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Mechanisms to explain purse seine bycatch mortality of coho salmon

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Abstract. Research on fisheries bycatch and discards frequently involves the assessment of reflex impairment, injury, or blood physiology as means of quantifying vitality and predicting post-release mortality, but exceptionally few studies have used all three metrics concurrently. We conducted an experimental purse seine fishery for Pacific salmon in the Juan de Fuca Strait, with a focus on understanding the relationships between different sublethal indicators and whether mortality could be predicted in coho salmon (*Oncorhynchus kisutch*) bycatch. We monitored mortality using a ~24-h net pen experiment ($N = 118$) and acoustic telemetry ($N = 50$), two approaches commonly used to assess bycatch mortality that have rarely been directly compared. Short-term mortality was 21% in the net pen experiment (~24 h) and estimated at 20% for telemetry-tagged fish (~48–96 h). Mortality was predicted by injury and reflex impairment, but only in the net pen experiment. Higher reflex impairment was mirrored by perturbations to plasma ions and lactate, supporting the notion that reflex impairment can be used as a proxy for departure from physiological homeostasis. Reflex impairment also significantly correlated with injury scores, while injury scores were significantly correlated with plasma ion concentrations. The higher time-specific mortality rate in the net pen and the fact that reflexes and injury corresponded with mortality in that experiment, but not in the telemetry-tagged fish released into the wild could be explained partly by confinement stress. While holding experiments offer the potential to provide insights into the underlying causes of mortality, chronic confinement stress can complicate the interpretation of patterns and ultimately affect mortality rates. Collectively, these results help refine our understanding of the different sublethal metrics used to assess bycatch and the mechanisms that can lead to mortality.

Key words: *acoustic telemetry in fisheries management; biodiversity conservation; bycatch; conservation physiology; fisheries management; gillnet; interior Fraser coho salmon; RAMP; scale loss.*

Introduction

Bycatch mortality in marine commercial fisheries is a leading conservation issue (Kappel 2005) because it can contribute to population or species imperilment (Lewison et al. 2004, Read et al. 2006) and affect the sustainability of fisheries (Milton 2001, Coggins et al. 2007, Davies et al. 2009). A large component of bycatch is typically discarded, often with the hope that it survives, whereas in reality, post-release mortality rates can be substantial but are often unknown (Davis 2002). For management, post-release bycatch mortality is a particular problem in marine systems because it often represents an unquantified source of fishing mortality that is viewed as wasteful and may negatively affect threatened species (Hall et al. 2000, Coggins et al. 2007).

In recent years, a growing body of research has arisen on the use of tools for rapidly assessing delayed bycatch mortality, with several examples of indices of animal condition that have been shown to predict delayed mortality (e.g., Davis 2007, Campbell et al. 2010, Stoner 2012). Increasing in popularity is an approach where a suite of simple reflex responses is rapidly (<20 seconds) tested for impairment in an animal to be released, and

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validated as a predictor of mortality (i.e., reflex action mortality predictors, RAMP; Davis 2010). RAMP goes beyond the traditional approach of categorizing bycatch condition as "good" or "poor" because it involves checking for the presence or absence of simple responses using a conservative, consistent, and objective approach, which then produces a condition score. Injury and blood physiology can also be used as predictors, given that injuries can strongly influence post-release mortality by compromising osmoregulatory function (Olsen et al. 2012), impairing animal locomotion (Ramsay and Kaiser 1998), and providing a pathway for infection (Udomkusonsri and Noga 2005). Blood plasma measures, such as lactate, provide a glimpse of underlying physiological disturbance at the time of release and can be predictive of mortality in some contexts (e.g., Moyes et al. 2006). Rapid and simple mortality predictors are powerful because of (1) the cost and logistical challenges involved in monitoring mortality in wild animals, and (2) the utility of having post-release mortality estimates for different contexts. The notion that a greater departure from homeostasis predicts a greater likelihood of subsequent mortality is intuitively appealing; this is the premise upon which RAMP (and other mortality predictors) is largely based (Raby et al. 2012). Needed, however, is evidence that reflex impairment reflects underlying physiological disturbance: Evidence that could help fisheries managers decide whether implementing the use of RAMP assessments in fisheries observer programs is based on sound science. Moreover, physiological measurements are regarded as objective measures of fish welfare and health (Iwama 2007, Arlinghaus et al. 2009) and have the potential to reveal the cause-and-effect relationships needed to justify management action (Cooke et al. 2013*b*).

Pacific salmon (*Oncorhynchus* spp.) are a useful model for studying discard fate because they are discarded from multi-sector fisheries (commercial, aboriginal, recreational) while migrating on a known trajectory toward their natal spawning grounds. Migration trajectories for individuals can be determined by a noninvasive DNA biopsy, making assessment of mortality using biotelemetry straightforward (Cooke et al. 2008). Of particular concern in British Columbia (Canada) is coho salmon (*O. kisutch*), which are caught incidentally in fisheries targeting other species of salmon (primarily pink salmon *O. gorbuscha* and sockeye salmon *O. nerka*). A major population of wild coho salmon that spawn in the upper watersheds of the Fraser River basin (referred to as interior Fraser coho) collapsed in the 1990s (Bradford and Irvine 2000). The population collapse led to interior Fraser coho being listed as "endangered" by COSEWIC (Committee on the Status of Endangered Wildlife in Canada) and the closure of all directed harvest of wild Fraser River coho salmon in British Columbian waters. Fisheries management (Fisheries and Oceans Canada, referred to as DFO) have also strategically adjusted the timing and extent of harvest fisheries for other species to minimize fishing mortality of coho salmon via bycatch. Coho salmon bycatch mortality limits are set by DFO, both overall and for specific fishery openings. If those limits are reached, based on a product of the total bycatch and the mortality rate used, that fishery is typically closed for the season. Mortality rates used vary extensively by fishery and fishing area (e.g., 5% for in-river beach seines, up to 70% for purse seines, and 60–70% for gillnets; DFO 2013). Application of accurate bycatch mortality rates facilitates DFO meeting spawning escapement targets.

Virtually all bycatch mortality rates used by DFO are based on captivity studies, in which fish are captured and held, usually in a net pen, for a short period of 1–3 days. Temporary captivity is also by far the most common method used to estimate mortality throughout the bycatch literature (Rogers et al. 2014). As an approach to monitoring mortality, short-term captivity has some disadvantages such as the elimination of post-release predation (Raby et al. 2014) and potential imposition of chronic confinement stress (Portz et al. 2006); the latter appears to be especially true for migrating adult salmon (Donaldson et al. 2011). Biotelemetry studies are the most common alternative to net pens, and offer the benefit of monitoring animals released back into the wild (Donaldson et al. 2008). The drawbacks of biotelemetry include costs and logistical constraints, difficulty in quantifying tagging effects, and the inability to physically verify the fate of individuals. Side-by-side comparisons of net pen captivity and biotelemetry have been exceptionally rare in fisheries science, and such work could help improve our understanding of the functional benefits and drawbacks of estimating mortality using both approaches: Knowledge that would be of use both to fisheries managers and scientists.

This study used coho salmon caught by purse seine in Juan de Fuca Strait to address two primary objectives: (1) compare methods of estimating bycatch mortality, and (2) evaluate the interaction of RAMP, injury, body size, and blood physiology in relation to predicting short-term bycatch mortality. To evaluate mortality, we conducted ~24-h net pen holding experiments and an acoustic telemetry tagging experiment using separate groups of fish. This study provides a rare comparison between net pen confinement and biotelemetry as means of monitoring mortality. It is also the first evaluation of RAMP as a vitality index and mortality predictor in salmon caught in the marine environment, after some success in freshwater salmon fisheries (Donaldson et al. 2012, Raby et al. 2012, 2013) and in numerous other fish and crustaceans (Davis 2010, Stoner 2012). Moreover, this study takes the novel step of exploring relationships between different sublethal effects of fisheries capture: injury, reflex impairment, and blood physiology. Understanding relationships among sublethal metrics used to assess bycatch condition could lead to refined research and management practices in a range of fisheries.

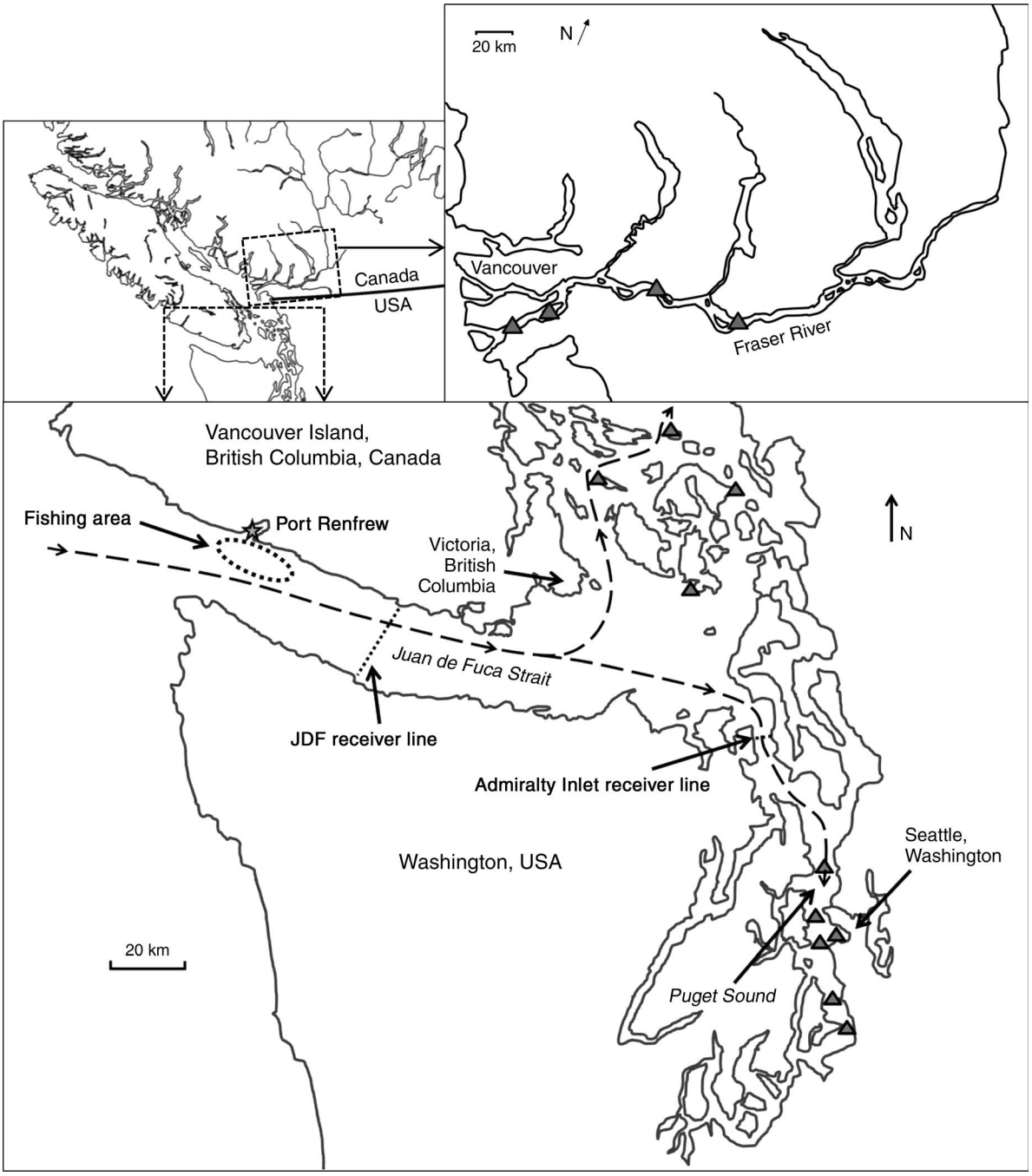

FIG. 1. Map of the study area. The dashed line with directional arrows indicates the general direction of the spawning migration of coho salmon (*Oncorhynchus kisutch*) through the study area (and past our fishing area and acoustic receivers). The Juan de Fuca (JDF) and Admiralty Inlet receiver lines are shown with dotted lines. Other individual receivers where coho salmon were detected are shown by gray-filled triangles. The location where the ship moored overnight for the ~24-h confinement study is indicated by a star.

MATERIALS AND METHODS

Fish capture

This study took place from 20 to 27 August, 2012, in the Juan de Fuca Strait near Port Renfrew, British Columbia (Canada). In total, 30 purse seine sets were carried out on the Canadian side of the strait, between Sombrio Point (48°29′ N, 124°17′ W) to the east and adjacent to Bonilla Point on the western-most edge of the strait (48°36′ N, 124°43′ W), ranging in depths from 86 m (near shore) to 220 m (at the international boundary; see Fig. 1). A 549 m long × 55 m deep seine

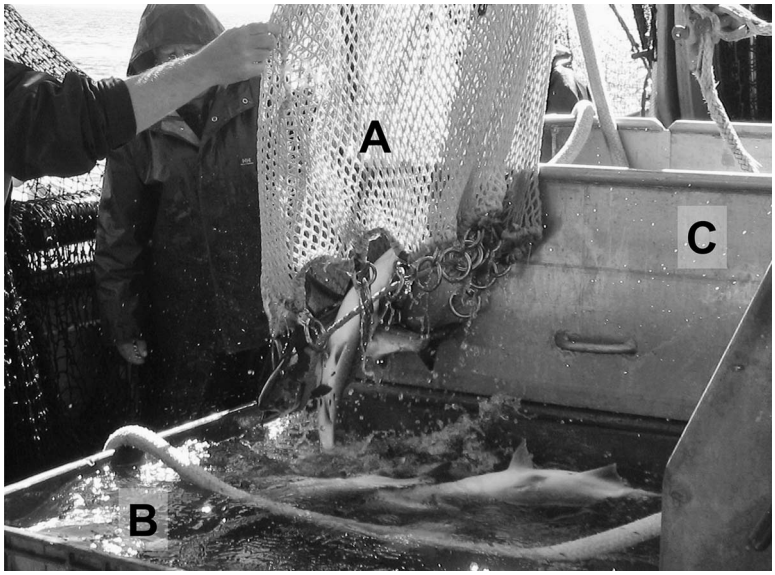

FIG. 2. Photo showing the process by which the purse seine catch was brought on board; by transferring fish using (A) a brailer into (B) an industry-standard recovery tote. In a true fishery, catch would be brailed into the (C) metal sorting bin, and then sorted into recovery totes (or onto ice, in the case of the target species).

with 100-mm bunt mesh was deployed and towed by the *Franciscan No. 1* and her power skiff. The net was always deployed such that it was stretched from north to south and towed from east to west, against the general direction of movement of adult salmon homing to coastal spawning streams, as is standard for open water purse seine salmon fishing in Juan de Fuca Strait. Towing time was always 20 min, and the distance covered varied depending on tidal flow (up to 1.5 km towing distance) before the net was closed and pursed. After 20 min, the two ends of the net were pulled together such that the fish were fully encircled (which required 6 min 26 s ± 3 min 25 s [mean ± SD]), at which point the net was drawn in and up (pursed) to the side of the boat such that the fish could be retrieved (which required a further 24 min 9 s ± 1 min 45 s). Water temperatures recorded by the boat's subsurface digital thermometer ranged from 8.5°C to 9.8°C (mean = 9.3°C) during net sets. Weather conditions were consistent during the eight-day study: mostly sunny, little precipitation (6 mm total), with a mean daily air temperature of 14.3°C (range: 12.8°–15.5°C), and a mean daily maximum temperature of 19.4°C (18°–22°C). All fishing was conducted during daylight hours, with set landing times ranging from 08:37 to 16:19 h.

Fish were brought from the pursed seined net into the boat using an industry-standard brailer (a large dip net operated with the assistance of a hydraulic winch; see Fig. 2), directly into a recovery tote (61 cm deep × 109 cm × 119 cm) that was continuously flushed with fresh seawater (Fig. 2). Brailing resulted in ~20 s of air exposure and potential physical trauma due to crushing. In cases where the number of salmon brailed into the recovery tote resulted in severe crowding (e.g., >20 fish in the tote), a portion of the fish were rapidly transferred with dip nets to a second, smaller recovery tote (61 × 71 × 119 cm). The two recovery totes used were industry standard "half totes" normally used to revive bycatch before release. Catches were not brailed into the metal sorting bin (Fig. 2C) prior to transfer to recovery totes, as in a standard fishery, because in each net set virtually all of the catch was bycatch coho salmon. Catch rates were sufficiently small that only one brailer load was required for each set (the largest catch was 82 fish). The small catch sizes and direct brailing into recovery totes were the only two ways in which our capture process differed markedly from standard commercial practice. Coho salmon were in recovery totes for a mean duration of 27 min 41 s (range = 10 s–1 h 47 min) before they were processed by the research team. The crew was directed to conduct one experimental set where fish were brailed into the sorting bin and then transferred to the recovery totes, which added ~60 s of air exposure and the potential for further dermal injury. Coho salmon from that set were used only to test for differences in plasma measures, RAMP, and injury (see next section), and not mortality assessment. On an added day of the study, the purse seine netting was changed from the industry standard 100-mm bunt to a 70-mm knotless nylon mesh to test for differences in catch composition and whether it could improve the condition of coho salmon bycatch. Data collected from those experimental sets and from the comparison between the two brailing approaches are summarized in the Appendix.

Reflex, injury, and blood measurements

Once fish were on board, individual coho salmon were randomly selected from the recovery totes and dipnetted

to an adjacent V-shaped foam-padded sampling trough that was continuously flushed with seawater. First, fish were held supine for withdrawal of a 1–2 mL sample of blood via caudal puncture (only a subset of fish were blood sampled; 21 gauge needle, 3-mL vacutainer coated with lithium heparin; BD, Franklin Lakes, New Jersey, USA). All fish were assessed for reflex impairment (i.e., RAMP; Davis 2010), which required <15 s to complete and involved checking for the presence of five reflexes (tail grab, body flex, head complex, orientation, vestibular–ocular response) that have been previously described and validated in adult Pacific salmon (Donaldson et al. 2012, Raby et al. 2012, 2013, Nguyen et al. 2014) and other marine fishes (Brownscombe et al. 2013, Cooke et al. 2014). The reflex responses were used to calculate a RAMP score of 0–1 that represents the proportion of reflexes that were impaired. Thus, a high RAMP score corresponds to a fish in poor condition.

For each coho salmon, we also recorded observations about whether gillnet-like injuries were visible around the head (0/1), or the center of the body (0/1). Gilling injuries were characterized by a combination of dark contusion lines and focused rings of scale loss. In addition, we noted whether overall scale loss was low (<5% of total body), moderate (~5–30%), or high (>30%), which was scored as a 0, 1, or 2, respectively. Finally, we recorded whether any other injuries were present (0/1), such as old hooking or predator wounds. The injury scores were synthesized into an overall injury score (proportion) in a similar manner to RAMP scores, by dividing by the highest possible total (i.e., 5). Thus, a higher injury score represented a fish that experienced greater and/or more numerous injuries. For some analyses, we also used a combined condition score that was a combined proportion of RAMP and injury scores.

After blood sampling (when applicable) and RAMP-injury assessments, fish were measured (fork length, FL, nearest cm), followed by removal of a ~0.5-g piece of adipose fin tissue (except for hatchery-origin salmon whose adipose fins are removed prior to release to the wild as smolts) that was stored in 95% ethanol for later analysis of DNA. Finally, coho salmon were tagged for the net pen holding experiment (mix of hatchery-origin fish and wild fish) or telemetry tracking (wild fish only). Additional fish were assessed for reflex impairment, injury, and blood physiology to increase our sample sizes for evaluation of interactions among capture variables, and those fish were released untagged following assessment (i.e., if the holding experiment was already underway or to conserve transmitters for other net sets or study days). Many of the coho salmon caught were not used in this study and were merely counted; some were lethally sampled for a separate study, while others were simply released because of time limitations (i.e., so that fishing could resume). We did not collect tissue for population identification of hatchery-origin fish (e.g., from operculum clips). Hatchery-origin fish (identifiable by missing adipose fins) were likely to have consisted of a mix of origins in the United States and Canada, but with the majority coming from tributaries of the Puget Sound basin (based on high hatchery production there for stock enhancement purposes and the high proportion of Puget Sound fish among wild fish sampled; see *Results*). Hatchery-origin coho salmon were included to ensure adequate sample sizes (except for in the telemetry component) because they consisted of ~50% of the total catch of coho salmon. Although they have inherently different conservation value than do wild fish, the same post-release mortality rates are applied to wild and hatchery coho salmon for management purposes. Both sets of fish would have presumably migrated to sea from freshwater ~1.5 years earlier after either leaving natural rearing areas (wild) or having being released from (often adjacent) hatchery facilities.

Adipose fin tissue samples collected from coho salmon, for which mortality was assessed, were used for population origin identification via analysis of variation of 17 microsatellite loci (Beacham et al. 2011). Fish were identified to individual spawning streams, but spawning stream fidelity within subpopulations can be low in some years if low flows necessitate finding an alternate spawning site (R. Bailey, DFO, *personal communication*). For that reason and for statistical power, fish were categorized by major population groupings for analyses (Beacham et al. 2011).

Blood plasma samples were stored in an ice slurry for <1 h before they were centrifuged at 68646.55 m/s^2 (7000 × g) for 5 min, after which plasma was separated and stored in liquid nitrogen until later transfer to a −80°C freezer. Plasma was then analyzed in the laboratory for cortisol (enzyme-linked immunosorbent assay [Neogen, Lansing, Michigan, USA] with Spectramax 240pc plate reader [Molecular Devices, Sunnyvale, California, USA]), chloride (digital chloridometer [Haake Buchler Instruments, Paramus, New Jersey, USA]), sodium and potassium (model 410 single-channel flame photometer [Cole-Palmer North America, Vernon Hills, Illinois, USA]), osmolality (3320 freezing-point osmometer [Advanced Instruments, Norwood, Massachusetts, USA]), and lactate and glucose (2300 Stat Plus analyzer [YSI, Yellow Springs, Ohio, USA]) using methods described by Farrell et al. 2001.

24-h holding study

One of the two large, below deck holds on the *Franciscan No. 1* that is normally used for packing target catch on ice was used as a holding tank for ~24 h after capture to monitor short-term mortality. A ~24-h duration was chosen because it has been a common timeframe used in past studies of discard mortality for salmon fisheries (e.g., Farrell et al. 2001, Thomas and Cahusac 2012), and because longer durations of confinement would restrict the number of replicates of the holding experiment that could be completed (and thus the total sample) during our eight-day fishing charter.

TABLE 1. Dates, capture variables, coho salmon (*Oncorhynchus kisutch*) condition, and holding durations for each of the four replicates of the 2012 holding study.

Replicate	Date	Total catch for sets used (no. fish)	Net set water temperature (°C)	Time in recovery tote (min:s)		RAMP score	Injury score
				Mean	Range		
1	20–21 Aug	58, 20, 28	9.6, 9.7, 9.2	36:20	00:30–104:00	0.47 ± 0.19	0.14 ± 0.16
2	22–23 Aug	46	9.4	35:57	00:59–75:20	0.35 ± 0.12	0.23 ± 0.18
3	24–25 Aug	15, 50	9.2, 8.7	32:17	02:10–70:07	0.30 ± 0.20	0.20 ± 0.22
4	26–27 Aug	66	9.5	33:15	02:02–67:03	0.32 ± 0.14	0.24 ± 0.19
	Weighted mean	48.13	9.32		34:29	0.36	0.20

Notes: The holding duration varied based on when a fish was captured, processed, and the exact time when the holding experiment was terminated. For survival rates, moribund fish were included as mortalities (which were 3 of 10, 0 of 3, 3 of 9, and 1 of 3 total mortalities counted for each of the four replicates, respectively). Higher reflex action mortality predictors (RAMP) scores (shown as mean ± SD) correspond to fish in poorer condition, and simply represent the proportion of reflexes that were impaired (see *Methods* and Raby et al. 2012). Injury scores (shown as mean ± SD) were based on the extent of scale loss and the number of different injury types that occurred, with higher values indicating a fish in poorer condition (see *Methods*). Differences among replicates were statistically significant for RAMP score such that fish in replicate 1 of the experiment had significantly higher RAMP scores than in replicates 3 and 4 (Kruskal-Wallis ANOVA $P < 0.001$, post hoc differences assessed using multiple comparisons). Fish in replicate 1 were also held in the net pen for a longer duration, on average, than the other three replicates (based on Tukey HSD comparison, ANOVA $F_{3, 113} = 36.4$, $P < 0.001$). Differences among replicates for injury scores (Kruskal-Wallis ANOVA) and time in recovery totes (ANOVA) were not significant ($P > 0.10$ in both cases). The number of mortalities was not significantly different among the four replicates of the holding study (Chi-square test, $\chi^2 = 0.82$, df = 3, $P = 0.84$).

Only the area of the tank immediately below the 1.8 × 1.4 m hatch was used: This area was sectioned off by means of a net pen that could be easily drawn to the surface as needed to retrieve fish. The net pen was 1.8 m wide × 1.4 m long × 2 m deep and made from 70-mm diamond soft nylon mesh, within a tank that was approximately double the volume of the net pen. The tank was continuously flushed with fresh seawater using an on-board bilge pump, and pure oxygen was bubbled through a diffusion plate at the bottom of the net pen, as needed, to maintain saturation between 85–115% through the 24-h period. The tank was kept covered and in darkness during the entire holding period. Fishing operations were able to continue throughout the period while fish were held below deck. Seas were mostly calm during the eight-day study, and periodic visual inspection confirmed that the water in the tank remained hydrodynamically neutral while the boat rolled over waves, with no apparent swaying of fish within the tank. Overnight, the boat was anchored inside Port San Juan across from Port Renfrew (48°33′ N, 124°28′ W; Fig. 1). In that location, the water was visibly more turbid than in the Strait, ~2°C warmer, and likely contained some mixture of freshwater. The following day while in Juan de Fuca Strait (i.e., "fishing area" in Fig. 1), 24 h after the most recently tagged fish entered the tank, the fish in the holding study were removed individually by dip net and processed in the sampling trough.

All fish placed in the holding tank for monitoring were tagged with a uniquely numbered spaghetti tag (Floy Tag and Manufacturing, Seattle, Washington, USA) that was threaded through the dorsal musculature just posterior to the dorsal fin and tied with a double reef knot. Sixty (60) of 118 fish in the holding study also had "dummy" transmitters (8 mm diameter, 20.5 mm length, made from high density plastic) threaded onto the spaghetti tags to the test short-term retention of the transmitter to be used in the telemetry component. A subset of fish was blood sampled: 32 of 60 fish with dummy transmitters, and 33 of 58 with only spaghetti tags. Of the 118 fish that were used for the 24-h net pen experiment, 84 were wild fish (intact adipose fins), while the other 34 were hatchery-origin fish (adipose fin clipped). Post-24-h processing included a RAMP assessment, a blood sample for a subset of fish, removal of the spaghetti tag, and description of macroscopic condition, followed by release overboard after removal of any tags. The net pen experiment was replicated four times ($N =$ 36, 25, 32, and 25 salmon, respectively), each iteration comprising fish caught in three, one, two, and one net set(s), respectively (for each of the four rounds). Fish were taken from multiple net sets in some cases in order to increase sample sizes, which resulted in a range of durations of net pen confinement. The longest gap from first to last entry into the net pen (which was in round one of the holding study) was 7.5 h, such that the fish in that replicate group were held for 24–31.5 h depending on when they were caught and tagged relative to when the experiment was terminated (see Table 1 for further details on each replicate of the experiment). The overall mean duration of net pen monitoring (from tagging to retrieval) was 26.80 h, but the duration was significantly longer for the first iteration of the holding study than for the three other replicates, because fish were taken from three net sets (based on Tukey HSD post hoc comparisons after significant among-group differences for analysis of variance [ANOVA]; $F_{3, 113} = 36.4$, $P <$ 0.001; see Table 1).

Telemetry tracking

From 21–27 August (2012), 50 wild coho salmon were externally affixed with acoustic transmitters and immediately released to resume their migrations. We used a

TABLE 1. Extended.

Holding duration (h)		Survival	
Mean	Range	Rate (%)	No. fish
28.97	24.00–31.45	72	26 of 36
25.30	24.55–26.06	88	22 of 25
26.26	24.95–28.04	72	23 of 32
25.78	25.07–26.31	88	22 of 25
26.80		79	93 of 118

"backpack"-style attachment method with 8 mm diameter acoustic tags that transmitted their unique codes every 25–65 s (random time delay; V8-4X with pre-installed end caps, 30 mm length, 2.95 g in air; VEMCO, Bedford, Nova Scotia, Canada). The transmitters were threaded onto a spaghetti tag posterior to the dorsal fin such that they laid flush against the back of the fish, behind the dorsal fin (Fig. 3). This method of tagging required <30 s to complete, involved no anesthetic or suturing, and had no risk of regurgitation that can be a concern when gastrically tagging adult salmon that may continue feeding during the coastal approach to freshwater. The 50 coho salmon we tagged were selected at random from 21 different net sets, representing a range of post-capture recovery times, RAMP scores, and injury scores. Blood samples were not taken from telemetry-tagged fish to minimize handling and injury that could affect post-release mortality.

For this paper, the acoustic telemetry data were used solely to assess survival. Fish were classed as short-term survivors if they were detected by the Juan de Fuca (JDF) acoustic receiver line, which consisted of 30 receivers arranged in a straight line from Sheringham Point on the Canadian shoreline (48°22′ N, 123° 55′ W) to Pillar Point on the American shoreline (48°13′ N, 124°6′ W; Fig. 1). The JDF line was en route to spawning areas for all populations of coho salmon we encountered, except for two fish DNA-identified to a coastal Washington (USA) stream (Clearwater Creek). Those two fish were nevertheless detected on the JDF line and classified as short-term survivors. On average, the JDF line was 32 km from the release site to the first receiver at which fish were detected (range: 25–41 km), and thus represented an appropriate short-term migration checkpoint. That receiver line appeared to have 100% detection efficiency because all of the fish detected from this study were detected on multiple receivers on the line, often by two or more receivers simultaneously, and no fish detected at subsequent receivers was undetected there.

Long-term survival was assessed for 43 telemetry-tagged fish whose migration pathway toward natal tributaries (identified via DNA analysis) had appropriately placed receiver infrastructure. For Puget Sound-origin coho salmon, long-term survival was based on success moving past a 13-receiver array that crossed Admiralty Inlet (between 48°4′ N, 122° 40′ W and 48°4′ N, 122°37′ W; Fig. 1), the entrance to Puget Sound. Out of 14 coho salmon we tagged that were detected on various receivers in Puget Sound beyond the Admiralty line, three were never detected on the Admiralty line (21.4%). Thus, detection efficiency for that line was 78.6%. That detection efficiency was used as a correction factor for estimating the total number of individuals that were successful in reaching the Admiralty receiver line (i.e., long-term survival for Puget Sound stocks). In addition, two Puget Sound fish were detected on receivers in the San Juan Islands, but not elsewhere beyond the JDF line. Those fish were

FIG. 3. Photo depicting the external "backpack" acoustic transmitter attachment method used for this study. The spaghetti tags and dummy transmitters used in the net pen holding study were attached in the same way.

included as long-term survivors. For Fraser River stocks, long-term survival was assessed based on detection in the Fraser River. All fish detected in the Fraser River progressed upstream past all four receiver arrays there, with the upstream-most receiver array ~70 river km upstream of river entry, at Mission (49°7′ N, 122°18′ W). We assumed a 100% detection efficiency for the lower Fraser River lines because each fish was detected on all four lines. The few fish from other spawning populations (e.g., Vancouver Island, coastal Washington) were not included in analysis of long-term survival due to lack of appropriately placed receivers along their presumed migration pathway, beyond the JDF line.

Data analysis and statistics

Principal components analysis (PCA) was used to distill the original variables into one or more synthetic variables (factors) for further analyses. Initially, a PCA was run using all blood samples (at-capture and post-24 h, $N = 231$). From that initial PCA, variables were successively removed and the analysis rerun if (1) a variable had a Kaiser-Mayer-Olkin (KMO) measure of sampling adequacy of <0.5 (Field et al. 2012), or (2) a plasma variable's only factor loading >|0.6| was for a factor that had no >|0.6| factor loadings from other variables (and thus, that factor would largely be replicating one of the original variables rather than synthesizing two or more). Scores from the resulting factor (PC1, un-rotated) along with four original plasma variables not included in the final PCA were compared between at-capture and post-24 h using Welch's t test. We similarly used t tests and Kruskal-Wallis ANOVA to explore whether there were effects of the following at-capture variables on post-24-h physiology: loss of orientation (0/1), attachment of dummy transmitter (0/1), caudal puncture blood biopsy (0/1), and injury score (0/0.2/0.4). Loss of orientation was used in place of RAMP score for comparisons of 24-h blood data because it was shown to be particularly influential in 24-h mortality, whereas RAMP score was not (see *Results*). Since we ran each of the above tests with five separate blood variables, α was set at a conservative 0.01.

The plasma variables we measured change along a predictable trajectory following exhaustive exercise stress (e.g., Wood et al. 1983, Milligan et al. 2000, Marcalo et al. 2006), and fish in this study were sampled after varying amounts of time in the recovery tote after capture. Thus, there was a need to control for the effect of sampling time on blood physiology (see Cooke et al. 2013*a*) to assess the influence of other variables such as RAMP or injury scores, which did not change over time in the recovery tote (see *Results*, below). To do so, each of the five plasma variables (PC1 scores, plasma lactate, cortisol, potassium, and glucose) was regressed using linear regression against time spent in the recovery tote prior to blood sampling (Fig. 4). For regressions that were significant, the residuals were used for further analyses of at-capture blood physiology in place of the raw values. To assess relationships between RAMP and injury scores and the five physiological responses (time controlled) we used Kruskal-Wallis ANOVA. If Kruskal-Wallis ANOVA was significant ($\alpha = 0.01$), we assessed directional trends across groups using Spearman rank-order correlation and the Jonckheere-Terpstra test ($\alpha = 0.05$). The Jonckheere-Terpstra test is analogous to Kruskal-Wallis ANOVA except groups are ordinal such that the test evaluates whether the order of group medians is meaningful (Field et al. 2012). Because the combined condition score involved up to 10 condition levels, only Spearman rank-order correlations were used to assess its relationship with plasma variables.

Predictors of post-release mortality were assessed using forced entry logistic regression. For the on-board holding experiment, we ran two models. One model used only the five physiological variables (time controlled) for the subset of fish that were blood sampled prior to the holding study ($N = 63$), and a second model with seven non-blood variables ($N = 118$): whether a blood sample was taken at capture (0/1), whether a dummy transmitter was attached (0/1), fork length (cm), time spent in the recovery tote (s), injury score, RAMP score, and loss of orientation at capture (0/1). Similarly, two models were used for post-release mortality among telemetry-tagged fish: one for short-term mortality ($N = 50$) and one for long-term mortality ($N = 43$). Those models used the same predictor variables as those used to model 24-h net mortality, except that the effects of blood sampling and dummy transmitters were not included (not applicable). All statistical procedures were completing using R (v. 3.0.2; R Development Core Team 2013).

Results

Catch characteristics

Total catch of salmon for individual purse seine sets was consistently low (mean = 37 adult salmon using the standard netting, range = 0–82 fish), but coho salmon did make up 83% of this total (approximately one-half were hatchery origin). The remaining Pacific salmon were sockeye (10% of catch), Chinook *O. tshawytscha* (6%), chum salmon *O. keta* (<0.5%), and pink salmon (<0.5%). Of the 137 wild coho salmon for which we determined population origin (using DNA), 67% belonged to populations in the Puget Sound (Washington, USA) basin and only 8% were identified to the interior Fraser River watershed populations (10% to the Fraser River overall). RAMP scores (Kruskal-Wallis ANOVA, $\chi^2 = 4.7$, df = 5, $P = 0.46$), injury scores ($\chi^2 = 4.8$, df = 5, $P = 0.44$), and fork length ($\chi^2 = 10$, df = 5, $P = 0.08$) were not significantly different among major population groupings (detailed population origin results available in the Appendix).

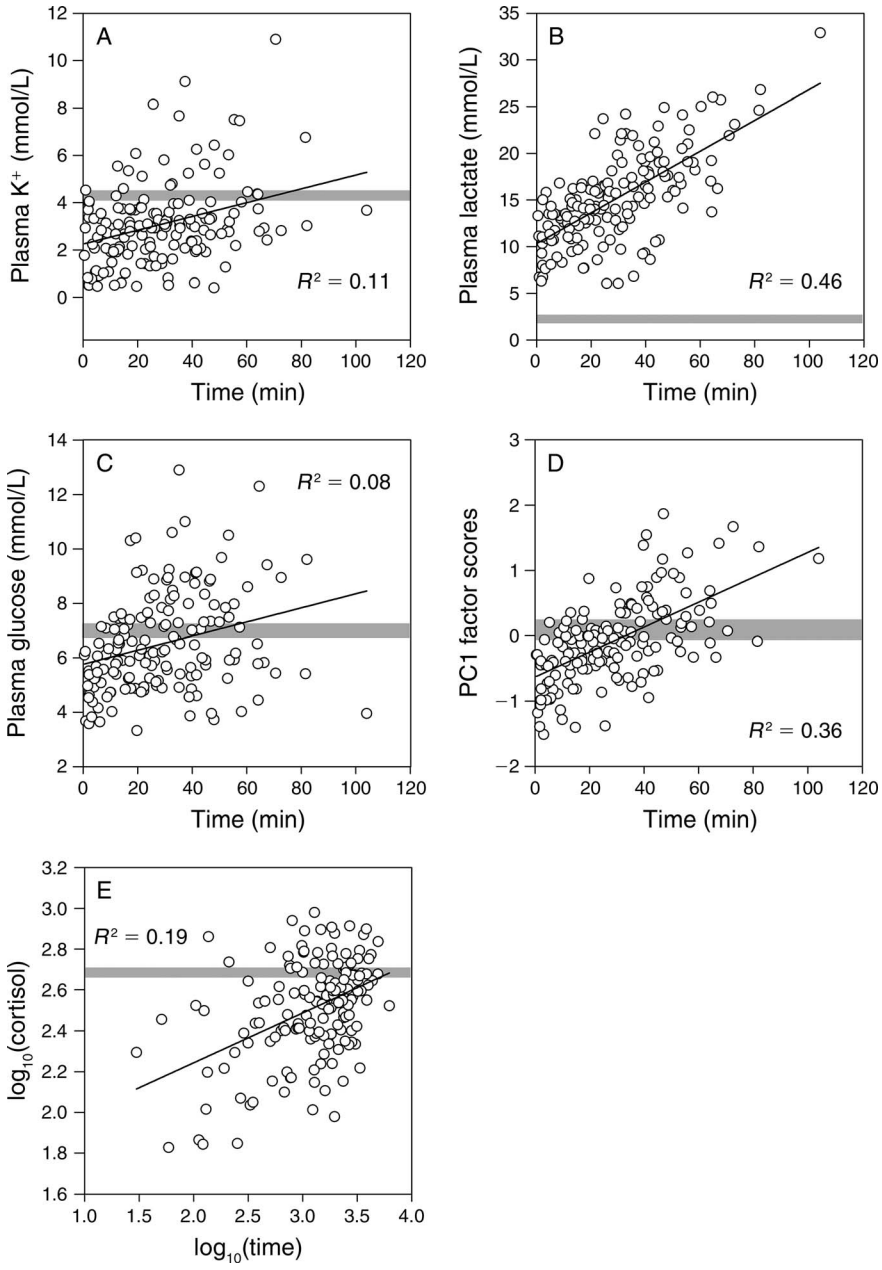

FIG. 4. Linear regressions between five plasma variables in purse-seined coho salmon and the time elapsed from capture (and transfer to a recovery tote) to blood sampling ($N = 157$ for each regression). PC1 score is a principal component variable that represents chloride, sodium, and osmolality (all positively correlated with PC1; see Table 3). The shaded area represents mean (±SE) values from fish sampled after being held 24 h in the below-deck holding tank ($N = 59$). The residuals of each of these regressions were used for subsequent statistical analyses to control for the effect of sampling time, except in the case of potassium where the assumption of heteroscedasticity could not be met. Each regression was significant ($P < 0.001$), and the lines were described by (with standard error of the slope): (A) $[K^+] = [4.9\times10^{-4}\ (1.1\times10^{-4}) \times \text{time (s)}] + 2.25$; (B) [lactate] = $[2.8\times10^{-3}\ (2.4\times10^{-4}) \times \text{time (s)}] + 1.03$; (C) [glucose] = $[4.3\times10^{-4}\ (1.1\times10^{-4}) \times \text{time (s)}] + 5.76$; (D) PC1 score = $[3.2\times10^{-4}\ (3.4\times10^{-5}) \times \text{time (s)}] + -0.63$; and (E) $\log_{10}(\text{cortisol}) = [0.24\ (0.04) \times \log_{10}(\text{time})] + 1.75$.

Bycatch mortality estimates by method

The immediate bycatch mortality rate (i.e., died during capture or in the revival totes) for coho salmon was 2% (15 of 673 fish). The ~24-h mortality rate for fish held in the on-board tank was 21% overall (25 of 118; Table 2), which included seven fish categorized as moribund at 24 h. Moribund fish were characterized by fungus-infected eyes that caused loss of the vestibular–ocular response reflex (and likely blindness; 6 of 7 cases) and some loss of other reflexes, or extreme lethargy

TABLE 2. Statistical output for logistic regression predicting ~24-h mortality (see Table 1) in the on-board net pen holding experiment using data for all fish held in the study ($N = 118$; top) and for a second model predicting survival using blood physiology responses (blood values corrected for sampling time course by using the residuals of their relationships with time; see Fig. 4; $N = 63$, bottom).

Predictor variable	β (SE)	95% CI for odds ratio		
		Lower	Odds ratio	Upper
Predicting mortality using non-blood variables				
Constant	−6.96 (4.15)			
Recovery tote time (s)	2.6×10^{-4} (2.0×10^{-4})	1.00	1.00	1.00
Fish size (FL, cm)	0.07 (0.07)	0.94	1.07	1.23
Injury score	4.68** (1.57)	5.77**	108**	3146**
RAMP score	−1.65 (2.12)	0.003	0.19	14.2
Orientation reflex	3.33** (1.00)	4.22**	27.9**	225**
Caudal sampling	0.49 (0.60)	0.51	1.62	5.44
Dummy transmitter	−0.86 (0.61)	0.12	0.42	1.34
Predicting mortality only using blood variables				
Constant	−1.47 (0.71)			
PC1	1.06 (0.73)	0.74	2.91	13.9
Lactate	−0.06 (0.12)	0.74	0.94	1.18
Cortisol	−0.66 (0.78)	0.10	0.52	2.34
Potassium	1.1×10^{-4} (0.19)	0.67	1.00	1.43
Glucose	−0.67* (0.30)	0.27	0.51	0.87

Note: Both models were significant overall: top, $\chi^2 = 40.1$, df = 7, $P < 0.001$, Nagelkerke $R^2 = 0.45$; and bottom, $\chi^2 = 11.3$, df = 4, $P = 0.045$, $R^2 = 0.25$.
* $P < 0.05$; ** $P < 0.01$.

characterized by complete loss of reflex actions. The logistic regression model predicting mortality in the net pen experiment that included all fish and excluded plasma variables explained more of the variation in mortality ($N = 118$, $\chi^2 = 40.1$, df = 7, $P < 0.001$, Nagelkerke $R^2 = 0.45$) than that which used only plasma variables ($N = 63$, $\chi^2 = 11.3$, df = 4, $P = 0.045$, $R^2 = 0.25$). Injury score ($P = 0.003$) and loss of the orientation reflex ($P < 0.001$; but not RAMP score) were both positive predictors of mortality within the main model (Table 2). The amount of time spent in the on-board recovery tote was not a significant predictor of survival. Among plasma variables, only glucose was predictive of 24-h mortality; higher glucose at capture (controlling for sampling time) was associated with lower mortality ($P = 0.02$; Table 2). The number of mortalities was not significantly different among the four replicates of the holding study (Chi-square test, $\chi^2 = 0.82$, df = 3, $P = 0.84$; Table 1).

Short-term post-release mortality of coho salmon (~48–96 h) based on telemetry tracking was 20% (10 of 50 fish were undetected on the JDF receiver line). The long-term mortality rate was 47% (20 of 43 failed to pass receiver lines in Puget Sound or the Fraser River). Among the five interior Fraser coho salmon that were telemetry tagged, four (80%) successfully reached the Fraser River and progressed upstream past all receivers there. Small sample sizes precluded statistical assessment of whether survival was different among populations. Logistic regression models predicting short-term (Nagelkerke $R^2 = 0.25$, $P = 0.14$) and long-term mortality ($R^2 = 0.21$, $P = 0.20$) for telemetry-tagged fish were not significant.

Blood physiology at capture and after 24-h confinement

PCA revealed very strong positive associations among plasma chloride, sodium, and osmolality in the overall dataset of seven plasma physiology variables (Table 3). Low KMO statistics (<0.5) and lack of sufficient shared variation within a factor among the other plasma variables meant that they were successively eliminated from the PCA until only three variables remained. Thus, the final PCA produced a single principal component (PC1) variable that was used in further analyses as an integrator of the strong shared variation (Table 3) between chloride, sodium, and osmolality. The other four plasma variables were used separately for further analyses.

There were some notable changes in plasma physiology in coho salmon following a ~24-h post-capture on-board net pen confinement (shaded areas in Fig. 4). Cortisol was significantly elevated after 24 h (541 ± 31 ng/mL) compared with the post-capture blood samples (331 ± 17 ng/mL; Welch's t test using log-transformed data; $t_{142} = -6.6$, $P < 0.001$; Fig. 4E). Circulating plasma lactate was significantly lower after 24 h in coho salmon (2.3 ± 0.16 mmol/L) than in the minutes following capture (13.8 ± 0.42 mmol/L; $t_{134} = 25.8$, $P < 0.001$; Fig. 4B). Log-transformed plasma glucose ($t_{134} = 25.8$, $P < 0.001$) and potassium ($t_{160} = -6.3$, $P < 0.001$) were also significantly elevated at 24 h relative to post-capture samples, while PC1 scores were not

TABLE 3. Output of the initial principal components analysis (PCA) including all seven blood variables (top) and the final analysis (bottom) used to synthesize chloride, osmolality, and sodium into a synthetic (PC1) variable for further analyses of the effects of capture on physiology ($N = 231$).

Plasma variable	PC1 loading	PC2 loading	PC3 loading	Communality, h^2
Overall PCA				
Eigenvalue	2.95	1.47	1.03	
Variance explained (%)	42%	21%	15%	
Cortisol	0.48	0.56	0.15	0.63
Glucose	0.39	0.17	**0.74**	0.87
Lactate	0.16	**−0.78**	0.47	0.92
Chloride	**0.88**	0.14	−0.37	0.94
Osmolality	**0.95**	−0.24	0.00	0.96
Sodium	**0.91**	−0.27	−0.18	0.93
Potassium	0.22	**0.61**	0.26	0.99
Final principal component used for subsequent analyses				
Eigenvalue	2.66			
Variance explained (%)	89%			
Chloride	**0.92**			0.84
Osmolality	**0.96**			0.91
Sodium	**0.95**			0.91

Note: The PCA was successively run with stepwise elimination of variables based on plasma variables having either (1) a low KMO statistic, or (2) not having a loading $>|0.6|$ (shown in boldface type) for any factor which also had other $>|0.6|$ loadings (i.e., not agreeing strongly with other variables within a factor).

significantly different between capture and 24-h samples ($t_{160} = -1.1$, $P = 0.30$; Fig. 4).

Post-24-h blood physiology was not significantly different between fish that did and did not have an attached dummy transmitter (Welch's t test; all $P > 0.10$) and was not affected by whether the fish had been previously blood sampled (all $P > 0.20$). Log-transformed plasma lactate was significantly higher at 24 h for fish that had lost orientation at capture than in fish that had not ($t_{16} = -5.3$, $P < 0.001$), but no other significant differences in 24-h blood physiology were predicted by loss of orientation (all $P > 0.20$). PC1 scores were significantly different among injury scores after 24 h (Kruskal-Wallis ANOVA; $\chi^2 = 13$, $P = 0.002$) with post hoc tests indicating significant elevation for PC1 at 0.4 relative to fish with an injury score of 0 at capture. The other physiological variables were not significantly different among injury scores (all $P > 0.01$).

Time from landing to blood sampling (i.e., time spent in the recovery tote) had a significant positive effect on each of the five plasma variables (Fig. 4). However, the relationship with potassium could not be transformed to meet both the assumptions of heteroscedasticity and normality, so that regression was not used for further analyses. For the other four plasma variables, the residuals of their regressions with sampling time (Fig. 4) were used for further analyses, as a means of controlling for the effect of sampling time.

Relationships among sublethal measures

RAMP and injury scores were positively correlated (Spearman's rank-order correlation, $r_S = 0.27$, $P < 0.001$). Catch size was positively correlated with RAMP score ($r_S = 0.36$, $P < 0.001$), injury score ($r_S = 0.19$, $P = 0.02$), and the combined condition score ($r_S = 0.35$, $P < 0.001$). Conversely, time spent in the recovery tote after capture did not correlate significantly with RAMP score, injury score, or the combined condition score (all $P > 0.20$). Moreover, for individual reflexes there was no apparent change over time: The duration of recovery in the revival totes did not differ significantly between fish with and without each of the reflexes (Welch's t test, all $P > 0.25$). RAMP score had a significant positive relationship with the plasma PC1 response (i.e., controlling for sampling time; see Fig. 4), plasma lactate response, and a significant negative relationship with plasma cortisol response (Table 4, Fig. 5). Similarly, there was a significant positive trend in the PC1 response relative to injury scores (Table 4, Fig. 6). The combined condition score (RAMP + injury) was positively correlated with PC1 and lactate responses and negatively correlated with the cortisol response (Table 4, Fig. 7), but there was no pattern with the other blood variables measured.

Overall, fork length was negatively correlated with injury score (Spearman correlation, $r_S = -0.18$, $P = 0.03$) and RAMP score ($r_S = -0.29$, $P < 0.001$). There was no correlation between fork length and time-corrected cortisol ($r_S = 0.15$, $P = 0.06$), or potassium ($P = 0.15$), but significant negative correlations did occur between fork length and lactate ($r_S = -0.20$, $P = 0.01$), glucose ($r_S = -0.17$, $P = 0.03$), and the PC1 response ($r_S = -0.29$, $P < 0.001$).

TABLE 4. Statistical relationships among sublethal measures: injury, reflex impairment, and blood physiology.

Plasma variable	Direction of relationship	Statistical test: Kruskal-Wallis test χ^2	Kruskal-Wallis test P	Jonckheere-Terpstra test, P	Spearman rank-order correlation r_S	Spearman rank-order correlation P
RAMP score						
PC1	positive	18	<0.001	0.013	0.34	<0.001
Lactate	positive	31	<0.001	0.013	0.42	<0.001
Cortisol	negative	15	0.002	0.013	−0.27	<0.001
Glucose	none	18	<0.001	0.05	0.15	0.06
Potassium	none	2	0.57	n/a	n/a	
Injury score						
PC1	positive	11	0.009	0.027	0.27	<0.001
Lactate	none	2	0.58	n/a	n/a	n/a
Cortisol	none	6	0.13	n/a	n/a	n/a
Glucose	none	1	0.83	n/a	n/a	n/a
Potassium	none	3	0.38	n/a	n/a	n/a
Combined condition score						
PC1	positive	n/a	n/a	n/a	0.38	<0.001
Lactate	positive	n/a	n/a	n/a	0.30	<0.001
Cortisol	negative	n/a	n/a	n/a	−0.30	<0.001
Glucose	none	n/a	n/a	n/a	0.07	0.38
Potassium	none	n/a	n/a	n/a	−0.11	0.18

Notes: Blood physiology measures tested were the residuals of regressions with sampling time to correct for the confounding effect of the sampling time course. Note that this only involves using RAMP scores and injury scores from 0–0.6 (equaling four groups). If the Kruskal-Wallis test was not significant ($P > 0.05$), the Jonckheere-Terpstra test and Spearman rank-order correlation tests were not conducted (n/a represents not applicable). Total $N = 153$.

DISCUSSION

The present study is among the first to directly compare the use of short-term captivity with biotelemetry as methods for estimating post-release bycatch mortality. The short-term mortality rates observed in the net pen experiment and acoustic telemetry component were similar: 21% and 20%, respectively. However, while the net pen experiment lasted an average of 26.8 h, the telemetry component involved monitoring survival over distance along the migration route, rather than time. The time from release to arrival at the first receiver line (JDF; Fig. 1) was highly variable (mean = 125 h, median = 95 h, min = 14 h, max = 587 h), and 32 of 40 fish required >48 h. Thus, although the two mortality estimates appear quite similar, the short-term mortality estimate for telemetry-tagged fish represented a considerably longer monitoring period in almost all cases. The long-term mortality rate for telemetry-tracked fish, which represented a period of 10–20 days in most cases, was 47%. That longer duration meant that there was increased potential for latent pathogenic mortality to occur via injury (although there was no significant link between injury and mortality) and that some natural mortality would likely have occurred. In the discussion that follows, we attempt to make connections between our novel physiological findings and the RAMP, injury, and mortality patterns we observed in the two mortality experiments.

Predictors of post-release mortality

Mortality could be predicted by injury and reflex impairment in the more controlled net pen experiment, whereas there were no statistically significant predictors of mortality in the telemetry experiment with a smaller sample size (Table 2, Fig. 8). In the net pen, higher injury scores were positive predictors of mortality, consistent with our expectations and a number of past studies that demonstrate dermal injury is detrimental to fish (e.g., Kaimmer and Trumble 1998, Olsen et al. 2012). Reflex impairment was predictive of mortality, but it was the orientation reflex on its own that was a strong predictor rather than the full suite of five reflexes included in RAMP score. RAMP–mortality curves are often sigmoidal in shape whereby reaching a threshold score, often associated with the addition of a particular reflex (Fig. 8D), causes a large increase in mortality (Davis 2007). The fact that neither injury nor RAMP scores were predictive of mortality for telemetry-tagged fish was surprising, as it suggests that mortality was not influenced by either (1) the extent of departure from homeostasis, or (2) the extent of dermal injury. In light of a number of other studies, our results show that RAMP may fail to predict delayed mortality in some contexts and further work will be needed to understand why this is the case. However, we caution that, because few fish in the telemetry component (8 of 50) experienced high RAMP scores (≥0.6), there was limited power to calibrate RAMP to post-release outcomes,

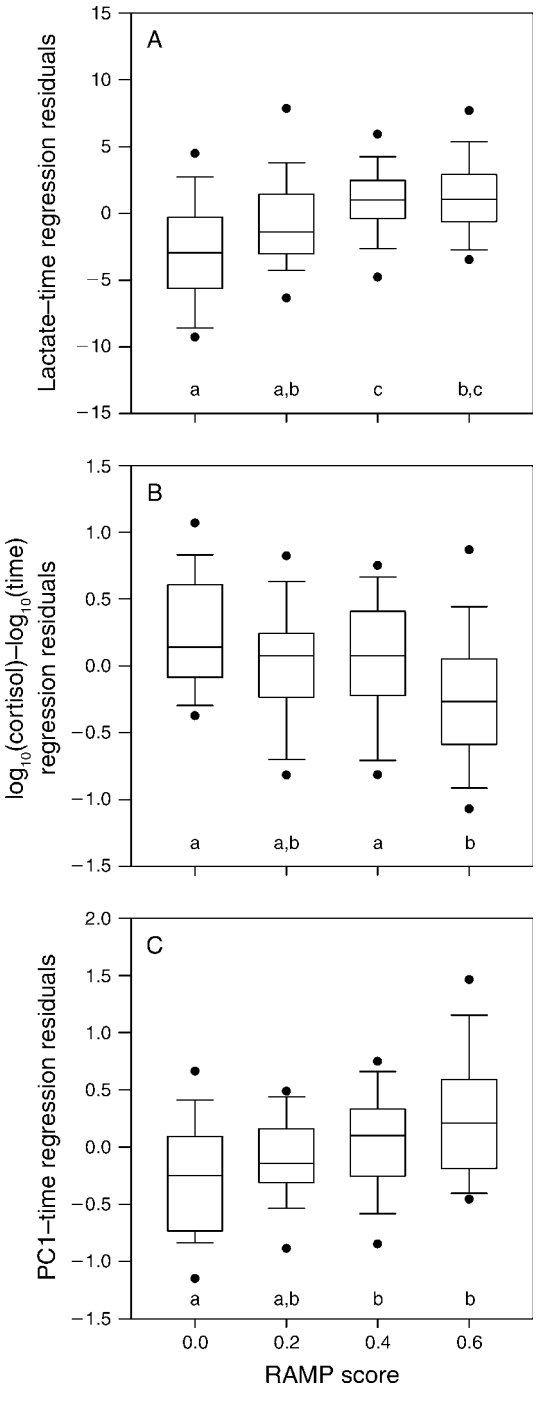

FIG. 5. Differences in physiological responses among fish assigned different reflex action mortality predictors (RAMP) scores (see Table 4 for statistics), with statistically significant post hoc differences indicated by dissimilar letters under the boxplots. The *y*-axis values represent blood plasma concentrations of (A) lactate, (B) cortisol, and (C) a combination of sodium, chloride, and osmolality (the PC1 variable; Table 4) corrected for the time point at which they were sampled after capture, using the residuals of a regression with sampling time (Fig. 4). Higher RAMP scores correspond to fish in poorer condition, and simply represent the proportion of reflexes that were impaired (see *Methods* and Raby et al. 2012). The line within each box represents the median, the upper and lower bounds of each box are the 75th and 25th percentiles (respectively), and the whiskers represent the 10th and 90th percentiles of the data. Beyond the whiskers, outliers are shown.

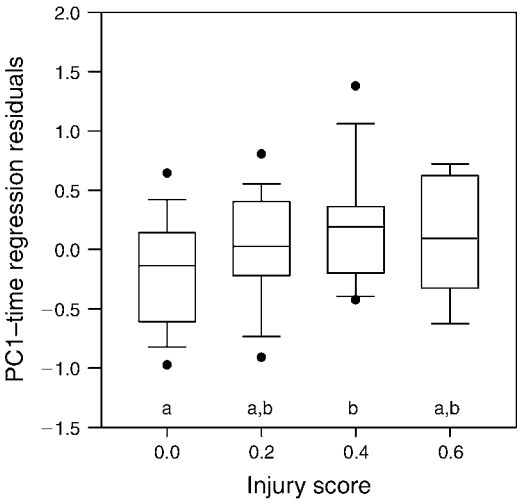

FIG. 6. Differences in the PC1 (i.e., a synthetic variable that combined plasma sodium, chloride, and osmolality; see Table 3) response of coho salmon assigned different injury scores (for statistics, see Table 4). Statistically significant post hoc differences are indicated by dissimilar letters under the boxplots. Injury scores were based on the extent of scale loss and the number of different injury types that occurred, with higher values indicating a fish in poorer condition (see *Methods*). See Fig. 5 for a definition of the boxplots.

especially given that mortality was strongly associated with RAMP scores $\geq$0.6 in the net pen experiment and for coho salmon bycatch in freshwater (Raby et al. 2012). In a future study with a larger sample size and a greater range of stressor severity and RAMP scores (e.g., in a real fishery with thousands of fish caught rather than dozens), it is entirely possible a strong RAMP–mortality relationship would occur in telemetry-tagged fish. Regardless, this and other studies on RAMP in Pacific salmon show that it is an effective approach for rapidly assessing animal vitality (Davis 2007, Donaldson et al. 2012, Raby et al. 2012, 2013).

Mechanisms of reflex impairment and mortality

RAMP scores in coho salmon bycatch were clearly mirrored by certain plasma variables, providing confirmation that a simple reflex assessment can be used to assess the extent of physiological departure from homeostasis in bycatch. In particular, RAMP score was positively correlated with plasma lactate and the PC1 response (i.e., plasma sodium, chloride, and osmolality). Previous work on chum salmon and pink salmon also showed that plasma lactate increases at higher RAMP scores (Raby et al. 2013). However, that study involved discrete experimental groups with controlled durations of exercise and air exposure, rather

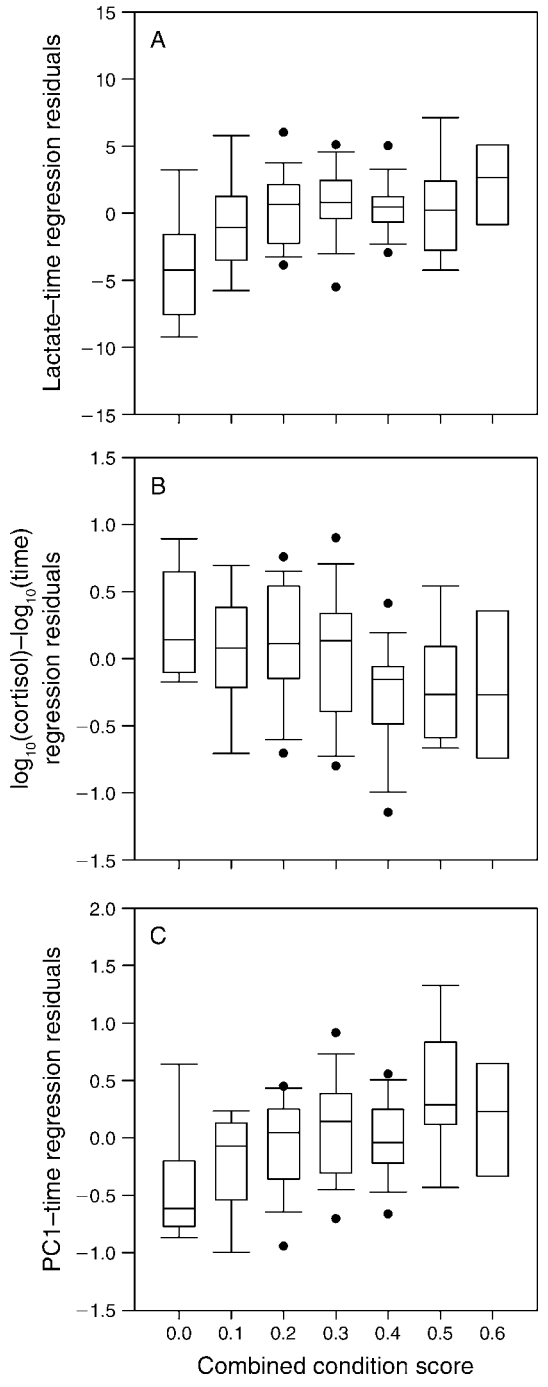

FIG. 7. Differences in physiological responses across combined condition scores, which represent a simple combination of RAMP and injury scores (see Table 4 for statistics, and Figs. 5 and 6 for clarification scores). The *y*-axis values represent blood plasma concentrations of (A) lactate, (B) cortisol, and (C) a combination of sodium, chloride, and osmolality (the PC1 variable; Table 4) corrected for the time point at which they were sampled after capture, using the residuals of a regression with sampling time (Fig. 4). See Fig. 5 for a definition of the boxplots.

than the continuous range of stressor intensity that occurs in a real fishery. Our results are also supported by a recent study on silver seabream (*Pagrus auratus*) in which higher reflex impairment scores were reflected by greater accumulation of blood lactate and depressed muscle pH (McArley and Herbert 2014). In the present study, significant elevations in the PC1 and lactate responses of fish were both likely the result of excessive anaerobic metabolism in the white muscle. Lactate produced in muscle cells is partly extruded into blood, and the osmotic pressure created by intracellular metabolic acidosis causes water to move from plasma into white muscle cells, thereby increasing the concentration of ions in plasma (i.e., the PC1 response; Wood et al. 1983). In salmon, a RAMP score of 0.4 almost always represents the combined loss of the tail grab and body flex responses (Raby et al. 2012, 2013); two responses that, when positive, involve use of white muscle to burst forward in water (tail grab) or struggle free of the handler in air (body flex). RAMP scores of 0.6 (or higher) virtually always reflect the additional impairment of the orientation response (as we found, and Raby et al. 2012, 2013). Increases in plasma lactate (Raby et al. 2013) and PC1 response appear to occur up to a RAMP score of 0.4 but not beyond (Fig. 5), suggesting the mechanism for loss of body flex and tail grab reflexes is metabolic acidosis and exhaustion of white muscle, whereas orientation (RAMP score of 0.6) becomes impaired via another pathway and requires a more severe capture stressor. Loss of orientation may be analogous to fainting in humans (i.e., syncope), a loss of consciousness typically caused by insufficient oxygen delivery to the brain (i.e., cerebral hypoxia or hypoperfusion; Low and Mathias 2011). Exhaustive exercise during net hauling and asphyxiation via entanglement or air exposure could, when combined, lead to a drop in the oxygen partial pressure of the blood sufficiently large that could, in turn, cause loss of consciousness (i.e., loss of orientation).

Given that the orientation reflex was predictive of mortality in the net pen, we assessed whether it was associated with patterns in blood physiology after 24 h. Our data provide support for the connection between loss of orientation and mortality based on the novel finding that lactate was higher after 24 h for fish that had lost orientation at capture (but survived 24 h), representing a departure from homeostasis extreme enough that metabolic recovery was impaired. In addition, cortisol was very high for fish in the net pen (Fig. 4), and elevated cortisol has previously been associated with slowed clearance of lactate in exercised fish (Milligan et al. 2000). The 24-h elevation in lactate we observed for fish recovering from loss of orientation may not have occurred in fish that were telemetry-tagged and released because those fish did not experience confinement stress. This key difference between the net pen and immediately released fish could explain why orientation predicted mortality in one and not the other. However, small

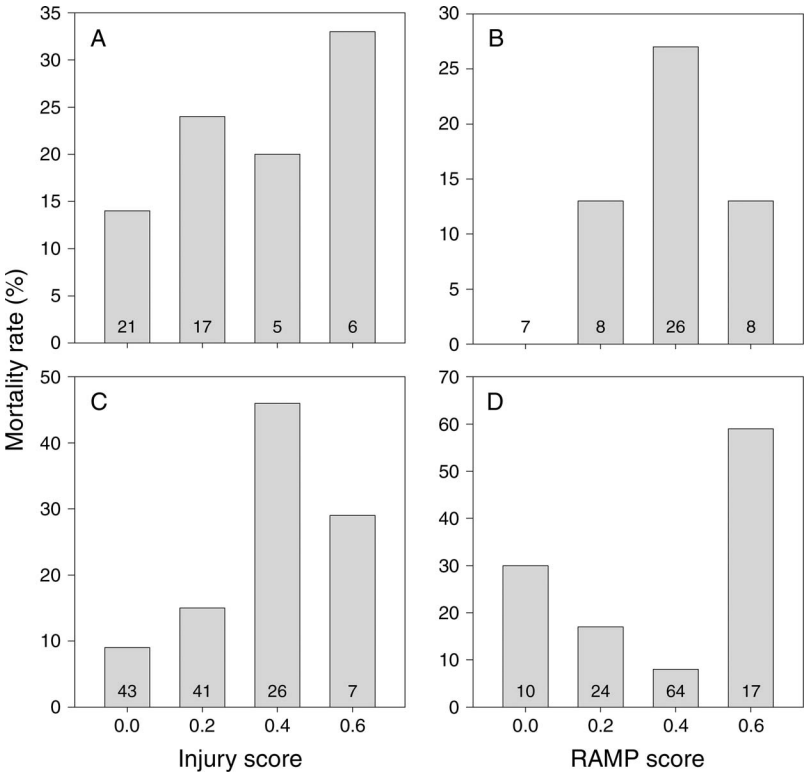

FIG. 8. Mortality rates specific to fish assigned different injury scores in the (A) telemetry study and (C) net pen experiment and specific to different RAMP scores: (B) telemetry and (D) net pen. The numbers in each bar represent the total number of fish assigned that condition score. RAMP and injury scores represent proportions, whereby higher scores denote fish in poorer condition (i.e., more reflexes impaired or more injury).

numbers of fish at higher RAMP scores precluded a rigorous calibration between RAMP and mortality for telemetry-tagged fish.

Perplexingly, plasma cortisol was lower for fish that lost orientation (i.e., RAMP score of 0.6; Fig. 5) than for vigorous fish (RAMP score of 0). This result is contrary to our expectation that the same processes that result in stress and the release of cortisol during fisheries capture would also cause reflex impairment. In reality, however, all fish in this study were clearly quite stressed, and it is unclear whether a particularly elevated cortisol response after capture would have been adaptive or maladaptive for the fish (but see Cook et al. 2014). In addition, a caveat with respect to plasma cortisol is that sex has a strong influence on circulating cortisol in migrating adult Pacific salmon (Hinch et al. 2006, Baker and Vynne 2014), and sex was not identified for the fish in this study (i.e., they were morphologically indistinguishable and we did not measure sex steroids). Mature female Pacific salmon have higher baseline cortisol than do males (Sandblom et al. 2009, Donaldson et al. 2010, Raby et al. 2012, Baker and Vynne 2014) because of cortisol's role in mediating other sex hormones that drive reproductive maturation and egg production (Hinch et al. 2006, Baker et al. 2013). If the male coho salmon in our study were, because of a morphological or behavioral difference, more prone to severe net entanglement and associated exhaustive exercise, a greater number of males would have been assigned RAMP scores of 0.6, lowering the mean cortisol concentration for that RAMP score. Future studies on reproductively maturing Pacific salmon should ensure that sex is identified (e.g., via sex steroid analysis) and controlled when using cortisol as an indicator of stress.

In the present study, injury score was an index that incorporated both the extent of scale loss and the number of different injuries occurring for a single individual (i.e., gillnet-like marks on the head, the body, or other injuries). Injury score had a positive, medium-strength correlation with RAMP score, perhaps because fish exposed to injurious entanglement are likely to struggle more vigorously, become more exhausted, and thus suffer greater reflex impairment. Among blood physiology variables, injury score was only significantly associated with the PC1 response (Fig. 6) with a significantly elevated response at an injury score of 0.4 (e.g., moderate scale loss + gilling marks on body) relative to a score of 0 (i.e., low or no scale loss, no notable injuries). The relationship between injury score and PC1 response was weaker and less linear than its RAMP equivalent, suggesting it is likely indirect and not causal. However, there is a physiological basis and

evidence in the literature to expect that dermal injury could cause latent problems (Gadomski et al. 1994, Zydlewski et al. 2010, Olsen et al. 2012); including signs of osmoregulatory distress we observed in our study whereby PC1 scores remained elevated after 24 h for fish with higher injury scores at capture. Similar evidence exists that scale loss causes increases in plasma ions over a period of days in Atlantic herring (*Clupea harengus*; Olsen et al. 2012) and outmigrating Atlantic salmon smolts (*Salmo salar*; Zydlewski et al. 2010) in seawater. The mechanism for this phenomenon is not clear, but likely includes direct loss of body water via the compromised integument, although research on herring suggests water is lost directly from the gills because of a secondary stress response caused by scale loss (Olsen et al. 2012). Injury score predicted mortality in the net pen, and the 24-h plasma data suggest that failure to restore osmoregulatory homeostasis, as a result of dermal injury, may have contributed to that mortality. Adult Pacific salmon remodel their osmoregulatory physiology during their approach to freshwater (Cooperman et al. 2010), and any latent and sustained effects of scale loss on osmoregulation during this period could therefore be detrimental to migration success. Loss of osmoregulatory capacity is also a precursor to pre-spawn mortality for maturing sockeye salmon held in freshwater tanks, where large decreases in plasma chloride, sodium, and osmolality (i.e., PC1 in our study) precede death (Jeffries et al. 2011).

As with the orientation reflex, the fact that injury predicted mortality in the net pen, but not in telemetry-tagged fish could be related to confinement stress. Plasma cortisol was remarkably high after 24 h of net pen holding (Fig. 4E); higher even than in post-capture samples that were drawn from fish at a time point when cortisol typically peaks in the stress response of fish (i.e., within two hours of initiation of the stressor; Mommsen et al. 1999). Cortisol has a role in regulating gill ATPase activity, and is considered a saltwater adapting hormone because cortisol treatment has been shown to promote saltwater tolerance (Clarke and Hirano 1995, Mommsen et al. 1999). Olsen et al. (2012) point out that sustained cortisol elevation caused by scale loss could therefore be explained by fish attempting to regain osmoregulatory control, but if left unconstrained, such a response could initiate a lethal spiral via impaired immune function and accelerated depletion of glucose stores. However, in our study, cortisol was not significantly higher after 24 h for fish with higher injury scores. On its own, cortisol elevation caused by crowding stress in aquaculture facilities can cause skin damage and infections in fish (Iger et al. 1995, Udomkusonsri and Noga 2005). It is possible that in our net pen experiment, a magnified and prolonged cortisol response (caused by confinement) worsened existing injuries in ways that did not occur for the fish we released overboard with transmitters. In the net pen, these factors would have both increased overall mortality and strengthened the association between injury and mortality.

Management relevance and future research

Few past studies have directly compared short-term captivity and biotelemetry as means of estimating post-release mortality. The collective evidence from our study and past research (Milligan et al. 2000, Donaldson et al. 2011, Olsen et al. 2012) highlight caveats associated with the use of net pen experiments as a means of assessing short-term mortality in wild fish, particularly in fisheries where substantial dermal injury occurs. DFO assign post-release mortality rates to bycatch in all major Pacific salmon fisheries as a means of estimating total incidental fishing mortality each year, in efforts to ensure spawning escapement targets are met (DFO 2013). Nearly all of the mortality rates currently used by DFO are based on short-term (~24–48 h) captivity studies (e.g., using a net pen). Wild adult salmon clearly experience severe stress from short-term confinement (as we found, and Farrell et al. 2001, Donaldson et al. 2011), and sustained cortisol elevation can compound problems that lead to mortality. As an alternative means of assessing survival, biotelemetry poses a different set of issues such as the long-term negative effects of the transmitters (largely unknown), lack of controls, and the inability to physically verify the fate of the animals. However, we noted that survival (i.e., migration success) was quite high for telemetry-tagged fish and that two individuals were recaptured in spawning areas, meaning that the external tag application did not necessarily prevent some fish from reaching freshwater and completing >400 km upstream migrations. Managers and scientists should carefully consider the benefits and drawbacks of both net pens and biotelemetry when designing mortality experiments and using resultant data in management models. Moreover, some weight should be given to long-term survival (>10 d), which can be effectively monitored using biotelemetry (Donaldson et al. 2008, 2011).

The mortality estimates provided by this study are not appropriate for direct application to management of purse seine fisheries that release coho salmon. The catch sizes were very small (largest catch = 82 fish) because our fishing charter occurred during a year of low sockeye salmon and pink salmon abundance, and catches were brailed directly into the recovery tote (although we saw no effect of the alternate dry box sorting on RAMP or injury scores; see Appendix). Actual seine fisheries often land thousands of salmon in single sets, and such catches would necessitate longer entanglement times, higher crowding, and more brailer loads. In Atlantic herring and mackerel (*Scomber scombrus*) purse seine fisheries, longer crowding time and higher crowding densities are correlated with greater stress, behavioral impairment, and mortality (Marcalo et al. 2006, 2010, 2013, Huse and Vold 2010, Tenningen et al. 2012). Even within our small range of catch sizes there was a positive

correlation with both RAMP and injury scores, leading to the prediction that larger sets of fish would likely see higher mortality than in our study. Thus, our short-term bycatch mortality estimate of 20–21% likely represents a "best case" scenario, while being lower than the 70% rate currently applied to the fishery. We suggest further research under a more representative fishing scenario to get a better understanding of the underlying mechanisms to explain this large discrepancy.

Some of our data reveal possible avenues for future work aimed at developing ways to reduce or better manage salmon bycatch mortality in purse seines. We noted that reflex impairment showed no improvement over time for fish placed into the on-board recovery tote (Fig. 2B); a revival technique currently used for bycatch in British Columbian purse seine fisheries. Also, time spent in the recovery tote did not significantly affect survival. There may therefore be some value in investigating whether other revival techniques could promote survival, although options like the Fraser recovery box (Farrell et al. 2001) are inappropriate for the simultaneous revival of dozens of adult salmon since they can only hold two fish at a time. Fork length was significantly correlated with injury and RAMP scores, but was not directly predictive of mortality. While we did determine population origin via DNA analyses (see Appendix), larger sample sizes are required to generate any real conclusions about differences among populations in size, post-release mortality, or other metrics. Our physiological data help demonstrate that RAMP can be an effective tool for assessing the condition of bycatch relative to different capture, handling, and revival practices, but more work is needed to establish whether it can be an effective mortality predictor in ocean-caught coho salmon. The integration of reflex and injury assessments with blood physiology in the net pen experiment provide key insights into the mechanisms of mortality and the limitations associated with the two mortality assessment methods.

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Supplemental Material

Ecological Archives

The Appendix and Supplement are available online: http://dx.doi.org/10.1890/14-0798.1.sm