Could residual oil from the *Exxon Valdez* spill create a long-term population "sink" for sea otters in Alaska?

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Abstract. Over 20 years ago, the *Exxon Valdez* oil tanker spilled 42 million L of crude oil into the waters of Prince William Sound, Alaska, USA. At the time of the spill, the sea otter (*Enhydra lutris*) population inhabiting the spill area suffered substantial acute injuries and loss. Subsequent research has resulted in one of the best-studied species responses to an oil spill in history. However, the question remains: Is the spill still influencing the Prince William Sound sea otter population? Here we fit time-varying population models to data for the sea otter population of western Prince William Sound to quantify the duration and extent of mortality effects from the spill. We hypothesize that the patchy nature of residual oil left in the environment has created a source–sink population dynamic. We fit models using the age distributions of both living and dying animals and estimates of sea otter population size to predict the number of sea otters in the hypothesized sink population and the number lost to this sink due to chronic exposure to residual oil. Our results suggest that the sink population has remained at just over 900 individuals (95% CI: 606–960) between 1990 and 2009, during which time prime-age survival remained 2–6% below pre-spill levels. This reduced survival led to chronic losses of ~900 animals over the past two decades, which is similar in magnitude to the number of sea otter deaths documented in western Prince William Sound during the acute phase of the spill. However, the unaffected source population appears to be counterbalancing these losses, with the model indicating that the sea otter population increased from ~2150 individuals in 1990 to nearly 3000 in 2009. The most optimistic interpretation of our results suggests that mortality effects dissipated between 2005 and 2007. Our results suggest that residual oil can affect wildlife populations on time scales much longer than previously believed and that cumulative chronic effects can be as significant as acute effects. Further, source–sink population dynamics can explain the slow recovery observed in the spill-affected western Prince William Sound sea otter population and are consistent with available data.

Key words: AIC; ecological risk assessment; Enhydra lutris; *oil spills; Prince William Sound, Alaska, USA; sea otter; source–sink models.*

INTRODUCTION

On 24 March 1989, the tanker vessel *Exxon Valdez* ran aground on Bligh Reef in northeastern Prince William Sound (PWS), Alaska, USA, spilling an estimated 42 million L of Prudhoe Bay crude oil (Spies et al. 1996). At that time, this spill was the largest recorded accidental release of oil into U.S. waters. Oil spread by current and winds in a southwesterly direction, leaving a heavy layer on numerous beaches within western Prince William Sound (WPWS) before exiting Montague Strait and other passages at the southwest corner of the Sound (Fig. 1). Islands in the central portion of WPWS were in the direct path of the moving oil and not far from the source, and were heavily contaminated. The Knight Island complex (Fig. 1)

included some of the heaviest oiled habitat in WPWS. Oil eventually covered over 26000 km^2 of water in WPWS and the Gulf of Alaska and coated over 1900 km of coastline (Morris and Loughlin 1994, Spies et al. 1996).

At the time of the *Exxon Valdez* oil spill (EVOS), toxicological studies generally examined immediate, acute effects on wildlife populations. In the case of the EVOS, acute effects were dramatic, and spill response personnel recovered thousands of dead or injured birds and mammals in the first few months following the spill (Piatt et al. 1990, Garrott et al. 1993). One of the more severely affected inhabitants of WPWS was sea otters (*Enhydra lutris*), which are highly susceptible to oil-related injuries and death (Costa and Kooyman 1982, Siniff et al. 1982, Williams et al. 1988). In the weeks following the spill, sea otters suffered lung, liver, and kidney damage due to acute oil exposure via inhalation, consumption, and external contact (Lipscomb et al. 1993, 1994, Rebar et al. 1995). By September 1989, over
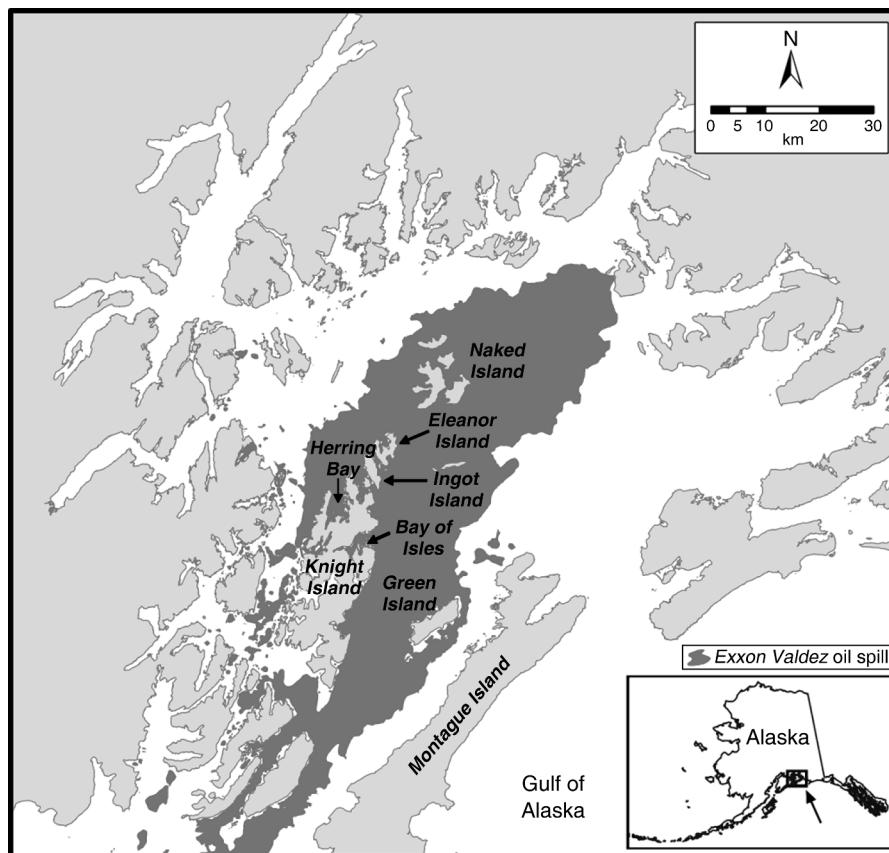
FIG. 1. Map of Prince William Sound, Alaska, USA, and the trajectory of oil from the 1989 *Exxon Valdez* oil spill (EVOS). Sea otter (*Enhydra lutris*) carcasses were collected in oiled portions of western Prince William Sound (WPWS) with systematic collections at Green Island and opportunistic collections at Naked Island, the northern Knight Island complex, and other islands and passages south of Knight Island. Aerial surveys of WPWS included all waters west of Montague Island, including the western shore of Montague Island.

600 carcasses and oiled otters had been collected from WPWS, and nearly 1000 dead otters had been recovered over the entire spill area, including areas outside PWS in the Gulf of Alaska (Ballachey et al. 1994). Total immediate mortality due to the spill was likely higher, but difficult to quantify (Garrott et al. 1993, DeGange et al. 1994, Loughlin et al. 1996, Garshelis 1997). Total pre-spill abundances are not known; however, Burn (1994) estimated a minimum of ~2300 sea otters occupied oiled areas of WPWS based on shoreline surveys conducted in 1984–1985. These data suggest that at least 25% of the WPWS sea otter population died within a few months of the spill.

After the spill, initial studies focused on damage assessment, followed by a series of longer term studies designed to assess the recovery status of the WPWS nearshore species (Bodkin et al. 2002, Esler et al. 2002, Golet et al. 2002, Bowyer et al. 2003). Further studies identified relatively unweathered and toxic residual hydrocarbons remaining in the environment >15 years after the spill (Short et al. 2004, 2006, 2007, Li and Boufadel 2010). Sea otters and other nearshore benthic-

feeding organisms forage in intertidal soft-sediment habitats where reservoirs of oil contamination remain (Wolfe et al. 1994, Short and Babcock 1996, Hayes and Michel 1999, Short et al. 2004, 2006, 2007). This creates the potential for continued exposure to hydrocarbons during recovery and ingestion of infaunal and epifaunal prey. Peterson et al. (2003) summarized numerous studies that conclude long-term exposure has resulted in effects to benthic-feeding predators including sea otters (Esler et al. 2000, 2002, Fukuyama et al. 2000, Trust et al. 2000, Dean et al. 2002, Bodkin et al. 2002, Golet et al. 2002, Jewett et al. 2002, Peterson and Holland-Bartels 2002).

Monson et al. (2000a) quantified the long-term survival effects of the EVOS on sea otters in WPWS, using population models fit to the age distributions of beach-cast sea otter carcasses collected each spring. They found that from 1989 to 1998, survival rates in the WPWS population had declined relative to pre-spill rates, and that the magnitude of the decline depended on the age class of animals and time since the spill (Fig. 2a, b). The strongest patterns included an immediate
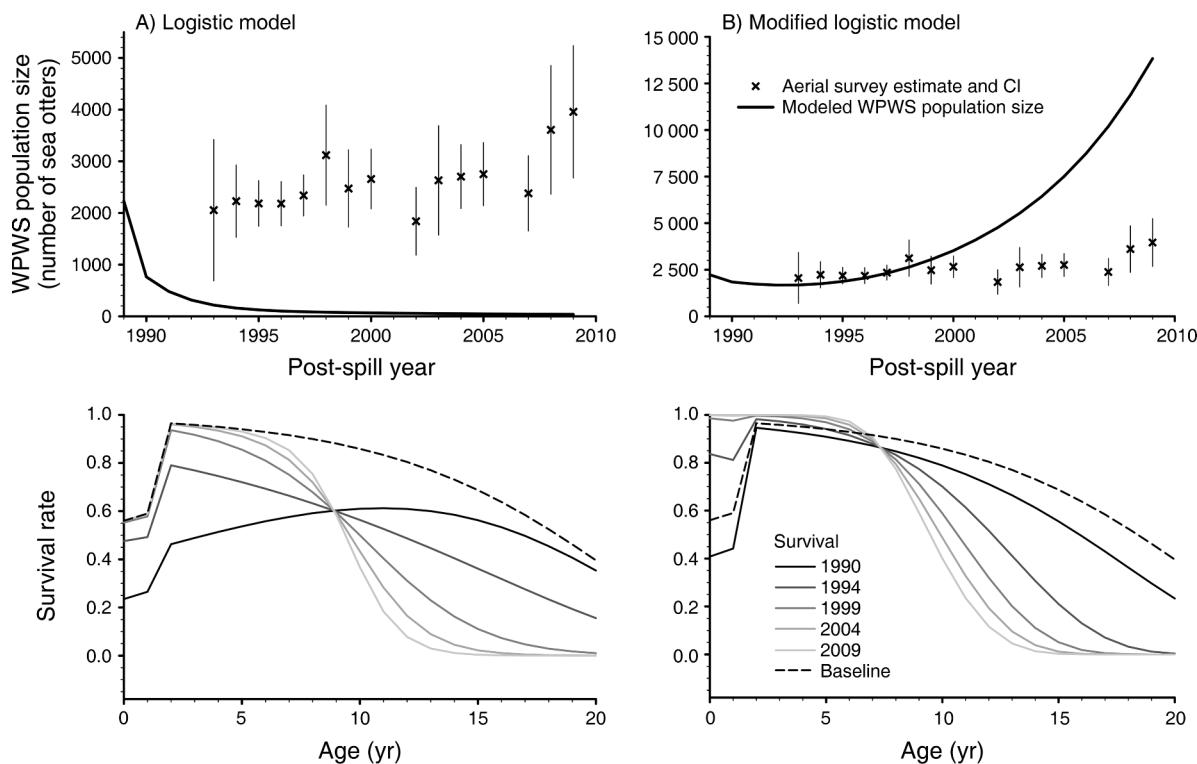
FIG. 2. Predicted age-specific survival rates (bottom panels) and resulting population change in sea otters for 20 years post-spill (top panels) using the (a) logistic model and (b) modified logistic model and parameter estimates from Monson et al. (2000*a*), applied to the entire WPWS sea otter population (i.e., homogenous population effect). Note the modeled population change (solid black line) relative to actual aerial survey estimates (black X's; values are provided in the Supplement).

dramatic reduction in survival of younger age classes the year following the spill, with survival of older adults less affected during the first few years following the spill. However, survival of younger age classes (<9 years) increased with time, while survival of older animals (>9 years) declined with time (Fig. 2a, b). Negative survival effects were not limited to animals alive at the time of the spill (i.e., individuals that potentially survived acute exposure), suggesting that chronic exposure to residual contaminants in the environment was not trivial. Other evidence that these patterns of survival were caused by lingering oil include low survival rates of marked juvenile sea otters following the spill (Monnett and Rotterman 1995, Ballachey et al. 2003) and the discovery in other nearshore species of elevated bio-marker activity associated with hydrocarbon exposure (Esler et al. 2002, 2010, Golet et al. 2002, Jewett et al. 2002, Bowyer et al. 2003, Ricca et al. 2010).

Monson et al. (2000*a*) assumed mortality was spatially homogeneous, affecting the entire WPWS sea otter population, and that changes in survival rates through time were monotonic and could not return to "normal" any time after the spill. These assumptions may not be appropriate given the observed patterns of population change (Fig. 2a, b), and the locations of residual oil. In reality, residual oil was limited to geographically scattered locations within only a few areas of WPWS

(Wolfe et al. 1994, Short and Babcock 1996, Hayes and Michel 1999, Short et al. 2004, 2006, 2007), and thus, the entire WPWS sea otter population would not be equally exposed to residual contaminants.

Support for heterogeneous exposure levels and mortality effects include diverging population trends between heavily oiled areas and areas with little or no residual oil (Bodkin et al. 2002). For example, during post-spill damage assessment studies, the Knight Island complex (Fig. 1) was chosen as a study site because it received heavy oiling in 1989, and the area continued to hold considerable amounts of oil on scattered beaches 20 years after the spill (Short et al. 2007). Acute sea otter mortality at the time of the spill approached 90% in this area (Bodkin et al. 1993), and sea otter numbers remained reduced through at least 2005 (Supplement; Bodkin et al. 2002). In contrast, over the two decades since the spill, sea otter numbers increased in WPWS as a whole (Fig. 2a, b; Supplement) and within unoiled areas in particular (Dean et al. 2002, Bodkin et al. 2002).

Subsequent research found that lack of food resources could not explain the lack of recovery of the Knight Island complex sea otter population (Dean et al. 2002). In fact, reduced sea otter densities in this area apparently allowed prey populations to increase, as by the mid-1990s, estimated prey biomass per otter was approximately four times higher in the northern Knight

Island complex than in a non-affected area (Dean et al. 2002). High dispersal rates of young animals born in the spill area or other mechanisms of behavioral avoidance could explain the lack of recovery. A few small, localized areas have had few otters over the two decades since the spill, suggesting animals may avoid those specific areas within the oiled area. However, most habitat within the spill area is occupied, and telemetry and tagging studies conducted over that past two decades found that otters remained within the overall oiled area where they were tagged (Ballachey et al. 2003; USGS, *unpublished data*). Instead, the demographic effects suggested by Monson et al. (2000*a*) appeared to be the explanation most consistent with available data.

Monson et al. (2000*a*) concluded that the diverging population trends of sea otters in oiled and unoiled areas occurred because chronic oil effects influenced only the subset of the population residing in the most heavily oiled areas. Specifically, they hypothesized that the oil-affected subpopulation was acting as a population sink (Pulliam 1988, Pulliam and Danielson 1991, Dunning et al. 1992) where chronic exposure continued to remove some unknown number of individuals from local oil-affected areas, thus constraining recovery of the larger WPWS population to some unknown extent.

Here we explicitly looked for long-term heterogeneous effects of the 1989 EVOS in Prince William Sound by modeling the WPWS sea otter population as two subpopulations in a source–sink framework, and explore the hypothesis that the spill created oil-affected "sink" and non-affected "source" subpopulations. Similar to Monson et al. (2000*a*), our analyses utilize age-at-death data acquired from beach-cast carcasses. In addition, we incorporated sea otter population survey data for WPWS and the age distributions of live captured animals residing in both oiled and unoiled areas (see Bodkin et al. 2002 for survey and capture methods). We used time-varying, age-specific demographic models to predict changes in sea otter survival rates in the hypothesized source and sink subpopulations. We fit these models by iteratively changing survival rates in both subpopulations away from pre-spill, baseline values (Udevitz et al. 1996, Udevitz and Ballachey 1998) until the predicted age distributions of both living and dead animals and trends in population numbers best matches observed values. The "best fit" model parameter values are determined by calculating negative log-likelihood values based on the difference between the observed and predicted values (Burnham and Anderson 2002). These methods allowed us to identify the most likely ways in which the spill has influenced the demography of the hypothesized sink population (Doak and Morris 1999, Tinker et al. 2006), thus providing additional insight into the magnitude and persistence of potential chronic effects of the EVOS on WPWS sea otters. Our goals included (1) estimating the size of the hypothesized sink population relative to the total WPWS population, (2) estimating the number of

animals lost to the sink population since the EVOS, and (3) explicitly looking for a point in time when survival rates in the sink subpopulation may have returned to normal, or at least changed in some way relative to the mortality patterns demonstrated immediately after the spill. Our methods also illustrate the flexibility and complexity that a source–sink model structure provides to simple population models, and their utility in assessments of environmental impacts on populations subject to nonuniformly distributed stressors.

METHODS

Observations

We collected three types of empirical data relating to the status of the Prince William Sound sea otter population: (1) annual carcass collections in oiled areas to determine the age structure of winter mortality, (2) nearly annual aerial surveys to estimate population size of the WPWS population as a whole, and in oiled and unoiled subareas, and (3) nearly annual captures of live sea otters in both an oiled and unoiled area to monitor population health and indices of exposure, from which we also estimated the age structure of the living population. Carcass collections from 1976 through 1998 have been described (Monson et al. 2000*a*), and similar collections continued through 2008. Green Island (Fig. 1) was the site of pre-spill collections, and we continued to collect carcasses there through 2008. In 1990 and 1991, carcasses were collected opportunistically by beach clean-up and monitoring crews throughout the spill area. Beginning in 1996, in addition to the Green Island collections, we systematically collected carcasses on or near shorelines throughout WPWS that received heavy oiling in 1989, including areas where residual oil was found through at least 2008 (Fig. 1; Short et al. 2004, 2006; USGS, *unpublished data*). Because the sex of dead animals often was not determined, we combined the sexes when examining age-at-death distributions. We collected the skull when present, and we removed a tooth (preferentially a premolar) for age analysis, although we identified pups by open skull sutures and deciduous teeth. Longitudinal sections of each tooth were decalcified for cementum annuli readings, generally providing age estimates ± 1 year (Bodkin et al. 1997). Matson's Laboratory (Milltown, Montana, USA) sectioned and aged all teeth.

We estimated the total population size in WPWS from aerial surveys conducted each year from 1993 (the first year they were conducted) to 2009 (Bodkin and Udevitz 1999, Bodkin et al. 2002). We obtained age distribution information for the living population by capturing animals at both an unoiled site on the northwest coast of Montague Island and within the oiled Knight Island complex (Fig. 1). We captured animals each summer from 1996 to 1998 and then again from 2001 to 2008. Capture methods included both tangle nets and diver-operated traps (Ames et al. 1983). Sea otters were

TABLE 1. Functions used in source–sink models in sea otters (*Enhydra lutris*) in western Prince William Sound (WPWS), Alaska, USA.

Main effect	Functional form	Model calculation
1) Survival rate	$fn_{logit} = \exp(\theta_1 + \theta_2 t + \theta_3 i + \theta_4 ti)$ or $fn_{cloglog} = \exp(\theta_1 + \theta_2 t + \theta_3 i + \theta_4 ti)$ and $fn_{mod.logit} = \exp(\theta_5 + \theta_6 t + \theta_7 i + \theta_8 ti)$	$S_{t,i} = R_{i,sex} \times [fn_{logit}/1 + fn_{logit}]$ $S_{t,i} = R_{i,sex} \times [1 - \exp(-fn_{cloglog})]$ $S_{t,i} = [fn_{mod.logit}/1 + fn_{mod.logit}]^{(\ln(R_{i,sex})/\ln(1/2))}$
2) Sink population size	$y_0 + \theta_9/(1 + \exp[-(i - x_0)/\theta_{10}])$ or θ_9	$N_t = y_0 + \theta_9/(1 + \exp(-(i - x_0)/\theta_{10})$ or $N_t = \theta_9$
3) Shift year	θ_{13} (range 5–18)	$t_{shift} = 20 - \theta_{13}$
4) WPWS N_0	θ_{14}	$N_0 = \theta_{14}$

Notes: Modeled survival rates ($S_{t,i}$) were found by modifying baseline age and sex-specific sea otter survival rates ($R_{i,sex}$) where t is year and i is age, and sex is specific to males and females. Theta is the symbolic representation of the parameters actually estimated in the models (i.e., θ_1 through θ_{14}). The actual estimated values of these parameter values for the single best model are provided in the Appendix. For example, θ_1 represents an intercept parameter, θ_2 is a "time" parameter and modifies survival depending on year since the spill, θ_3 is an "age" parameter and modifies survival depending on animal's age, and θ_4 is a "time" by "age" interaction parameter, and so on. The sink population survival rates were estimated by modifying baseline rates with the logistic or complementary log–log function (cloglog), while source population survival rates were estimated by modifying baseline rates with a modified logistic function. Models also estimated the sink population size at the beginning of each time step (N_t), which was either a constant value through time or was allowed to decline via a sigmoid function. All models used various combinations of these functions to form unique families of models (Table 2). At a minimum, each model uses (1) a source population survival modifier function $S_{t,i,source}$ (modified logistic function with or without $t \times i$ interaction), (2) a sink population survival modifier function $S_{t,i,sink1}$ (logistic or cloglog function with or without $t \times i$ interaction), (3) a sink population size modifier function $N_{sink,i}$ (constant or sigmoidal function), and (4) an intercept term that estimates WPWS population size in 1990 (N_0). The most complex models add (5) a second sink population survival modifier function $S_{t,i,sink2}$ (logistic or cloglog function with or without $t \times i$ interaction) and (6) a parameter to estimate the most likely year (t_{shift}) when the secondary sink population survival function should be applied.

sedated (Monson et al. 2001), and body measurements taken and blood drawn to measure body condition and health of the individuals. For each independent otter, we extracted a premolar for age determination (Bodkin et al. 1997). On the rare occasions when a tooth age was not determined (tooth not available or broken), we used an estimated age at capture, based on tooth wear, morphometrics, and coloration. Each otter >10 kg was also tagged with a uniquely color-coded combination of flipper tags for visual identification. Following reversal from anesthesia, we released sea otters in the same vicinity as captured. We resighted or recaptured some individuals in subsequent years. For the purposes of determining the age distribution of living animals each year, we assumed these individuals resided in their respective areas for all intervening years between initial capture and their last observation or recapture.

Model construction

We constructed source–sink population models using two subpopulation matrices (i.e., a source and a sink subpopulation) connected by emigration/immigration. Within each subpopulation, survival rates varied independently via their own time-varying age- and sex-structured demographic models. Table 1 shows the number of parameters and functional form of each component of the population models. Table 2 describes how the various model components were put together to form the four categories of model architecture examined in this manuscript. See the Appendix for details of the

overall model form. We initialized each model using baseline age and sex-specific survival estimates from smoothed maximum likelihood analyses of ages-at-death based on pre-spill carcass collections (Udevitz et al. 1996, Udevitz and Ballachey 1998). Baseline survival rates (see Appendix) were then modified in post-spill years using the same functional forms described by Monson et al. (2000a) (i.e., logistic and modified logistic), plus the complementary log-log form (cloglog; Table 1). The modified logistic function is obtained by raising the logit function by log(baseline rate)/log(0.5):

$$\text{(logit function)}^{\log(\text{baseline rate})/\log(0.5)}.$$

This function constrains modeled survival rates to vary between {0, 1}, but, unlike the logistic or cloglog functions, the modified logistic function allows modeled survival rates to rise above the estimated baseline rates, as might occur with release from density dependent effects such as food limitation.

Based on the observed population growth in unoiled portions of WPWS, we assumed the spill did not negatively affect source population survival rates. Thus, the model structure needed to allow for growth in the source population. The baseline survival and fecundity rates produce a stable population with lambda (λ) ≈ 1. The modified logistic function allows survival rates to rise above pre-spill baseline values, thus the source population survival rates were only modified using this function, which allows $\lambda \geq 1$. In contrast, the sink

TABLE 2. Description of source–sink models used to examine post-*Exxon Valdez* oil spill (EVOS) changes in sea otter survival in Prince William Sound (PWS).

Model number	Survival rate modifier function†	Number of sink population survival functions	Sink population size	$S_{t,i}$, includes $t \times i$ interaction	Number of model parameters
1a, 1c	sink = logistic	one	stationary	a = no, c = yes	a = 8, c = 10
1b, 1d	sink = logistic	one	sigmoid decline	b = no, d = yes	b = 11, d = 13
2a, 2c	sink = cloglog	one	stationary	a = no, c = yes	a = 8, c = 10
2b, 2d	sink = cloglog	one	sigmoid decline	b = no, d = yes	b = 11, d = 13
3a, 3c	sink1 and 2 = logistic	two	stationary	a = no, c = yes	a = 12, c = 15
3b, 3d	sink1 and 2 = logistic	two	sigmoid decline	b = no, d = yes	b = 15, d = 18
4a, 4c	sink1 and 2 = cloglog	two	stationary	a = no, c = yes	a = 12, c = 15
4b, 4d	sink1 and 2 = cloglog	two	sigmoid decline	b = no, d = yes	b = 15, d = 18

Notes: Models include two population matrices connected by immigration of equal numbers of one- and two-year-old sea otters with 75% of these immigrants being male and 25% female. Models 1 and 2 modify sink population survival rates with one set of parameter values (i.e., single trend), while models 3 and 4 use independent parameter estimates to modify sink population survival rates before and after year t_{shift} (i.e., two survival trends). Models 1 and 3 use the logistic function to modify sink population survival rates, while models 2 and 4 use the cloglog function to modify sink population survival rates. The sink population is held constant in "a" and "c" models but is allowed to change at the beginning of each time step in "b" and "d" models. Survival functions for "a" and "b" models do not include time × age interaction terms (i.e., $S_{t,i} = \theta_1 + \theta_2 t + \theta_3 * i$), while survival functions for "c" and "d" models do include the time × age interaction (i.e., $S_{t,i} = \theta_1 + \theta_2 t + \theta_3 i + \theta_4 ti$).

† Source population survival rates are always modified with the modified logistic function.

population appeared to remain at reduced numbers for at least a decade following the spill, so we assumed that the mortality effects of the spill must be negative, and age-specific post-spill survival must be less than pre-spill values. The logistic and cloglog functional forms only allow modifications that are less than or equal to baseline rates, so we modified survival in the sink population with one of these two functional forms (i.e., potential $\lambda \leq 1$). As a result, this model structure allows for growth in the source population that could potentially counter the negative survival effects expected in the sink population.

We also modeled movement of individuals to connect the source and sink populations, but did so in a simple way, commensurate with the data available to fit the model. In the model, the sink population is constrained to a constant size in a given time step, $t = 1$ year. Each one-year time step begins after the spring birth peak. If changes to the sink population survival rates produced a population decline over a given one-year time step, "immigrants" were moved from the source population into the sink population to counter this loss. Young animals and particularly males are the most likely to emigrate (Kenyon 1969, Garshelis et al. 1984, Riedman and Estes 1990), thus immigrants were drawn from the youngest age classes of the source population (50% one-year-old otters and 50% two-year-old otters) with most drawn from the male pool of these age-classes (75% male and 25% female). We defined direct chronic loss as the cumulative number of immigrants required to maintain the sink population within each one-year time step.

We explicitly examined the possibility that negative spill effects were dissipating through time. To do this, we added features allowing sink population survival rates to return to pre-spill "baseline" values. We hypothesized that a return to pre-spill survival rates could happen in two ways. First, the sink population itself could simply decline through time until the number of negatively

affected individuals becomes small (e.g., because the extent of oil contaminated beaches declined with time through weathering processes), even though the survival effects on those individuals remaining in the sink population are significant. At some point, only the source population survival rates would be in effect and these presumably would be greater than or equal to pre-spill values. This possibility is modeled by allowing the sink population size to begin at a new start value at the beginning of each one-year time step, although within each time step, sink population size is still required to remain stationary (i.e., the source population still provides emigrants at the end of each time step to bring the sink population size back to its start value). We modeled the decline in sink population size with a four-parameter sigmoid function (Table 1). Second, age-specific survival rates in the sink population could begin to improve at some point in time, even if the number of animals affected remains the same. To accommodate this, we created models that allowed a secondary change in sink population survival rates at some year post-spill (t_{shift}) by using two unlinked sink population survival functions each fit independently before and after year t_{shift}. We looked for the most likely year when sink population survival trends may have changed by examining t_{shift} over the range from 2 to 15 years post-spill (Table 1).

The most complex source-sink models (models 3d and 4d; Table 2) allowed time (t), age (i), and time × age-specific changes in survival in both the source population ($S_{t,i-source}$ [intercept + 3 parameters = 4 parameters]) and in the sink population before and after t_{shift} ($S_{t,i-sink1}$ [4 parameters] + $S_{t,i-sink2}$ [4 parameters] + Y [1 parameter] = 9 parameters), and allow the sink population size to change through time (N_t [4 parameters]) for a total of 17 parameters. In addition, all models used one additional parameter to estimate the most likely 1990 WPWS population size (N_0). A large

number of less complex source-sink models could be formed within this framework by leaving out various interactions and main effects, but we only fit the subset of 12 possible model forms, which reflected our judgment of the most biologically plausible alternate descriptions of the population (Table 2). Models numbered 1 and 2 modify sink population survival rates with one set of parameter values, while models numbered 3 and 4 use a second set of parameter values to produce a secondary trend in sink population survival rates after the year t_{shift}. Models with "a" or "c" subscripts held the sink population constant over time, while "b" and "d" models allowed it to change. Models with "a" or "b" subscripts exclude the time $\times$ age interactions in all survival functions, while "c" and "d" models include the interaction in all survival functions. For example, the simplest models (models 1a and 2a; Table 2) left out the time $\times$ age interactions in both source and sink population survival functions [-2 parameters], did not have a secondary sink population survival function or t_{shift} parameter [-5 parameters], and kept the sink population stationary over time [-3 parameters], for a total of $18 - 10 = 8$ parameters. Models 1c and 2c added the time $\times$ age interactions [$+2$ parameters], and progressively more complex models utilized the functions that modified sink population size and post-t_{shift} sink population survival rates (Table 2).

Model fitting

We fit source-sink models by comparing their predictions to four empirical data sets: (1) the ages-at-death of otters dying in the sink population (based on carcass collections from within oiled areas of WPWS; see Supplement), (2) the size of the WPWS sea otter population each year since the EVOS (based on aerial survey data; see Supplement), (3) the age distribution of the living females captured within an oil-affected area (see Supplement), and (4) the age distribution of the living females captured within an unoiled area of WPWS (see Supplement). We calculated the minimum negative log-likelihood ($-L$) values for each data set, and took the sum as the total $-L$ estimate for each model (Hilborn and Mangel 1997, Hobbs and Hilborn 2006, Tinker et al. 2006).

Relative negative log-likelihoods from each year's age-at-death distributions were estimated using multinomial probabilities for observer error (Hilborn and Mangel 1997, Doak and Morris 1999), and the sum of yearly $-L$ values became $-L1$ as follows:

$$-L1 = \sum_{t=1}^{20} \sum_{i=1}^{20} N_{t,i} \times \log P_{t,i} \qquad (1)$$

where $N_{t,i}$ is the number of carcasses of age i collected in year t, and $P_{t,i}$ is the relative frequency of carcasses of age i predicted to be produced in year t by a particular model. Age 0 animals (pups) were removed when calculating relative frequencies and not used in the

calculation of $-L1$ as they tend to be underrepresented in carcass collections because their small size allows them to be easily removed by scavengers (Bodkin and Jameson 1991). This analysis assumes each carcass is an independent sample, which we believe is a reasonable assumption. Sea otters do segregate by sex (Garshelis et al. 1984), and carcasses collected at "male areas" in particular might be considered less than fully independent observations. While we acknowledge this possibility, within our carcass survey area, females of all ages and their pups along with territorial males occupy the vast majority of the habitats with one traditional male resting area utilized by younger males and non-territorial adults located off the east side of Green Island (Fig. 1). However, the male resting area is in close proximity to adjacent female areas, and does not preclude non-territorial males from feeding and traveling through female areas, and we do not believe this segregation represents a significant violation of the assumption of independence.

The population survey-based negative log-likelihood ($-L2$) is the sum of the survey $-L$'s:

$$-L2 = \sum_{t=1}^{20} \log \sigma_t + \frac{1}{2} \log 2\pi + \frac{(N_{\mathrm{obs},t} - N_{\mathrm{exp},t})^2}{2(\sigma_t^2)} \qquad (2)$$

where σ_t^2 is the estimated survey variance from the WPWS survey data in year t, $N_{\mathrm{obs},t}$ is the point estimate for the WPWS population in year t, and $N_{\mathrm{exp},t}$ is the population size predicted with a particular model in year t (Hilborn and Mangel 1997).

Negative log-likelihoods from each year's living female age distributions in both the sink and source population ($-L3$ and $-L4$, respectively) were estimated similarly to the age-at-death $-L$ using multinomial probabilities for observer error:

$$-L3 \text{ and } -L4 = \sum_{t=1}^{20} \sum_{i=1}^{20} N_{t,i} \times \log P_{t,i} \qquad (3)$$

where $N_{t,i}$ is the number of females of age i captured in year t from either the oiled ($-L3$) or unoiled ($-L4$) area, and $P_{t,i}$ is the relative frequency of females of age i predicted to be living in year t in either the oiled ($-L3$) or unoiled ($-L4$) area by a particular model. We removed age 0 otters (pups) when calculating relative frequencies and did not use them in the calculation of $-L3$ or $-L4$ because they are again expected to be underrepresented, as females with small pups tended to avoid net capture, and were sometimes avoided during diving captures.

For each model, we solved for the parameter values that minimize the total negative log-likelihood ($-L_{\mathrm{total}} = -L1 + -L2 + -L3 + -L4$), and then used Akaike's Information Criterion (AIC), ΔAIC, and Akaike weights (w_i) for model selection (Burnham and Anderson 2002, Johnson and Omland 2004). We determined one-dimensional 95% likelihood profile confidence intervals for each parameter in the model by varying a

parameter of interest, while maximizing all other parameters conditional on the value of the parameter of interest. We then plotted the values for the parameter of interest against $-L_{total}$ to find the parameter values where delta $-L_{total} = +1.92$ (Burnham and Anderson 2002). For the best model, we also applied this same procedure to determine the effect of varying the sex ratio of immigrants.

Several predictions of the model (e.g., the chronic loss estimate and source and sink population lambda values) are based on the interactions of multiple parameters in the model and their values are less certain. We approximated the confidence intervals for these predictions by choosing random parameter values from a uniform distribution for each parameter in the model, and determining the $-L_{total}$ value and resulting predictions using these random parameter values. We limited the range of randomly selected values to within the 95% likelihood profile confidence intervals for each parameter to increase the number of outcomes within a reasonably close $-L_{total}$ to the original best estimate (i.e., delta $-L_{total} \leq 5$). We repeated this procedure 10 000 times, and plotted the resulting predictions against $-L_{total}$, using the lower bounds on the likelihood values for each predicted value as an approximation for the likelihood profile; this is essentially a direct search approach to determining the likelihood profile.

We estimated maximum likelihood parameter values for each model using algorithms that explored the entire constrained parameter space. Minimizations were performed in MATLAB (Mathworks 2010) using the optimization toolbox TOMLAB (Holmström 1999). To ensure that global minima were identified, we first used the "glcfast" function, with the results of this routine used as start values for the "snopt" function, a sequential quadratic programming algorithm for large-scale linear or nonlinear optimizations (Gill et al. 1984, Gill et al. 2005). Fitting these complex, nonlinear models to multiple data sets added challenges to finding the "best" parameter estimates, and required use of constraints on some parameters and fitting penalties (Reklaitis et al. 1983) to enforce those constraints, which eliminate the possibility of converging on mathematically possible but biologically unreasonable parameter values. For example, we did not use 0 age animals (dependent pups) in the age-at-death distributions so pup survival could vary between 0 and 1 without affecting the $-L$ values. The modifier function used to control source population survival rates allowed changes within this entire range. However, even though mathematically possible, a survival rate of 0 or 1 is not biologically reasonable. Thus, we put limits to source population survival rates and when these limits were exceeded, a penalty of 1 was added to the $-L$ value. The limits for 0 age survival were determined from empirical estimates obtained from both stable, equilibrium populations (lower limit) and growing, non-food limited populations (upper limit; Monson and DeGange 1995,

Monson et al. 2000b). In addition, we enforced a sink population change in models that allowed it to avoid having the fitting algorithm settle on a small, insignificant population change biologically indistinguishable from models that held the sink population constant. In this case, if the sink population size did not decline by at least 100 over 20 years we added 10 to the $-L$ value, and if it did not decline by at least 250, an additional penalty was added, which was inversely proportional to the size of the change (i.e., the greater the change, the less the penalty). We did this mainly for computational efficiency as the penalty removed the possibility that the fitting processes would settle on local minima, producing small, biologically insignificant declines in sink population size. This approach should not overly constrain the results, as small declines in the sink population should have effects similar to the models where no decline was allowed, and no constraint was placed on finding parameter values that would produce large declines in the sink population size.

After identifying the best model forms and most likely parameter values, we used Kolmogorov-Smirnov (K-S) tests (Sokal and Rohlf 1995) to determine the goodness of fit between the predicted and observed age-at-death distributions summed over two post-spill periods (before and after t_{shift}). We assessed the predicted population trends graphically by plotting the predicted and observed values.

Model application

We had three goals when constructing these models. The first was to estimate the size of the hypothesized sink population, which was an explicit parameter in the model. The second was to estimate chronic losses that may have occurred in the sink population since the EVOS. We assumed the number of immigrants from the source population required to maintain the sink population each year represented direct losses to the sink population, with the sum of these immigrants over all years equaling total chronic loss. In addition, at each time step, we calculated λ absent any immigration/ emigration for each subpopulation given the modeled survival rates based on the dominant eigenvalue of each population matrix. Lambda values changed each year as survival rates changed through time; however, we only contrast λ values for each subpopulation at the end of 2009, assuming the estimated survival rates at that time persist into the future. After finding the best-fit parameter values for each model, we "disconnected" them by removing the emigration/immigration link, allowing the source population to grow without emigration (i.e., without losses to the sink). We subtracted the direct losses (i.e., immigrants) estimated by the linked models from the difference between source population sizes in the linked and unlinked models, and interpreted the remainder as the lost reproductive potential that occurred due to direct losses. Our third goal was to look for an "end point" when spill effects

Fig. 3. Observed age distribution of sea otters found dead on WPWS beaches from 1976 to 2008 (data are provided in the Supplement). Numbers in parentheses are the total number of carcasses in each distribution, and age class 2–8 years is considered "prime-age." Post-spill age distributions are separated by those previously presented in Monson et al. (2000a) and additional carcasses collected since that time. Yearly age-specific distributions are the basis for the age-at-death-derived negative log-likelihood value ($-L1$).
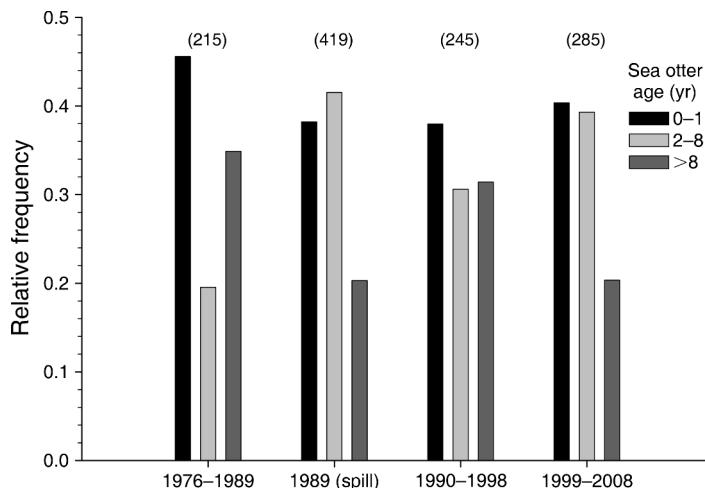
may have decreased to negligible levels. We looked at this in two ways: (1) Is there evidence that survival rates are returning to normal in the sink population? And/or (2) is the size of sink population declining through time?

We used model-averaging methods to arrive at predictions of key parameters over all models fit (Burnham and Anderson 2002). However, the overwhelming support for a single model rendered these results nearly identical to those for the best model, and we concentrate on the single "best" model results in our presentation.

RESULTS

Observations

We recovered 157 sea otter carcasses during pre-spill collections from 1976 to 1985, and another 58 carcasses in 1989 that were deemed pre-spill mortalities (total = 215). From 1990 to 1998, we recovered 245 carcasses, which were the basis for data used in Monson et al. (2000a). From 1999 to 2008, we recovered another 285 carcasses (Supplement). The proportion of prime-age (2–8-year-old) animals in the 1999–2008 collection was high compared to pre-spill collections (0.39 vs. 0.20) and was higher than the proportion observed in the 1990–1998 collection (0.31; Fig. 3). Most importantly, the difference between the pre-spill and 1999 to 2008 age distributions do not suggest a return to pre-spill demographic rates in the second decade following the spill.

We conducted aerial sea otter surveys nearly annually between 1993 and 2009 (Supplement). Growth in the WPWS sea otter population began sometime between 1997 and 2003, with potentially the most rapid increase apparent in 2008 and 2009 (Fig. 4). In contrast, the number of animals residing in the oiled northern Knight Island study area was stable (but below pre-spill numbers) until 2001 when it declined until at least 2005, with no population growth evident until 2007 (Supplement, Bodkin et al. 2002). Population size in the

Knight Island area was variable but averaged ~65 animals over the 20 post-spill years; however, we assumed this area represents the population trend in the hypothesized sink population but not the size of the
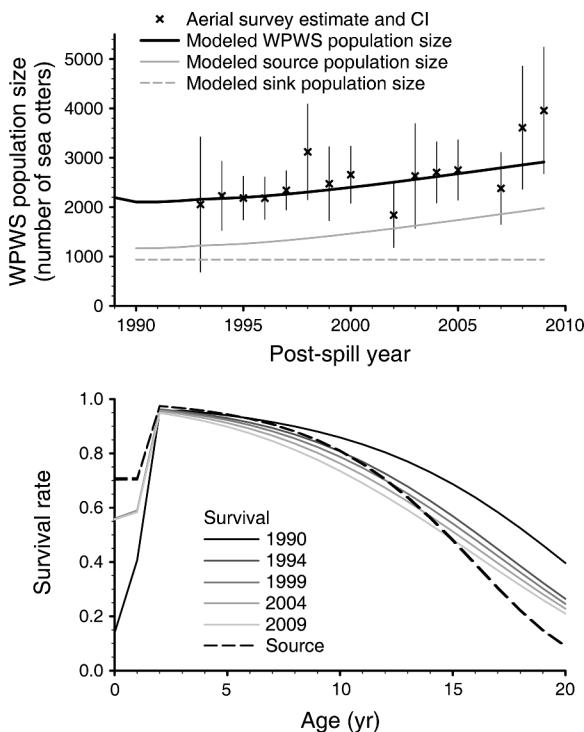

Fig. 4. The top panel illustrates the predicted population trajectories of the best source–sink model (4a) compared with empirical population estimates for WPWS (values provided in the Supplement). The thick black line represents the predicted size of the entire WPWS sea otter population (i.e., source + sink), while the thin gray lines indicate the predicted source (solid gray) and sink (dashed gray) population sizes. The lower panel illustrates the age-specific survival estimates (solid lines represent sink population survival rates, while the dashed line represents the source population survival rates).

TABLE 3. Relative support for 16 source–sink models fit to $-L_{total}$ (the sum for the four negative log-likelihood values).

Model	Number of parameters	$-L$					Based on $-L_{total}$		
		Total	Age-at-death	WPWS survey	KNI live	MON live	AIC_i	Δ_i	w_i
1a	8	2205.9	1041.3	117.7	707.8	339.0	4427.9	13.8	<0.001
1b	11	2207.4	1038.6	118.2	709.6	341.0	4436.8	22.7	<0.001
1c	10	2215.3	1046.4	117.5	713.1	338.2	4450.5	36.4	<0.001
1d	13	2203.1	1038.6	117.6	708.7	338.2	4432.2	18.1	<0.001
2a	8	2205.1	1038.9	117.9	709.4	339.0	4426.2	12.1	0.001
2b	11	2201.9	1037.6	117.1	708.0	339.1	4425.7	11.6	0.001
2c	10	2214.1	1047.7	118.3	710.0	338.1	4448.2	34.1	<0.001
2d	13	2213.2	1053.1	117.7	704.7	337.7	4452.5	38.4	<0.001
3a	12	2199.4	1036.7	118.0	705.7	339.0	4422.8	8.7	0.003
3b	15	2208.7	1044.5	119.1	703.0	342.1	4447.3	33.2	<0.001
3c	15	2203.0	1038.5	120.4	701.1	343.0	4436.1	22.0	<0.001
3d	18	2220.2	1042.1	118.3	721.3	338.5	4476.4	62.3	<0.001
4a	12	2193.4	1037.4	117.8	699.1	339.2	4410.9	0.0	0.996
4b	15	2207.1	1045.3	117.5	702.6	341.7	4444.2	30.1	<0.001
4c	15	2207.2	1040.6	117.8	711.4	337.5	4444.3	30.2	<0.001
4d	18	2224.3	1037.3	116.8	726.7	343.5	4484.6	70.5	<0.001

Note: In this case, $-L1$ is the negative log-likelihood value derived from the age-at-death data, $-L2$ is derived from the entire western Prince William Sound (WPWS) survey data, $-L3$ is derived from the Knight Island (KNI) live female age distribution (assumed to be a sink population), and $-L4$ is derived from the Montague Island (MON) live female age distribution (assumed to be a source population). The sum of these four negative log-likelihood values is $-L_{total}$.

entire sink population, and we did not use these surveys in $-L_{total}$ calculations.

We captured 158 otters in the oiled northern Knight Island complex between 1996 and 2008. Of these, we recaptured 42 individuals 1–3 times for a total of 60 recaptures. We captured 201 otters in the unoiled Montague Island area during this same time. Of these, we recaptured six individuals just one time. The ages of these individuals at capture formed the bases for the living age distribution in the oiled and unoiled areas (see the Supplement).

Source–sink models

Source–sink model 4a produced the best fit (AIC = 4410.9, $w_i = 0.996$), with very little support for any other model (Table 3). The model-averaged results are essentially the same as for model 4a because of the overwhelming support for this model (Table 4). Model

TABLE 4. Individual predictions for 16 evaluated source–sink models.

| Model	1990 WPWS N_0	1990 N_{sink}†	2009 N_{sink}†	λ_{source}	λ_{sink}	Total loss to sink‡	Lost reproductive potential§	t_{shift}	w_i
1a	2003	719 (0.36)	719 (0.18)	1.04	0.92	683	32	NA	<0.001
1b	2241	1817 (0.81)	1529 (0.39)	1.17	0.89	1181	927	NA	<0.001
1c	2196	973 (0.44)	973 (0.25)	1.05	1.00	377	12	NA	<0.001
1d	2279	402 (0.18)	299 (0.08)	1.04	0.95	213	0	NA	<0.001
2a	2000	895 (0.45)	895 (0.23)	1.05	0.90	692	36	NA	0.001
2b	2221	471 (0.21)	32 (0.01)	1.05	0.67	2	0	NA	0.001
2c	2016	863 (0.43)	863 (0.22)	1.01	1.00	370	0	NA	<0.001
2d	2000	216 (0.11)	103 (0.03)	1.02	0.97	96	0	NA	<0.001
3a	2013	891 (0.44)	891 (0.23)	1.06	0.91	893	131	1991	0.003
3b	2154	1305 (0.61)	1050 (0.27)	1.11	1.00	622	578	1998	<0.001
3c	2000	238 (0.12)	238 (0.06)	1.10	0.97	202	36	1995	<0.001
3d	2167	1330 (0.61)	1124 (0.28)	1.04	0.81	1040	326	2004	<0.001
4a	2156	937 (0.43)	937 (0.24)	1.05	0.95	822	78	1994	0.996
4b	2334	339 (0.15)	131 (0.03)	1.04	0.99	89	0	1992	<0.001
4c	2000	890 (0.45)	890 (0.22)	1.05	0.87	382	0	2000	<0.001
4d	2341	1708 (0.73)	1104 (0.28)	1.19	0.52	1383	1903	1991	<0.001
Model average	2156	936 (0.43)	936 (0.24)	1.05	0.95	822	78	1994	

Note: Abbreviations are: N_0, sea otter population size in 1990 (i.e., year 0 for the model runs); λ, instantaneous population growth rate for the source or sink populations at the end of the model runs (2009); t_{shift}, predicted year that changes to survival rates take on a new trajectory (i.e., the second survival rate modifying function takes effect); w_i, Akaike weight value or relative support for each of the models; and NA, not applicable.

† The numbers in parentheses are the final parameter estimate linked to the sink population size prediction, which equates to the proportion of the entire western Prince William Sound population estimated to be in the sink population in 1990 and 2009. This number multiplied by the actual estimated western Prince William Sound population size in these years produces the actual estimated number of sea otters in the sink population in 1990 and 2009, which is the number *not* in parentheses.

‡ The total number of sea otters predicted to have been prematurely lost to the sink population between 1990 and 2009.

§ The predicted additional number of sea otters lost to the sink population due to the lost reproductive potential of the direct losses.

FIG. 5. Relationship between sink population size (N_{sink}) and chronic loss estimates between 1990 and 2009 derived from 10 000 predictions of the best source–sink model (4a) using random values for all parameters chosen between their respective 95% likelihood profile confidence interval values. Symbol fills are shaded based on Akaike weights for each given sink population size value (black, $w_i = 0.11$; dark gray, $w_i = 0.01$–0.05; lighter gray, $w_i = 0.005$–0.01; open, $w_i < 0.005$), and the solid line is the regression through these points weighted by the Akaike weight values.
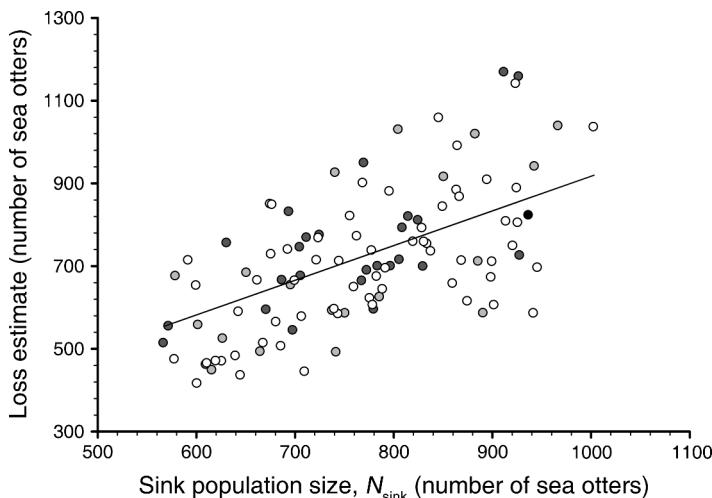
4a suggests the most likely WPWS population size in the spring of 1990 one year after the *EVOS* was ~2156 animals (95% likelihood CI = 1900–2430; Table 4, Appendix), which is in close agreement with the empirical estimates that averaged 1991 (± 606) otters living in the oiled areas of WPWS in the summer of 1990 (Burn 1994), and covers most of our WPWS survey area. More importantly, the model suggests a stable sink population size of ~937 animals (CI = 606 to 960; Table 4; Appendix A). The model further suggests juvenile (0–2 years old) survival rates within the sink population were severely reduced in 1990, but improved to near pre-spill levels by 1994, while prime-age animals (with the exception of two-year old otters) initially had near normal survival rates, from 1990 to 1993, after which their survival rates declined and remained 2–10% below pre-spill values for the next 20 years (Fig. 4). Under the scenario portrayed by this model, the sink population is predicted to experience an average of 125 (range 98 to 137) mortalities per year (excluding pup mortalities), with 40–50 of these deaths considered "premature" by pre-spill baseline survival rate standards (i.e., they would not have occurred had pre-spill survival rates been applied to the sink population). These premature losses result in a cumulative loss estimate of ~820 animals since the spring of 1990 (Table 4). However, chronic loss estimates are influenced by the values of nearly all other parameter estimates, thus reducing our confidence in this particular prediction. In particular, the estimated sink population size has a positive influence on predictions of chronic loss (Fig. 5). We collected an average of 20 (range 6 to 47) non-pup carcasses per year in spill-affected areas. As the model architecture moves mostly males (75% of immigrants) into the sink population to replace these losses, the lost reproductive potential in the source population is minimal and estimated to be <80 individuals over the same 20-year period (Table 4). Fig. 6 indicates that our assumption that 75% of immigrants were male was

reasonable with the best fit $-L_{total}$ for all other potential sex ratios significantly higher.

In contrast, the model suggests that survival rates in the source population were above pre-spill levels for juveniles and essentially unchanged for prime-age animals (the most demographically important age group) throughout the 20 years post-spill (Fig. 4). This dichotomy in prime-age survival rates between the source and sink population results in a source population that would be growing by 5% per year in 2009 when, at the same time, the sink population would be declining by 5% (Table 4). Fig. 7 illustrates the confidence intervals around the 2009 λ estimates for the source and sink populations, based on the 10 000 iterations of randomly selected parameter values. Clearly, the growth potential of the source population was more than enough to counter the losses in the sink population such that by 2009, the model predicts the entire WPWS should have grown by ~1000 animals to an estimated ~3000 individuals. This prediction is below
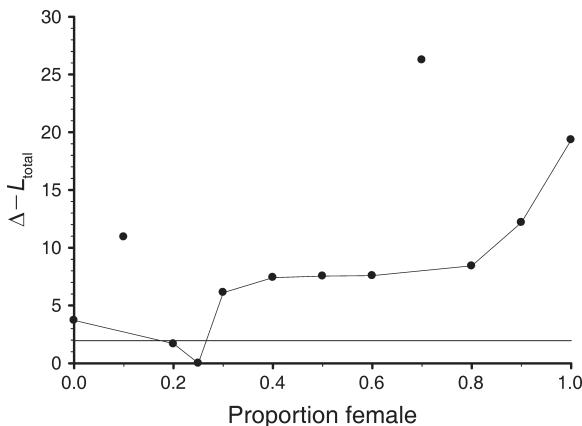

FIG. 6. Likelihood profile intervals for estimate of the sex ratio of immigrants into the sink population. The best model was the assumed 25% females and 75% males ($\Delta - L_{total} = 0$).
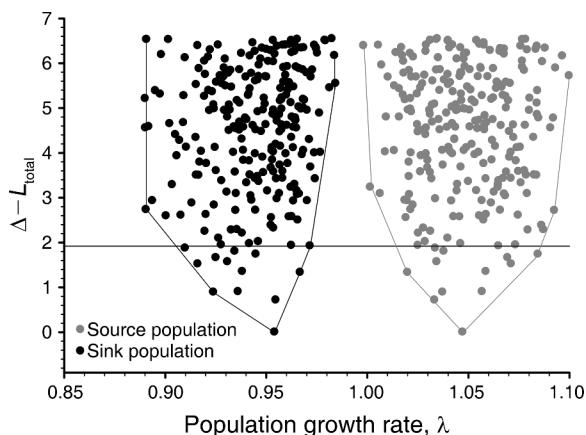
Fig. 7. Likelihood profile intervals for estimates of population growth rate (λ) in the source and sink populations in 2009 based on the best source–sink model (4a). Each point is derived from the $-L_{total}$ values from 10 000 predictions of the best source–sink model (4a) using random values of all parameter estimates chosen from within the likelihood profile confidence intervals of each parameter value. The horizontal line represents the 95% likelihood profile confidence interval ($\Delta-L_{total} \leq 1.9205$).

the actual 2009 empirical WPWS population estimate of nearly 4000 otters, although it is within the survey confidence intervals (Fig. 4).

Goodness of fit

The population trajectories predicted by the source–sink models match the observed population trend in WPWS with the exception that the abundance estimates from surveys indicate population growth in WPWS may have accelerated to higher than predicted levels in 2008 and 2009, although model predictions are within the 95% confidence interval of the survey estimates (Fig. 4). The predicted sink population age-at-death distributions were not significantly different from the observed distributions for either the 1990–1993 or the 1994–2008 periods (K-S test, $D \leq 0.10$, $P > 0.05$; Fig. 8).

DISCUSSION

Monson et al. (2000a) identified significant long-term mortality effects within the spill-affected WPWS sea otter population. The magnitude of these mortality effects would have reduced the WPWS sea otter population to a fraction of its original number within five years post-spill had they been affecting the entire population. Instead, the population as a whole demonstrated slow but steady growth, while a subpopulation of otters inhabiting the most heavily oiled areas remained at reduced numbers for at least 15 years following the spill despite having ample prey resources for population growth. These conflicting outcomes suggested a portion of the WPWS population was acting as a sink (Pulliam 1988, Pulliam and Danielson 1991). Population sinks can be created by a variety of anthropogenic mechanisms including habitat fragmentation or restructuring

(Naranjo and Bodmer 2007, Smith and Person 2007, Feeley and Terborgh 2008, Lees and Peres 2008, Hawlena et al. 2010, 2010, Schaub et al. 2010), and these sinks can represent a significant threat to population persistence depending upon the life-history characteristics of the species (Lees and Peres 2008). Here we have explored the possibility that habitat degradation created by residual oil in the environment created a population sink for sea otters in WPWS. The distribution of residual oil in WPWS is patchy, but where protected from the "cleaning" action of storms, subsurface oil has persisted in a concentrated, relatively unweathered state for at least 20 years (Irvine et al. 1999, 2006, Short et al. 2004, 2006, 2007, 2008). Because of this patchy distribution, it is reasonable to expect that oil exposure levels for nearshore predators such as the sea otter will vary as oiled habitats will not be equally available to all individuals in the population. In addition, the prey preferences of individual sea otters (Estes et al. 2003, Tinker et al. 2007, 2008) imply that exposure levels will not necessarily be equal among individuals with access to oiled habitats.

Here we modeled the WPWS sea otter population as two subpopulations in a source-sink framework. The model outcomes were consistent with earlier models presented by Monson et al. (2000a), which indicate juvenile age classes were most affected for the first few years following the spill with older age classes experiencing declining survival rates through time. We can speculate on a plausible hypothesis explaining this result. Specifically, the first few years following the spill, exposure levels within the sink population likely were relatively high, and pups and juveniles alive or born soon after the spill were the least able to deal with this exposure. Adults that had lived much of their life without oil exposure (the developmental stages of their
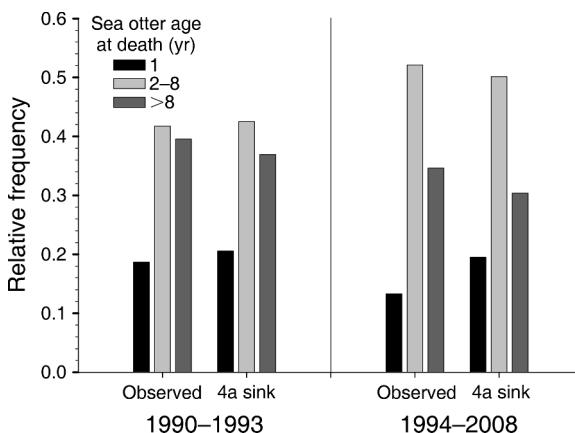

Fig. 8. Observed vs. predicted age-at-death distributions in the sink population for the best source–sink model (4a) for the initial (1990–1993) and secondary (1994–2008) post-spill survival periods. The predicted distributions were not statistically different from the observed distribution in either period (Kolmogorov-Smirnov [K-S] tests, $P > 0.05$ for both).

life in particular) were better able to deal with these high exposures, and thus their survival rates were relatively normal. In contrast, after the first few years of winter storms removed oil from many beaches, exposure levels generally declined and juvenile survival improved. However, otters born within the sink population faced chronic, low level exposures during development (as pups and juveniles), which presumably could lead to decreased survival rates in adulthood.

The model also suggests a stable sink population size from 1990 to 2009, and this number comprised ~43% of the WPWS population in 1990, but only ~20–25% of the WPWS population in 2009 due to growth in the source population. Reductions in survival within the sink population appear to have resulted in the cumulative loss of ~900 animals since 1990, which is of a similar magnitude to the number of acute phase losses documented within WPWS at the time of the spill. Emigrants from the source population moving into and replacing these losses in the sink population would result in additional lost reproductive potential within the source population; however, if most of the emigrants were males (as this model's architecture currently assumes), this effect would be minimal.

Most importantly, we found no indication from this model that mortality effects of the spill have dissipated through time, with estimates of absolute numbers of animals lost annually predicted to be nearly constant from 1994 to 2009. This finding confirms assertions that residual contamination from oil spills can affect wildlife populations far longer than previously believed (Peterson et al. 2003, Bejarano and Michel 2010), and that the magnitude of losses due to chronic effects can be as great as acute phase losses. Our most optimistic time line for the end of oil-related mortality effects comes from comparisons of the predicted and observed sea otter population sizes. The most recent sea otter survey results suggest the WPWS population is growing more rapidly than predicted starting sometime between 2006 and 2008. If true, it could be a sign that mortality effects were finally dissipating sometime between 2005 and 2007. Consistent with this interpretation, the northern Knight Island population finally appeared to experience positive population growth between 2007 and 2009 (Supplement) when the model indicates it should still be declining. Both the magnitude and duration of the mortality effects suggested by our model are similar to those documented in Harlequin Ducks, another near-shore predator of the WPWS system (Esler and Iverson 2010, Iverson and Esler 2010).

These model outcomes are conservative in one particular way: they did not include animals lost in a "secondary mortality event" the first winter following the spill (Burn 1994), which has largely been overlooked. Boat-based survey data suggest a secondary spill-related mortality event occurred, with the loss of several hundred animals during the first winter following the spill. This "delayed" mortality pulse in sea otters is perhaps similar to a delayed mortality event observed for marine iguanas (*Amblyrhychus cristatus*) on the Galápagos island of Santa Fe the year following an oil spill there (Wikelski et al. 2002). These losses likely reflected animals compromised by sublethal oil exposures that were unable to withstand the stressful conditions of the first winter following the spill. Regardless, this study considers the potential for spill-related losses after this first winter's mortality event as the model structure only allows smoothed gradual changes in population size and we did not attempt to model this initial first winter's decline.

Presumably, sea otters continued to be exposed to oil primarily by digging in oiled sediments. Sea otters occupying the Knight Island area utilized intertidal habitats for, on average, nearly 15% of their feeding dives, and at peak times in the spring, for up to 45% of feeding dives (USGS, *unpublished data*). The intertidal foraging activity of a sink population of ~900 individuals implies that collectively these animals have excavated literally millions of intertidal pits over the more than two decades since the spill. Any physical disturbance to lower intertidal sediments will expose subsurface oil and enhance microbial biodegradation, and sea otters, with their physical digging of pits in the intertidal, are likely the most effective mitigation process that has been active continuously in Prince William Sound for the last 20 and more years.

Large-scale anthropogenic contamination events like the EVOS will almost never affect all individuals in a population equally because long-term, chronic exposure levels will be heterogeneous. This heterogeneity stems from the likely patchy distribution of residual contaminants, and the mobility and spatial distribution of individuals of various species. Even where all individuals have equal access to contaminated habitats, exposure levels will vary with individual habitat use and prey preferences (Estes et al. 2003, Tinker et al. 2007, 2008). This heterogeneity in exposure levels is an important consideration as it helps explain why detecting chronic effects, and linking them to contamination events, can be so difficult.

Variable exposure levels can lead to variable health effects ranging from none to subclinical to clinical and even death. It is only with the final two stages that direct connections are easily made between health and contaminants in the environment. However, the probability of sampling clinically sick animals in the wild is low as individual residence time within this stage is likely short. Furthermore, in remote areas, the probability of finding animals that have recently died also is extremely low. However, we can use the record left behind by the dying animals in the form of the age distribution at death to inform demographic models. By incorporating additional data sets in the fitting process, such as information on the living population age structure and population trend, we can examine more detailed and complex candidate models. Future environmental catas-

trophes are inevitable, as demonstrated by the "Deepwater Horizon" spill in the Gulf of Mexico in 2010. The lessons learned from the EVOS, and the potential for the creation of areas that become population sinks and limit the recovery of endangered or otherwise important species, should be at the forefront of planning for research into the potential long-term effects of this or other future widespread contamination events.

LITERATURE CITED

Ames, J. A., R. A. Hardy, and F. E. Wendell. 1983. Tagging materials and methods for sea otters, *Enhydra lutris*. California Fish and Game 69:243–252.

Ballachey, B. E., J. L. Bodkin, and A. R. DeGange. 1994. An overview of sea otter studies. Pages 47–59 in T.R. Loughlin, editor. Marine mammals and the *Exxon Valdez*. Academic Press, San Diego, California.

Ballachey, B. E., J. L. Bodkin, S. Howlin, A. M. Doroff, and A. H. Rebar. 2003. Correlates to survival of juvenile sea otters in Prince William Sound, Alaska, 1992-1993. Canandian Journal of Zoology 81:1494–1510.

Bejarano, A. C., and J. Michel. 2010. Large-scale risk assessment of polycyclic aromatic hydrocarbons in shoreline sediments from Saudi Arabia: Environmental legacy after twelve years of the Gulf war oil spill. Environmental Pollution 158:1561–1569.

Bodkin, J. L., J. A. Ames, R. J. Jameson, A. M. Johnson, and G. M. Matson. 1997. Estimating age of sea otters with cementum layers in the first premolar. Journal of Wildlife Management 61:967–973.

Bodkin, J. L., B. E. Ballachey, T. A. Dean, A. K. Fukuyama, S. C. Jewett, L. McDonald, D. H. Monson, C. E. O'Clair, and G. R. VanBlaricom. 2002. Sea otter population status and the process of recovery from the 1989 *Exxon Valdez* oil spill. Marine Ecology Progress Series 241:237–254.

Bodkin, J. L., and R. J. Jameson. 1991. Patterns of seabird and marine mammal carcass deposition along the central California coast, 1980-1986. Canadian Journal of Zoology 69:1149–1155.

Bodkin, J. L., D. Mulcahy, and C. Lensink. 1993. Age specific reproduction in female sea otters (*Enhydra lutris*) from south-central Alaska: analysis of reproductive tracts. Canadian Journal of Zoology 71:1811–1815.

Bodkin, J. L., and M. S. Udevitz. 1999. An aerial survey method to estimate sea otter abundance. Pages 13–26 in G. W. Garner, S. C. Amstrup, J. L. Laake, B. F. J. Manly, L. L. McDonald, and D. G. Robertson, editors. Marine mammal survey and assessment methods. Balkema, Rotterdam, The Netherlands.

Bowyer, R. T., G. M. Blundell, M. Ben-David, S. C. Jewett, T. A. Dean, and L. K. Duffy. 2003. Effects of the *EXXON VALDEZ* oil spill on river otters: Injury and recovery of a sentinel species. Wildlife Monographs 153:1–53.

Burn, D. M. 1994. Boat-based population surveys of sea otters in Prince William Sound. Pages 61–80 in T. R. Loughlin, editor. Marine mammals and the *Exxon Valdez*. Academic Press, San Diego, California, USA.

Burnham, K. P., and D. R. Anderson. 2002. Model selection and inference: a practical information-theoretic approach. Second edition. Springer-Verlag, New York, New York, USA.

Costa, D. P., and G. L. Kooyman. 1982. Oxygen consumption, thermoregulation, and the effect of fur oiling and washing on the sea otter, *Enhydra lutris*. Canadian Journal of Zoology 60:2761–2767.

Dean, T. A., J. L. Bodkin, A. K. Fukuyama, S. C. Jewett, D. H. Monson, C. E. O'Clair, and G. R. VanBlaricom. 2002. Food limitation and the recovery of sea otters following the *Exxon Valdez* oil spill. Marine Ecology Progress Series 241:155–270.

DeGange, A. R., A. M. Doroff, and D. H. Monson. 1994. Experimental recovery of sea otter carcasses at Kodiak Island, Alaska, following the *Exxon Valdez* oil spill. Marine Mammal Science 10:492–496.

Doak, D. F., and W. F. Morris. 1999. Detecting population-level consequences of ongoing environmental change without long-term monitoring. Ecology 80:1537–1551.

Dunning, J. B., B. J. Danielson, and H. R. Pulliam. 1992. Ecological processes that affect populations in complex landscapes. Oikos 65:169–175.

Esler, D., T. D. Bowman, K. A. Trust, B. E. Ballachey, T. A. Dean, S. C. Jewett, and C. E. O'Clair. 2002. Harlequin duck population recovery following the "*Exxon Valdez*" oil spill: progress, process and constraints. Marine Ecology Progress Series 241:271–286.

Esler, D., and S. A. Iverson. 2010. Female harlequin duck winter survival 11 to 14 years after the *Exxon Valdez* oil spill. Journal of Wildlife Management 74:471–478.

Esler, D., J. A. Schmutz, R. L. Jarvis, and D. M. Mulcahy. 2000. Winter survival of adult female harlequin ducks in relation to history of contamination by the *Exxon Valdez* oil spill. Journal of Wildlife Management 64:839–847.

Esler, D., K. A. Trust, B. E. Ballachey, S. A. Iverson, T. L. Lewis, D. J. Rizzolo, D. M. Mulcahy, A. K. Miles, B. R. Woodin, J. J. Stegeman, J. D. Henderson, and B. W. Wilson. 2010. Cytochrome P4501A biomarker indication of oil exposure in harlequin ducks up to 20 years after the *Exxon Valdez* oil spill. Environmental Toxicology and Chemistry 29:1138–1145.

Estes, J. A., M. L. Riedman, M. M. Staedler, M. T. Tinker, and B. E. Lyon. 2003. Individual variation in prey selection by sea otters: patterns, causes and implications. Journal of Animal Ecology 72:144–155.

Feeley, K. J., and J. W. Terborgh. 2008. Direct versus indirect effects of habitat reduction on the loss of avian species from tropical forest fragments. Animal Conservation 11:353–360.

Fukuyama, A. K., G. Shigenaka, and R. Z. Hoff. 2000. Effects of residual *Exxon Valdez* oil on intertidal *Prototheca staminea*: mortality, growth, and bioaccumulation of hydrocarbons in transplanted clams. Marine Pollution Bulletin 40:1042–1050.

Garrott, R. A., L. L. Eberhardt, and D. M. Burn. 1993. Mortality of sea otters in Prince William Sound following the *Exxon Valdez* oil spill. Marine Mammal Science 9:343–359.

Garshelis, D. L. 1997. Sea otter mortality estimated from carcasses collected after the *Exxon Valdez* oil spill. Conservation Biology 11:905–916.

Garshelis, D. L., A. M. Johnson, and J. A. Garshelis. 1984. Social organization of sea otters in Prince William Sound, Alaska. Canadian Journal of Zoology 62:2648–2658.

Gill, P. E., W. Murray, and M. A. Saunders. 2005. SNOPT: An SQP algorithm for large-scale constrained optimization. SIAM Review 47:99–131.

Gill, P. E., W. Murray, M. A. Saunders, and M. H. Wright. 1984. Procedures for optimization problems with a mixture of bounds and general linear constraints. ACM Transaction on Mathematical Software 10:282–298.

Golet, G. H., P. E. Seiser, A. D. McGuire, D. D. Roby, J. B. Fischer, K. J. Kuletz, D. B. Irons, T. A. Dean, S. C. Jewett, and S. H. Newman. 2002. Long-term direct and indirect effects of the 'Exxon Valdez' oil spill on pigeon guillemots in Prince William Sound, Alaska. Marine Ecology Progress Series 241:287–304.

Hawlena, D., D. Saltz, Z. Abramsky, and A. Bouskila. 2010. Ecological trap for desert lizards caused by anthropegenic changes in habitat structure that favor predator activity. Conservation Biology 24:803–809.

Hayes, M. O., and J. Michel. 1999. Factors determining the long-term persistence of Exxon Valdez oil in gravel beaches. Marine Pollution Bulletin 38:92–101.

Heinrichs, J. A., D. J. Bender, D. L. Gummer, and N. H. Schumaker. 2010. Assessing critical habitat: Evaluating the relative contribution of habitats to population persistence. Biological Conservation 143:2229–2237.

Hilborn, R., and M. Mangel. 1997. The ecological detective. Princeton University Press, Princeton, New Jersey, USA.

Hobbs, N. T., and R. Hilborn. 2006. Alternatives to statistical hypothesis testing in ecology: a guide to self teaching. Ecology 16:5–19.

Holmström, K. 1999. The Tomlab optimization environment in Matlab. Advanced Modeling and Optimization 1:47–69.

Irvine, G. V., D. H. Mann, and J. W. Short. 1999. Multi-year persistence of oil mousse on high energy beaches distant from the Exxon Valdez spill origin. Marine Pollution Bulletin 52:572–584.

Irvine, G. V., D. H. Mann, and J. W. Short. 2006. Persistence of 10-year old Exxon Valdez oil on Gulf of Alaska beaches: The importance of boulder-armoring. Marine Pollution Bulletin 52:1011–1022.

Iverson, S. A., and D. Esler. 2010. Harlequin duck population injury and recovery dynamics following the 1989 Exxon Valdez oil spill. Ecological Applications 20:1993–2006.

Jewett, S. C., T. A. Dean, B. R. Woodin, M. K. Hoberg, and J. J. Stegeman. 2002. Exposure to hydrocarbons 10 years after the Exxon Valdez oil spill: evidence from cytochrome P4501A expression and biliary FACs in nearshore demersal fishes. Marine Environmental Research 54:21–48.

Johnson, J. B., and K. S. Omland. 2004. Model selection in ecology and evolution. Trends in Ecology and Evolution 19:101–108.

Kenyon, K. W. 1969. The sea otter in the Eastern Pacific ocean. North American Fauna Number 68. Bureau of Sport and Fisheries Wildlife, Washington, D.C., USA.

Lees, A. C., and C. A. Peres. 2008. Avian life-history determinants of local extinction risk in a hyper-fragmented neotropical forest landscape. Animal Conservation 11:128–137.

Li, H., and M. C. Boufadel. 2010. Long-term persistence of oil from the Exxon Valdez spill in two-layer beaches. Nature Geosciences 3:96–99.

Lipscomb, T. P., R. K. Harris, R. B. Moeller, J. M. Pletcher, R. J. Haebler, and B. E. Ballachey. 1993. Histopathologic lesions in sea otters exposed to crude oil. Veterinary Pathology 30:1–11.

Lipscomb, T. P., R. K. Harris, A. H. Rebar, B. E. Ballachey, and R. J. Haebler. 1994. Pathology of sea otters. Pages 265–280 in T. R. Loughlin, editor. Marine Mammals and the Exxon Valdez. Academic Press, San Diego, California, USA.

Loughlin, T. R., B. E. Ballachey, and B. A. Wright. 1996. Overview of studies to determine injury caused by the Exxon Valdez oil spill to marine mammals. Pages 798–808 in S. D.

Rice, R. B. Spies, D. A. Wolfe, and B. A. Wright, editors. Proceedings of the Exxon Valdez oil spill symposium. AFS Symposium 18. American Fisheries Society, Bethesda, Maryland, USA.

Mathworks. 2010. MATLAB. Version 7.11 (R2010b). Mathworks, Natick, Massachusetts, USA.

Monnett, C., and L. M. Rotterman. 1995. Movements of weanling and adult female sea otters in Prince William Sound, Alaska, after the T/V Exxon Valdez oil spill. Exxon Valdez Oil Spill State/Federal Natural Resource Damage Assessment Final Report. Marine Mammal Study 6-12. U.S. Fish and Wildlife Service, Anchorage, Alaska, USA.

Monson, D. H., and A. R. DeGange. 1995. Reproduction, preweaning survival, and survival of adult sea otters at Kodiak Island, Alaska. Canadian Journal of Zoology 73:1161–1169.

Monson, D. H., D. F. Doak, B. E. Ballachey, A. Johnson, and J. L. Bodkin. 2000a. Long-term impacts of the Exxon Valdez oil spill on sea otters, assessed through age-dependent mortality patterns. Proceedings of the National Academy of Sciences USA 97:6562–6567.

Monson, D. H., J. A. Estes, J. L. Bodkin, and D. B. Siniff. 2000b. Life history plasticity and population regulation in sea otters. Oikos 90:457–468.

Monson, D. H., C. McCormick, and B. E. Ballachey. 2001. Chemical anesthesia of northern sea otters (Enhydra lutris): results of past field studies. Journal of Zoo and Wildlife Medicine 32:181–189.

Morris, B. F., and T. R. Loughlin. 1994. Overview of the Exxon Valdez Oil Spill, 1989-1992. Pages 1–22 in T. R. Loughlin, editor. Marine mammals and the Exxon Valdez. Academic Press, San Diego, California, USA.

Naranjo, E. J., and R. E. Bodmer. 2007. Source-sink systems and conservation of hunted ungulates in the Lacandon Forest, Mexico. Biological Conservation 138:412–420.

Peterson, C. H., and L. Holland-Bartels. 2002. Chronic impacts of oil pollution in the sea: risks to vertebrate predators. Marine Ecology Progress Series 241:235–236.

Peterson, C. H., S. D. Rice, J. W. Short, D. Esler, J. L. Bodkin, B. E. Ballachey, and D. B. Irons. 2003. Long-term ecosystem response to the Exxon Valdez oil spill. Science 302:2082–2086.

Piatt, J. F., C. J. Lensink, W. Butler, M. Kendziorek, and D. R. Nyeswander. 1990. Immediate impact of the Exxon Valdez oil spill on marine birds. Auk 107:387–397.

Pulliam, R. H. 1988. Sinks, and population regulation. American Naturalist 132:652–661.

Pulliam, R. H., and B. J. Danielson. 1991. Sources, sinks, and habitat selection: a landscape perspective on population dynamics. American Naturalist 137:S50–S66.

Rebar, A. H., T. P. Lipscomb, R. K. Harris, and B. E. Ballachey. 1995. Clinical and clinical laboratory correlates in sea otters dying unexpectedly in rehabilitation centers following the Exxon Valdez oil spill. Veterinary Pathology 32:346–350.

Reklaitis, G. V., A. V. Ranindran, and K. M. Ragsdell. 1983. Engineering optimization. Methods and applications. Wiley-Interscience Publication, New York, New York, USA.

Ricca, M. A., A. K. Miles, B. E. Ballachey, J. L. Bodkin, D. Esler, and K. A. Trust. 2010. PCB exposure in sea otters and harlequin ducks in relation to history of contamination by the Exxon Valdez oil spill. Marine Pollution Bulletin 60:861–872.

Riedman, M. L., and J. A. Estes. 1990. The sea otter (Enhydra lutris): behavior, ecology and natural history. Biology Report Number 90. U.S. Fish and Wildlife Service, Washington, D.C., USA.

Schaub, M., A. Aebischer, O. Bimenez, S. Berger, and R. Ariettaz. 2010. Massive immigration balances high anthropogenic mortality in a stable eagle owl population: Lessons for conservation. Biological Conservation 143:1911–1918.

Short, J. W., and M. M. Babcock. 1996. Pre-spill and post-spill concentrations of hydrocarbons in mussels and sediments in Prince William Sound. Pages 149–166 in S. D. Rice, R. B. Spies, D. A. Wolfe, and B. A. Wright, editors. Proceedings of the *Exxon Valdez* oil spill symposium. AFS Symposium 18. American Fisheries Society, Bethesda, Maryland, USA.

Short, J. W., G. V. Irvine, D. H. Mann, J. M. Maselko, J. J. Pella, M. R. Lindeberg, J. R. Payne, W. B. Driskell, and S. D. Rice. 2007. Slightly weathered *Exxon Valdez* oil persists in Gulf of Alaska beach sediments after 16 years. Environmental Science and Technology 41:1245–1250.

Short, J. W., M. R. Lindeberg, P. A. Harris, J. M. Maselko, J. J. Pella, and S. D. Rice. 2004. Estimate of oil persisting on the beaches of Prince William Sound 12 years after the *Exxon Valdez* oil spill. Environmental Science and Technology 38:19–25.

Short, J. W., J. M. Maselko, M. R. Lindeberg, P. M. Harris, and S. D. Rice. 2006. Vertical distribution and probability of encountering intertidal *Exxon Valdez* oil on shorelines of three embayments within Prince William Sound, Alaska. Environmental Science and Technology 40:3723–3729.

Short, J. W., K. R. Springman, M. R. Lindeberg, L. G. Holland, M. L. Larsen, C. A. Sloan, C. Kran, P. V. Hodson, and S. D. Rice. 2008. Semipermeable membrane devices link site-specific contaminants to effects. PART II: A comparison of lingering Exxon Valdez oil with other potential sources of CYP1A inducers in Prince William Sound, Alaska. Marine Environmental Research 66:487–498.

Siniff, D. B., T. D. Williams, A. M. Johnson, and D. L. Garshelis. 1982. Experiments on the response of sea otters, *Enhydra lutris*, to oil contamination. Biological Conservation 23:261–272.

Smith, W. P., and D. K. Person. 2007. Estimated persistence of northern flying squirrel population in temperate rain forest fragments of Southeast Alaska. Biological Conservation 137:626–636.

Sokal, R. R., and F. J. Rohlf. 1995. Biometry. Third edition. W. H. Freeman and Company, New York, New York, USA.

Spies, R. B., S. D. Rice, D. A. Wolfe, and B. A. Wright. 1996. The effects of the *Exxon Valdez* Oil Spill on the Alaskan coastal environment. Pages 1–16 in S. D. Rice, R. B. Spies, D. A. Wolfe, and B. A. Wright, editors. Proceedings of the *Exxon Valdez* oil spill symposium. AFS Symposium 18. American Fisheries Society, Bethesda, Maryland, USA.

Tinker, M. T., G. Bentall, and J. A. Estes. 2008. Food limitation leads to behavioral diversification and dietary specialization in sea otters. Proceedings of the National Academy of Sciences USA 105:560–565.

Tinker, M. T., D. P. Costa, J. A. Estes, and N. Wieringa. 2007. Individual dietary specialization and dive behaviour in the California sea otter: Using archival time-depth data to detect alternative foraging strategies. Deep-Sea Research II 54:303–342.

Tinker, M. T., D. F. Doak, J. A. Estes, B. B. Hatfield, M. M. Staedler, and J. L. Bodkin. 2006. Incorporating diverse data and realistic complexity into demographic estimation procedures for sea otters. Ecological Applications 16:2293–2312.

Trust, K. A., D. Esler, B. R. Woodin, and J. J. Stegeman. 2000. Cytochrome P450 1A induction in sea ducks inhabiting nearshore areas of Prince William Sound, Alaska. Marine Pollution Bulletin 40:397–403.

Udevitz, M. S., and B. E. Ballachey. 1998. Estimating survival rates with age-structure data. Journal of Wildlife Management 62:779–792.

Udevitz, M. S., B. E. Ballachey, and D. L. Bruden. 1996. A population model for sea otters in western Prince William Sound. Exxon Valdez Oil Spill State/Federal Restoration Final Report. Restoration Study 93043-3. National Biological Service, Anchorage, Alaska, USA.

Wikelski, M., V. Wong, B. Chevalier, N. Rattenborg, and H. L. Snell. 2002. Marine iguanas die from trace oil pollution. Nature 417:607–608.

Williams, T. M., R. A. Kastelein, R. W. Davis, and J. A. Thomas. 1988. The effects of oil contamination and cleaning on sea otters (*Enhydra lutris*). I. thermoregulatory implications based on pelt studies. Canadian Journal of Zoology 66:2776–2781.

Wolfe, D. A., M. J. Hameedi, J. A. Galt, G. Watabayashi, J. W. Short, C. O'Clair, S. D. Rice, J. Michel, J. R. Payne, J. Braddock, S. Hanna, and D. Sale. 1994. The fate of the oil spilled from the Exxon Valdez. Environmental Science and Technology 28:561–568.

APPENDIX

Form of the transition matrix model and modifier functions used to model source and sink population demographic rates, along with baseline survival and fecundity rates and final "best-model" parameter estimates (*Ecological Archives* A021-131-A1).

SUPPLEMENT

All data used to fit modifications to source and sink population survival rates (*Ecological Archives* A021-131-S1).