxia/anoxia in sole *Solea solea*: the lactate paradox revisited. Marine Ecology Progress Series 154:79–90.

Wardle, C. S. 1972. The changes in blood glucose in *Pleuronectes platessa* following capture from the wild: a stress reaction. Journal of Marine Biology Association of the United Kingdom. 52:635–651.

Wardle, C. S. 1978. Nonrelease of lactic acid from anaerobic swimming muscle of plaice *Pleuronectes platessa* L.: a stress reaction. Journal of Experimental Biology 77:141–155.

Waring, C. P., R. M. Stagg, and M. G. Poxton. 1992. The effects of handling on flounder (*Platichthys flesus* L.) and Atlantic salmon (*Salmo salar* L.). Journal of Fish Biology 41:131–144.

Waring, C. P., R. M. Stagg, and M. G. Poxton. 1996. Physiological responses to handling in the turbot. Journal of Fish Biology 48:161–173.

Wedemeyer, G. A. 1996. Physiology of fish in intensive culture systems. Chapman and Hall, New York.

Wedemeyer, G. A., and W. T. Yasutake. 1977. Clinical methods for the assessment of the effects of environmental stress on fish health. U.S. Fish and Wildlife Service Technical Papers 89:1–18.

White, A., and T. C. Fletcher. 1986. Serum cortisol, glucose, and lipids in plaice (*Pleuronectes platessa* L.) exposed to starvation and aquarium stress. Comparative Biochemistry and Physiology 84A:649–653.

White, A., and T. C. Fletcher. 1989. The effect of physical disturbance, hypoxia, and stress hormones on serum components of the plaice, *Pleuronectes platessa* L. Comparative Biochemistry and Physiology 93A(2):455–461.

Williams, G. H., C. C. Schmitt, S. H. Hoag, and J. D.

Berger. 1989. Incidental catch and mortality of Pacific halibut, 1962–1986. International Pacific Halibut Commission, Technical Report 23, Seattle.

Wood, C. M., J. D. Turner, and M. S. Graham. 1983. Why do fish die after severe exercise? Journal of Fish Biology 22:189–201.