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Management and Conservation Article

Female Harlequin Duck Winter Survival 11 to 14 Years After the *Exxon Valdez* Oil Spill

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ABSTRACT In the mid- to late 1990s, nearly a decade after the 1989 *Exxon Valdez* oil spill, female harlequin ducks (*Histrionicus histrionicus*) suffered reduced winter survival in oiled areas of Prince William Sound, Alaska, USA, relative to unoiled areas. We conducted follow-up studies from winters 2000–2001 to 2002–2003 to determine whether differential survival persisted and to evaluate whether individual-level indices of oil exposure were related to survival. Using radiotelemetry, we tracked 138 female harlequin ducks from November through March over three winters. We analyzed variation in survival in relation to season, area oiling history, age class, body mass, and an index to exposure to residual oil based on cytochrome P4501A (CYP1A) induction. We determined that survival was most strongly related to season and age class, with evidence of higher survival in late winter and after hatch year (AHY) categories, respectively. We estimated cumulative winter survival for AHY females to be 0.837 (±0.064) and 0.834 (±0.065) on unoiled and oiled areas, respectively, and we estimated hatch-year female cumulative winter survival at 0.766 (±0.138) on unoiled areas and 0.758 (±0.152) on oiled areas. Despite persistence of oil in some intertidal areas and evidence of contaminant ingestion by harlequin ducks during and beyond this study, neither area nor CYP1A were strongly related to variation in survival, suggesting that direct effects of the oil spill on harlequin duck demography had largely abated by the winters 2000–2001 to 2002–2003. Our findings offer an unprecedented description of the timeline of effects of exposure to spilled oil and contribute to a body of literature that describe demographic effects of the *Exxon Valdez* oil spill that persisted over a much longer time than previously assumed. An appreciation for the timescale of chronic effects of oil spills, as well as potential for demographic effects related to much lower concentrations of oil than during the immediate period of acute effects following a spill, will provide wildlife managers with a basis for risk assessment and plans for mitigation when confronted with large spills or chronic pollution.

KEY WORDS demography, *Exxon Valdez*, harlequin duck, *Histrionicus histrionicus*, known fates model, oil spill, radiotelemetry, survival, winter.

The 1989 *Exxon Valdez* oil spill led to mortalities of hundreds of thousands of marine birds and mammals in the months immediately following the spill (Piatt et al. 1990, Garrott et al. 1993, Bodkin and Udevitz 1994, Garshelis 1997). However, effects on wildlife populations were not limited to these acute injuries. Exposure to residual oil and associated physiological and demographic effects were demonstrated for some species for nearly a decade following the spill (Bodkin et al. 2002, Esler et al. 2002). The persistence of oil in intertidal sediments, exposure of vertebrates to that oil, and the chronic population-level effects that were detected through 1998 represented a significant paradigm shift in the way that oil spill effects were perceived (Peterson et al. 2003). This new understanding was in stark contrast to the conventional wisdom at the time of the spill that effects on wildlife populations should be short-lived and present only in the acute phase of catastrophic spills.

Harlequin ducks (*Histrionicus histrionicus*) were one of the species that showed chronic population injury (Esler et al. 2002). Harlequin ducks were particularly vulnerable to *Exxon Valdez* oil spill effects because they inhabit the same nearshore intertidal and sub-tidal habitats where much of the oil was deposited (Galt et al. 1991), and their vulnerability was thought to be exacerbated by a number of natural history, life history, and metabolic traits (Esler et al. 2002). Postspill studies indicated that induction of cytochrome P4501A (CYP1A), a biomarker that is a sensitive indicator of exposure to polycyclic aromatic hydrocarbons, was significantly higher in harlequin ducks from oiled areas than from unoiled areas in 1998, nearly a decade after the spill (Trust et al. 2000). This indication of exposure to residual oil was corroborated by direct evidence of oil in the environment (Hayes and Michel 1999, Short et al. 2004) and CYP1A induction in other vertebrates captured in oiled areas during the same period (Woodin et al. 1997, Jewett et al. 2002). In addition, harlequin duck population surveys from fall 1995 through 1997 indicated declining numbers on oiled areas in contrast with stable numbers on unoiled areas (Rosenberg and Petrula 1998), and winter densities on oiled areas were lower than expected based on habitat attributes of surveyed areas (Esler et al. 2000*a*). Finally, adult female winter survival probabilities were found to differ between oiled and unoiled areas of Prince William Sound from winters 1995–1996 to 1997–1998, which provided evidence of direct effects of oil exposure on population demography and offered a mechanism by which other measures of injury (i.e., population trends and densities) could be explained (Esler et al. 2000*c*).

The results through 1998 indicated that exposure to oil and subsequent constraints to population recovery occurred over much longer time periods for harlequin ducks than anticipated at the time of the spill (Esler et al. 2002, Peterson et al. 2003). Moreover, research beyond 1998 determined that oil remained in some intertidal sediments (Short et al. 2004, 2006), indicating that continued

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consideration of effects of residual oil were warranted, including evaluation of potential continuing effects on harlequin duck survival and demography. Therefore, we quantified female winter survival during winters 2000–2001 through 2002–2003 to follow up on previous studies (Esler et al. 2000*c*) and determine whether differential survival persisted. Specifically, our study objectives were 1) to compare cumulative winter survival probability between oiled and unoiled portions of Prince William Sound, 2) to determine whether our index of individual-level exposure to residual oil was related to survival probability, and 3) to evaluate the predictive strength of area and our index of oil exposure relative to other biologically plausible variables (i.e., age, body mass, and season) for explaining variation in survival.

STUDY AREA

We conducted our study within Prince William Sound, Alaska, USA (60°N, 148°W), which was the site of the 1989 *Exxon Valdez* oil spill and the area in which much of the beached oil was deposited (Galt et al. 1991, Wolfe et al. 1994). We worked at unoiled Montague Island and several oiled sites (Green Island, Bay of Isles, Lower Passage, Crafton Island, and Foul Bay). These are the same locations used in earlier studies, allowing for direct comparison (Esler et al. 2000*c*). Our oiled sites included several for which presence of residual oil was confirmed during the period of our study (Short et al. 2004).

METHODS

Capture and Radiotelemetry

We captured harlequin ducks at all study sites during November of 2000, 2001, and 2002 using a modified floating mist-net trap (Kaiser et al. 1995). Our timing of captures (Nov) differed from that of earlier studies (Sept; Esler et al. 2000*c*) to facilitate direct consideration of oil exposure as an individual covariate in survival models. Previous work demonstrated population-level exposure to oil (Trust et al. 2000) and differences in survival (Esler et al. 2000*c*) in oiled and unoiled areas; however, the individuals for which CYP1A induction and survival were measured were different. By beginning tracking 2 months later, we ensured that birds were on wintering sites long enough prior to capture for exposure to potentially occur if residual oil was present, but still deployed radios before midwinter when area-specific survival probabilities diverged during earlier studies (Esler et al. 2000*c*). Also, by measuring CYP1A induction of radiomarked females, we could evaluate variation in survival as a function of individual oil exposure.

We placed captured harlequin ducks in portable pet carriers and transported them by skiff to a larger vessel for processing. We marked individuals with a uniquely coded United States Fish and Wildlife Service leg band. We identified sex based on plumage, and we estimated female age class (hatch yr [HY] or after hatch yr [AHY]) based on the depth of the bursa of Fabricius and plumage characteristics (Mather and Esler 1999). We measured body mass (±1 g) using an electronic balance.

We placed captured females under general anesthesia and surgically implanted very high frequency radiotransmitters (Holohil Systems Ltd., Carp, Ontario, Canada) in the coelomic cavity with an external antenna extending dorsally (Korschgen et al. 1996, Mulcahy and Esler 1999). This radio type has been shown to perform well for harlequin ducks (Esler et al. 2000*b*) and other sea ducks (Iverson et al. 2006), with no adverse effects detected after a 2-week postsurgery period. Transmitters weighed approximately 18 g, which is <3% of the body mass of the lightest wintering female harlequin duck that we captured. Transmitters were equipped with motion-sensitive mortality sensors; the pulse rate changed from 45 beats per minute to 90 beats per minute when mortality was indicated. Following recovery from surgery (at least 1 hr), we released ducks near their capture site. All methods were approved by the Animal Care and Use Committee at Simon Fraser University (Approval no. 743B05).

We monitored radio signals for 138 individuals from an aircraft approximately every 2 weeks from the time of marking through the end of March. On every flight, we recorded each bird's status (alive, dead, or not detected) and general location. We confirmed mortality status by either carcass recovery or detection of signals from upland habitats, which are not used by harlequin ducks during nonbreeding periods (Robertson and Goudie 1999).

Cytochrome P4501A Induction

During surgeries for radio implantation, we surgically biopsied the livers of captured females to obtain a small (approx. 0.10-g) sample for CYP1A analysis, the biomarker indicating exposure to oil (Trust et al. 2000). Immediately following biopsy, we placed liver samples in a cryogenic vial and froze them in liquid nitrogen. We shipped liver samples in a liquid nitrogen vapor shipper to Woods Hole Oceanographic Institute (Woods Hole, MA, USA) for subsequent preparation and analysis. Lab staff homogenized individual liver samples in 7 mL final volume of homogenizing buffer (0.05 M Tris, 0.15 M KCl, pH 7.4) and sedimented microsomes by differential centrifugation as described by Stegeman et al. (1979). Microsomes were resuspended in approximately 2 mL/g tissue with resuspension buffer (0.05 M Tris, 0.1 mM ethylenediaminetetraacetic acid, 1 mM dithiothreitol, 20% v/v glycerol, pH 7.4). Protein was determined in a 96-well plate using the microprocedure of Smith et al. (1985).

Lab staff measured 7-ethoxyresorufin-O-deethylase (EROD), the catalytic function of hydrocarbon-inducible CYP1A, using a kinetic modification of the plate-based assay of Kennedy et al. (1993). Technicians determined EROD activity in duplicate in a 48-well plate at 20° C using a Cytofluor® fluorescent plate reader (Millipore, Bedford, MA). Each well contained 200 µl consisting of 1 µl of microsomes (4–15 µg protein) and 2 µM 7-ethoxyresorufin in 50 mM Tris buffer, 0.1 M NaCl, pH 7.8. Lab technicians initiated catalytic activity by the addition of nicotinamide adenine dinucleotide phosphate in buffer to a final 1.67-mM concentration. They determined fluorescence at 1-minute

intervals over 6 minutes, and divided the linear slope (fluorescence/min) by the slope of the resorufin product standard curve (fluorescence/pmol) determined under the same conditions to yield catalytic rates (pmol/min/mg protein).

We found that the EROD activity data reported from the laboratory varied dramatically in magnitude across years, to a degree beyond what could be expected due to normal biological variation. This was confirmed by similar differences across the same years for liver biopsies collected under captive, controlled circumstances (Esler 2008). In consultation with the laboratory, we concluded that variation in analysis approaches and equipment had led to the observed interannual differences and that, although within-year comparisons between areas were valid, between-year comparisons were not appropriate without corrections. Therefore, we created an index for CYP1A values that allowed for interannual contrasts, in which we set average EROD activity for harlequin ducks that we captured at our unoiled area (Montague Island) to 1 for each year, and we adjusted all values accordingly within the same sample year (Esler 2008). Therefore, for each year, the average indexed EROD activity for birds from oiled areas is the ratio of oiled to unoiled averages. Further, we calculated the indexed values for each individual (i.e., the ratio of the observed data for the individual relative to the average of all birds from the unoiled area in that yr), which allowed consideration of relationships between individual variation in oil exposure and variation in survival. This index assumes that oil exposure, and hence EROD activity, at unoiled Montague Island was the same across years, which is reasonable because it is a relatively pristine area with little interannual variation in human activity and, hence, little variation in occurrence or concentrations of CYP1A-inducing compounds. Not all radiomarked individuals had corresponding EROD activity information, because some samples thawed before analysis, and we excluded samples with low protein yield and samples with results below detection limits (Esler 2008); we conducted some data analysis using the data set in which we only included individuals with EROD activity information ($n = 103$). Similarly, we had EROD data from some birds that we did not include in survival analyses because of death or loss of radio signal prior to the monitoring period; we included these data in comparisons of EROD activity across areas.

Data Analysis

We compared average, standardized EROD activity between oiled and unoiled areas in an information-theoretic context (Burnham and Anderson 2002), in which we contrasted fit of the data to 1) a model with a group effect for area and 2) a null model. This is roughly analogous to a *t*-test in a traditional hypothesis-testing paradigm.

We evaluated female harlequin duck survival using the known-fates modeling procedure in Program MARK (White and Burnham 1999). The known-fates procedure is derived from the Kaplan–Meier estimator (Kaplan and Meier 1958), with modifications to allow for staggered entry of subjects into the study population (Pollock et al. 1989) and likelihood inference based on binomial probabilities (White and Burnham 1999). Known-fates models assume a resight probability of 1 and assume that individuals that are censored from the data set because of radio failure or emigration from the study area have the same survival probability as uncensored birds (Pollock et al. 1989, Tsai et al. 1999). We are confident that our data met these assumptions because previous work in the same area with the same study species and transmitter type confirmed that return rates of radioed birds were the same as those that were not radiomarked (Esler et al. 2000*b*). Our modeling procedure was the same as used in previous studies (Esler et al. 2000*c*), thus facilitating direct comparisons.

We used information-theoretic methods to draw inference from our analyses (Burnham and Anderson 2002). First, we summarized data by month to build our encounter history file, because finer timescales did not improve explanatory value in previous analyses (Esler et al. 2000*c*). Within each calendar month from December through March, we counted the mortalities that occurred and the number of female harlequin ducks at risk of mortality (i.e., no. of radios detected), following methods of Pollock et al. (1989) and Bunck et al. (1995). We made an a priori decision to combine data across years to maximize power to detect differences over the entire study. Previous research indicated that the most appropriate time dependency for use in our analysis was seasonal variation, wherein survival probability differed between midwinter (Dec and Jan) and late winter (Feb and Mar; Esler et al. 2000*c*). We confirmed this (see Results) by comparing 3 basic models in which we allowed survival probability to vary by month, vary by season, and remain constant during the study. By determining the appropriate time dependency prior to the main analysis, we reduced the number of models in our final candidate model set and focused our analysis on variation related to individual covariates.

Our candidate model set included seasonal time dependency and all additive combinations of the 4 explanatory variables of interest: area (oiled and unoiled), age class (HY and AHY), body mass, and the index of exposure to residual oil (CYP1A). We also included a null model and a fully parameterized global model in which survival differed by month and the 4 individual covariates were incorporated, resulting in a candidate set of 18 models (Table 1). Because we did not have CYP1A data for all 138 birds that we successfully monitored, we applied these models to a data set with the 103 individuals with CYP1A information. Subsequent analyses included all 138 monitored individuals, based on findings from preliminary analyses (see Results). For these analyses, we reclassified age and area as group factors for our data input file and evaluated variation in survival for a dataset consisting of 62 AHY females from oiled areas, 10 HY females from oiled areas, 49 AHY females from unoiled areas, and 17 HY females from unoiled areas. As in the previous analysis, the candidate model set assumed seasonal time dependency and included all combinations of area and age, along with null and global

Table 1. Models used to evaluate variation in winter survival of female harlequin ducks in Prince William Sound, Alaska, USA, from 2000 to 2002.

Model[a]	AIC_c[b]	ΔAIC_c	w_i	K[b]	Parameter likelihoods[c]			
					Area	Age	EROD[d]	Mass
Season + age	133.76	0.00	0.16	3		0.16		
Season	133.92	0.16	0.15	2				
Season + age + EROD	134.76	1.00	0.10	4		0.10	0.10	
Season + EROD	134.84	1.08	0.10	3			0.10	
Season + age + mass	135.58	1.82	0.07	4		0.07		0.07
Season + area + age	135.69	1.93	0.06	4	0.06	0.06		
Season + area	135.95	2.19	0.05	3	0.05			
Season + mass	135.95	2.19	0.05	3				0.05
Season + area + age + EROD	136.33	2.57	0.05	5	0.05	0.05	0.05	
Season + age + EROD + mass	136.74	2.98	0.04	5		0.04	0.04	0.04
Season + area + EROD	136.80	3.04	0.04	4	0.04		0.04	
Season + EROD + mass	136.84	3.08	0.03	4			0.03	0.03
Season + area + age + mass	137.40	3.64	0.03	5	0.03	0.03		0.03
Season + area + mass	138.00	4.24	0.02	4	0.02			0.02
Null model	138.01	4.25	0.02	1				
Season + area + age + EROD + mass	138.18	4.42	0.02	6	0.02	0.02	0.02	0.02
Season + area + EROD + mass	138.83	5.07	0.01	5	0.01		0.01	0.01
Month + area + age + EROD + mass	140.11	6.35	0.01	8	0.01	0.01	0.01	0.01
				$(\Sigma w_i) =$	0.28	0.52	0.38	0.28

[a] We constrained time dependency to vary seasonally (midwinter and late winter) based on preliminary analyses, and the subsequent candidate set included models with seasonal time dependency and all additive combinations of area (oiled and unoiled), age class (adult and hatch yr), exposure to residual oil (CYP1A), body mass, and null and global models.
[b] AIC_c = Akaike's Information Criterion adjusted for small sample size; K = no. of parameters.
[c] We calculated parameter likelihoods as the sum of Akaike wt (Σw_i) for models including the explanatory variable of interest.
[d] EROD = 7-ethoxyresorufin-O-deethylase activity.

models as described above. By using all available data, this analysis provided the most robust survival estimates for use in demographic analyses. We used these results to generate survival estimates for each age, area, and season combination using model averaging across the candidate set.

We structured models in Program MARK using design matrices and we used a logit link function to bound parameter estimates. We incorporated area and age class as categorical covariates. Body mass and EROD were continuous covariates and were scaled by dividing values by 1,000 and 10, respectively, so that means would fall between 0 and 1 and within the range −3 to +3, thus ensuring numerical optimization of the program algorithm for survival estimation (White and Burnham 1999); note that this was a separate process from that in which we standardized EROD data across years, described above, and was strictly to enhance program performance. Goodness-of-fit statistics are currently unavailable for known-fate models; therefore, we evaluated potential effects of over-dispersion on model selection by adjusting the variance inflation factor ($\hat{c}$) in Program MARK from 1 (no overdispersion) to 3 (extreme overdispersion) in increments of 0.5 and examined the effect of this change on model rankings.

For each model, we calculated Akaike's Information Criterion with a second-order bias correction, AIC_c, to evaluate the degree of parsimony across models (i.e., the explanatory value of the model after accounting for the no. of parameters estimated) and ΔAIC_c, which is the difference between the most parsimonious model (lowest AIC_c) and each model in the candidate set. Comparison among models was facilitated by deriving an index of relative plausibility using normalized Akaike weights (w_i), with the ratio of w_i between any 2 models indicating the relative degree to which a particular model was better supported by the data than the other model (Burnham and Anderson 2002). To contrast the variation in survival explained by each explanatory variable, we used a balanced candidate set design and calculated combined Akaike weights for each variable. Combined Akaike weights are the sum of all Akaike weights (Σw_i) for models including the explanatory variable of interest and range from zero to 1, with values close to zero indicating little evidence that the explanatory variable is related to variation in survival, and values close to 1 indicating considerable support for the importance of that variable. Finally, we examined beta values (β) and standard errors (SE β) for each variable in the most parsimonious model that included the variable of interest. Beta values under the known-fates procedure in Program MARK are model-estimated slopes, which can be interpreted on the basis of direction and their effect size relative to associated variation. We used a criterion of SE β < β to identify variables that potentially had a biologically meaningful relationship with survival; we recognize that this is an arbitrary criterion and chose it based on the balance between power to identify important attributes and strength of inference.

RESULTS

Of the 103 radioed females with both relocation and CYP1A data, we captured 61 on oiled areas (52 AHY and 9 HY) and 42 on unoiled areas (29 AHY and 13 HY). Our index to CYP1A induction averaged (±SE) higher in oiled areas (2.89 ± 0.58; n = 67) than in unoiled areas (1.00 ± 0.21; n = 49). A model including an area term was much better supported by the data than a null model (w_i = 0.93 and w_i = 0.07, respectively), and the ΔAIC_c of the null

Table 2. Effect estimates (β), associated standard errors, and inferences drawn for select models used to estimate winter survival (S) for female harlequin ducks in Prince William Sound, Alaska, USA, during 2000–2002.

Model[a]	Variables	β	SE	Inference
Season + age	Intercept	3.4	0.7	
	Midwinter	−1.4	0.6	$S_{midwinter} < S_{latewinter}$
	AHY[b]	0.8	0.5	$S_{AHY} > S_{HY}$
Season	Intercept	4.0	0.6	
	Midwinter	−1.4	0.6	$S_{midwinter} < S_{latewinter}$
Season + age + EROD[c]	Intercept	3.1	0.7	
	Midwinter	−1.4	0.6	$S_{midwinter} < S_{latewinter}$
	AHY	0.8	0.5	$S_{AHY} > S_{HY}$
	EROD	1.0	1.2	No relationship
Season + area + age	Intercept	3.4	0.7	
	Midwinter	−1.4	0.6	$S_{midwinter} < S_{latewinter}$
	Oiled area	−0.2	0.5	No relationship
	AHY	0.9	0.5	$S_{AHY} > S_{HY}$
Month + area + age + EROD + mass	Intercept	5.1	4.6	
	Dec	−0.7	0.9	No relationship
	Jan	−1.4	0.8	$S_{January} < S_{March}$
	Feb	0.7	1.2	No relationship
	Oiled area	−0.5	0.6	No relationship
	AHY	1.1	0.6	$S_{AHY} > S_{HY}$
	EROD	1.3	1.4	No relationship
	Mass	−3.8	7.9	No relationship

[a] Models presented include the 2 models that were most parsimonious given the data, the most parsimonious model to include EROD as a covariate, the most parsimonious model to include area as a covariate, and the fully parameterized global model (see Table 1 for Akaike's Information Criterion values and weights).
[b] AHY = after hatch yr age class; HY = hatch yr age class.
[c] EROD = 7-ethoxyresorufin-O-deethylase activity.

model was 5.2. These findings provide strong support for the inference that exposure to residual *Exxon Valdez* oil was ongoing during our study.

We found that a model with seasonal time dependency (i.e., allowing survival to vary between midwinter and late winter) was best supported ($AIC_c = 133.92$; $w_i = 0.67$); w_i for the best-supported model was 2.8 times higher than that of a model in which survival varied monthly ($\Delta AIC_c = 2.06$; $w_i = 0.24$), and 7.4 times higher than that of a model with survival constrained to be constant ($\Delta AIC_c = 4.10$; $w_i = 0.09$). Therefore, in all subsequent analyses, we used season as our basis for time dependency, with the exceptions of using month within the fully parameterized global model and including no time dependency within the null model.

Of the candidate models, the most parsimonious included season and age as explanatory variables (Table 1). However, the model with season as the only explanatory variable received nearly the same support, and there were several other models with similar but only slightly lower degrees of support (Table 1). Despite the high degree of model uncertainty, the null model was poorly supported (ranked 15th of 18 models considered), and the evidence ratio for the most parsimonious model relative to the null was 8.4, indicating that the variables under consideration did explain significant variation in the data. Moreover, goodness-of-fit testing results indicated that our results were robust to moderate levels of over-dispersion. We found that the model including season as the only explanatory variable ranked ahead of the model including season and age when $\hat{c} > 1.5$; however, it also ranked ahead of the null model for all adjustments of $\hat{c} \leq 3.0$.

Combined Akaike weights and beta estimates for individual covariates offered additional insights into the variables that were related to survival (Table 1). The age variable received the highest support across models with $w_{age} = 0.52$, whereas combined Akaike weights for other variables were lower ($w_{EROD} = 0.38$, $w_{area} = 0.28$, and $w_{mass} = 0.28$), indicating that these were less closely related to survival (Table 1). Beta values were consistently higher for late winter than for midwinter, and for AHY females than for HY females in all high-ranking models, with standard errors smaller than the estimated slopes for season and age parameters (Table 2). However, beta values for area, mass, and EROD were smaller than the associated standard errors, suggesting a lack of meaningful effects.

Given the results described above, in which there was no support for effects of the continuous individual covariates of mass and EROD, we ran additional analyses without these variables on our full set of 138 females for which we had all necessary data. We found that model selection results and inferences drawn were entirely consistent with analyses above. Two models received similar levels of support from the data: the model with season and age (S[season + age]; $AIC_c = 183.39$; $w_i = 0.39$) and the one with season only (S[season]; $\Delta AIC_c = 0.14$; $w_i = 0.36$). No other models received support greater than that of the null (S[null]; $\Delta AIC_c = 1.66$, $w_i = 0.19$), including all models that included an area term.

We generated model-averaged survival estimates from the final analysis including all 138 individuals for all combinations of season, area, and age class, as well as cumulative winter survival estimates by area and age class (Fig. 1).

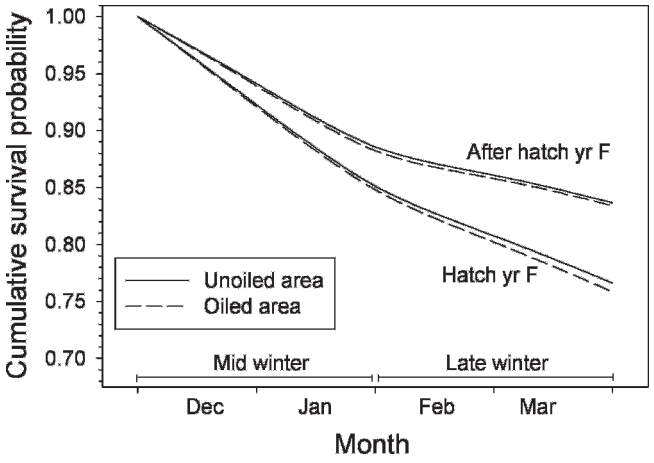

Figure 1. Cumulative winter survival probabilities of radiomarked female harlequin ducks (n = 138) from Prince William Sound, Alaska, USA, during winters 2000–2001 through 2002–2003. Lines represent model-averaged estimates across models allowing variation in relation to season (midwinter and late winter), age class (hatch yr and after hatch yr), and area (oiled during the *Exxon Valdez* oil spill and unoiled).

Effects of age class and season were evident, as was the lack of an important effect of area. We estimated survival (±SE) of AHY females to be 0.886 (±0.033) during midwinter and 0.945 (±0.031) during later winter on unoiled areas. We estimated very similar values for AHY females from oiled areas (Fig. 1), with survival estimates of 0.882 (±0.034) and 0.945 (±0.031) during midwinter and late winter, respectively. Survival estimates of HY females were consistently lower than those of AHY birds. On unoiled areas, we estimated HY female survival to be 0.851 (±0.073) in midwinter and 0.900 (±0.071) in late winter. Similarly, we estimated survival for HY females on oiled areas to be 0.848 (±0.075) in midwinter and 0.894 (±0.083) in late winter (Fig. 1). Cumulative winter survival estimates for AHY females were 0.837 (±0.064) and 0.834 (±0.065) on unoiled and oiled areas, respectively. We estimated HY female cumulative winter survival to be 0.766 (±0.138) on unoiled areas and 0.758 (±0.152) on oiled areas.

DISCUSSION

We found that female harlequin duck winter survival did not differ between areas oiled during the *Exxon Valdez* spill and nearby unoiled areas during winters 2000–2001 through 2002–2003, which was 11–14 years after the 1989 spill. Our findings contrast with those from directly comparable studies from the mid-1990s (6–9 yr after the spill), in which survival rates were 5.7% lower in oiled areas compared to unoiled areas (Esler et al. 2000*c*). Taken together, these data sets constitute an unprecedented documentation of the degree and duration of direct demographic effects related to chronic exposure to residual oil following catastrophic spills. Our results indicate that it took roughly a decade for survival of female harlequin ducks to recover following the *Exxon Valdez* spill, which is much longer than had been assumed that deleterious effects on wildlife populations would be expressed (Peterson et al. 2003).

The lack of differential survival between oiled and unoiled areas occurred despite documentation of persistence of *Exxon Valdez* oil in some intertidal areas within the spill zone during the time of our study (Short et al. 2004) and our own data on CYP1A induction indicating that harlequin ducks continued to be exposed to residual oil. These are not necessarily contradictory results and can be reconciled by recognizing that exposure to oil does not necessarily lead to overt, direct effects on demography. The CYP1A biomarker is sensitive and can be induced by exposure to very low concentrations of hydrocarbons (Payne et al. 1987, Goksøyr 1995) that may or may not have meaningful or discernable effects on demography of individuals or dynamics of populations. With the volume of oil remaining in intertidal beaches declining over time (albeit more slowly than anticipated; Short et al. 2004), one would expect diminishment of both exposure and effects, though not necessarily on the same timescale. That appears to be the case with harlequin ducks in Prince William Sound, with evidence of ongoing exposure to residual oil through at least March 2009 based on EROD activity data (D. Esler, Simon Fraser University, unpublished data), but with survival rates in oiled areas that are equivalent to unoiled based on our results.

It is likely that the level of exposure was sufficient to induce elevated CYP1A in some individuals but not high enough to affect their winter survival. However, we also recognize that individual-level CYP1A induction represents a single point in time when sample collection occurred, thus we are assuming that our samples represented overall risk of exposure to oil. Given that exposure to oil may be intermittent (Short et al. 2006) and we did not know the exposure history of captured birds, we are uncertain where each individual bird falls on the CYP1A response curve. Similarly, an individual unexposed at capture could subsequently encounter oil and potentially suffer associated effects. Nonetheless, given that we captured birds randomly, we believe our samples were representative of the population and likely included CYP1A variation associated with duration since oil exposure.

Our data indicate that female harlequin duck winter survival had converged between oiled and unoiled areas by the time of our study. We conclude that direct effects had largely abated 11–14 years following the spill because female survival was the demographic attribute that had been identified in previous studies as the most important constraint on population recovery imposed by continuing effects of the *Exxon Valdez* spill (Esler et al. 2000*c*, 2002). However, despite lack of evidence for effects of continued oil exposure on survival at either population or individual levels, we caution that subtle or sublethal effects are plausible (Carls et al. 2005). In other wildlife species, effects of oil exposure on sub-organismal systems have been described (Jessup and Leighton 1996, Alonso-Alvarez et al. 2007), and the metabolic intermediates produced during CYP1A induction have been shown to have toxic potential (e.g., Nebert et al. 2004). These could potentially influence individual demography at timescales and mechanisms (e.g.,

via reproduction) beyond what we measured. These would be relevant issues to address to fully consider effects of the *Exxon Valdez* oil spill on harlequin ducks. In addition, we recommend continued monitoring of CYP1A until levels become equivalent between oiled and unoiled areas, to fully document the timeframe over which exposure occurs and subsequently over which any sublethal or long-term effects might be expressed.

Our findings also provide additional insights into harlequin duck demography, unrelated to the oil spill. We confirmed findings of Esler et al. (2000*c*), which indicated that midwinter survival was lower than for other winter periods, suggesting the potential of midwinter survival as a demographic bottleneck. We also demonstrate that, after accounting for seasonal variation that affected all age classes, winter survival of HY females was consistently lower than that of AHY females, which also was described by Mittelhauser (2008). Similar annual survival rates of female harlequin ducks of both age classes (Cooke et al. 2000) may reflect a balance between higher mortality of young females during winter and mortality incurred by adult females on breeding streams (Mittelhauser 2008, Bond et al. 2009). Our findings contribute to a body of literature describing harlequin duck survival throughout the annual cycle, which will be important for understanding when and where demographic constraints on population growth are manifested (Cooke et al. 2000, Iverson and Esler 2007, Mittlehauser 2008, Bond et al. 2009).

MANAGEMENT IMPLICATIONS

Our studies define the duration (roughly a decade) of reduced female harlequin duck survival rates in areas contaminated during a major oil spill. This puts a timeline on persistence of direct effects that far exceeds conventional assumptions about how long these might be important (Peterson et al. 2003). This further highlights the importance of population-level effects during the chronic phase of exposure to residual oil, despite a common perception that most effects on wildlife populations occur during the acute phase, the weeks or months immediately following an oil spill. This understanding of the duration and mechanism of demographic effects is critical when applying management following large spills (e.g., considering the costs and benefits of removal of residual oil) and when applying risk assessment under scenarios of catastrophic contaminant releases. This also demonstrates the sensitivity of benthic-foraging vertebrates to residual oil from major spills, or chronic contamination, sequestered in sediments (Bodkin et al. 2002, Esler et al. 2002) and indicates that species with natural history and life history traits similar to those of harlequin ducks might be particularly vulnerable, and thus warrant enhanced conservation concern. Our findings can be combined with other measures of demographic attributes (e.g., Iverson and Esler 2006, 2007) to build population models that evaluate the chronology, mechanisms, and severity of negative effects of oil spills, as well as the processes by which population recovery occurs.

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