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MORTALITY SENSITIVITY IN LIFE-STAGE SIMULATION ANALYSIS: A CASE STUDY OF SOUTHERN SEA OTTERS

LEAH R. GERBER,[1,2,6] M. TIM TINKER,[3] DANIEL F. DOAK,[3] JAMES A. ESTES,[3,4] AND DAVID A. JESSUP[5]

[1]*School of Life Sciences, Arizona State University P.O. Box 874501, Tempe, Arizona 85287-4501 USA*
[2]*National Center for Ecological Analysis and Synthesis University of California, 735 State Street, Suite 300, Santa Barbara, California 93101-3351 USA*
[3]*Department of Ecology and Evolutionary Biology, University of California, A-316 Earth & Marine Sciences Building, Santa Cruz, California 95064 USA*
[4]*U.S. Geological Survey, Center, for Ocean Health, 100 Shaffer Rd., University of California, Santa Cruz, California 95060 USA*
[5]*California Department of Fish and Game, Marine Wildlife Veterinary Care and Research Center, 1451 Shaffer Rd., Santa Cruz, California 95060 USA*

Abstract. Currently, there are no generally recognized approaches for linking detailed mortality and pathology data to population-level analyses of extinction risk. We used a combination of analytical and simulation-based analyses to examine 20 years of age- and sex-specific mortality data for southern sea otters (*Enhydra lutris*), and we applied results to project the efficacy of alternative conservation strategies. Population recovery of the southern sea otter has been slow (rate of population increase $\lambda = 1.05$) compared to other recovering populations ($\lambda = 1.17$–1.20), and the population declined ($\lambda = 0.975$) between 1995 and 1999. Age-based Leslie matrices were developed to explore explanations for the slow recovery and recent decline in the southern sea other population. An elasticity analysis was performed to predict effects of proportional changes in stage-specific reproductive or survival rates on the rate of population increase. A life-stage simulation analysis (LSA) was developed to evaluate the impact of changing age- and cause-specific mortality rates on λ. The information used to develop these models was derived from death assemblage, pathology, and live population census data to examine the sensitivity of sea otter population growth to different sources of mortality (e.g., disease and starvation, direct human take [fisheries, gun shot, boat strike, oil pollution], mating trauma and intraspecific aggression, shark bites, and unknown). We used resampling simulations to generate random combinations of vital rates for a large number of matrix replicates and drew on these to estimate potential effects of mortality sources on population growth (λ). Our analyses suggest management actions that are likely and unlikely to promote recovery of the southern sea otter and more broadly indicate a methodology to better utilize cause-of-death data in conservation decision-making.

Key words: *age specific; cause of mortality; conservation; demography; disease;* Enhydra lutris; *life stage simulation analysis; population growth; sea otters; sex specific.*

INTRODUCTION

Age-specific mortality rates in long-lived species can be estimated in three ways: by following cohorts; by sampling the age structure of living populations; and by examining death assemblages (Caughley 1966, Caughley and Sinclair 1994, Ebert 1998). Mortality estimates obtained by the first two of these approaches usually lack detailed information about the actual cause of death because neither the death event itself nor the resulting carcass is necessarily observed. Death assemblages, in contrast, can provide extensive information on cause of mortality, especially when freshly dead individuals are recovered and examined. This latter approach has been used in a number of demographic studies (e.g., Dalls sheep, Caughley [1966]; California Condor, Meretsky et al. [2000]; molluscs, Warwick and Light [2002]). All three approaches assume that the individuals sampled are representative of the age structure of live populations. This assumption is least problematic for the cohort approach, which is carried out on living populations that may vary in condition across years, but requires numerous assumptions about the stability of demographic rates through time. It can be more problematic for static samples of living populations if these populations are not at a stable age distribution. The assumption is most problematic for the analysis of death assemblages because animals that die for different reasons often have unequal probabilities of recovery. For instance, an individual killed and eaten by a predator may be less likely to be found than one that died of starvation. Accounting for this uncertainty is among the greatest challenges in conducting demographic analyses based on the detailed mortality schedules that can be derived from death assemblages.

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[6] E-mail: leah.gerber@asu.edu

Currently, there are no generally recognized approaches for linking detailed mortality and pathology data to population-level analyses of extinction risk. A number of useful tools have been developed to measure potential effects of uncertainty and variation in vital rates on population growth, making optimal use of limited demographic data for the purposes of species conservation planning (e.g., Gerber and DeMaster 1999, Gross 2000, reviewed in Morris and Doak 2002). One such approach is life-stage simulation analysis (LSA), which relies on specified plausible or hypothesized levels of uncertainty, variation, and covariation in vital rates to create random combinations of vital rates for a large number of simulated matrix replicates. These demographic schedules are then used to estimate potential effects of each vital rate on λ (Wisdom et al. 2000). Here, we extend the LSA approach, using age- and cause-specific mortality data and limited demographic information to conduct sensitivity analyses for a variety of mortality sources for southern sea otters, thereby providing a framework for exploring and identifying management options for this population.

The Southern sea otter (*Enhydra lutris*) provides an interesting case study for examining the use of mortality data in population viability and management assessments because long-term data on reproduction, mortality, and population size are available from carcass recovery efforts; necropsies of fresh and decomposed carcasses have been conducted systematically for over 20 years, and range-wide population surveys have been conducted regularly over the same period (Estes et al. 2003). The southern sea otter population, once thought to be extinct, has recovered slowly over the past 100 years. Despite this recovery, the population is currently classified as threatened under the Endangered Species Act, depleted under the Marine Mammal Protection Act, and as a ''fully protected mammal'' under California state law (U.S. Fish and Wildlife Service 2000). By 1995 the population was thought to have recovered to a point where delisting under the Endangered Species Act was imminent (Ralls et al. 1996). Between 1995 and 2001, however, population recovery ceased.

There are several potential explanations for the recent change in the sea otter population. The California Department of Fish and Game (CDF&G) and the Biological Resources Division of the U.S. Geological Survey (USGS) have maintained a salvage network to collect beach-cast carcasses of sea otters since 1968 (Estes et al. 2003). Known causes of mortality include incidental losses in coastal fisheries (net drowning), shark predation, boat strikes, oil pollution, shooting, fighting and mating activities, fungal infections, parasites, and emaciation (Ames and Morejohn 1980, Riedman et al. 1994, Estes et al. 2003). An increase in any one of these sources of mortality could explain the observed change in sea otter population growth. Disease and parasite loading also may have contributed to the slow rate of population growth in the California population. Thomas and Cole (1996) documented infectious disease and parasites in 40% of freshly dead stranded animals. Lafferty and Gerber (2002) found that the percentage of otter mortality from acanthocephalan peritonitis in a particular year was negatively associated with population growth rates in the following year, suggesting that this source of mortality may directly contribute to variation in sea otter population trends. Otters become infected with acanthocephalan worms (*Polymorphus kenti*, a shorebird parasite) by consuming the intermediate-host sand crab, *Emerita analoga* (Thomas and Cole 1996). However, before focusing management efforts on disease-related issues, or indeed on any source of mortality, it is important to consider the likely effects of such efforts on population dynamics.

In this paper we seek to evaluate the relative importance of putative sources of mortality on Southern sea otters and to identify key information gaps in the ongoing necropsy and salvage programs.

Methods

A stochastic age-structured population model was developed to examine the sensitivity of sea otter population growth to different sources of mortality. We used demographic data from resampling simulations to establish random combinations of vital rates for a large number of matrix replicates, and employed these to estimate the potential effects of each mortality source on λ, the annual rate of population change. The sensitivity of the population to a particular source of mortality was indexed by the proportion of λ explained by the source of mortality. Because a substantial proportion of the estimated mortality in California sea otters can not be accounted for in the recovered beach-cast carcasses, the analyses are repeated using simulated data sets in which all dead animals (recovered and nonrecovered) are included to compare the sensitivity of λ to unrecovered carcasses, relative to the sensitivity of λ to recovered carcasses. This latter analysis provides a measure of the potential effect of biases in recovery rates on our results, in turn yielding an assessment of the degree to which management by mortality mitigation might be expected to influence future population trajectories.

The database

The CDF&G began recording stranded sea otters carcasses in California in 1968 (Estes et al. 2003). Sex, age class, cause of death, size, condition, and recovery location were recorded to the maximum possible degree (for many carcasses, sex and cause of death were impossible to determine because of their deteriorated condition). Veterinary pathologists examined nearly all fresh carcasses after 1992, and this subsample includes more detailed information on cause of death. For our analysis, we collapsed the 16 specific causes of death

TABLE 1. Categories for cause of death assigned to beach-cast sea otter carcasses, and numbers of deaths for each category by age class and time period.

Sea otter age class and years	Disease and starvation†	Shark bites‡	Trauma, interspecific§	Trauma, human-caused‖	Other¶	Unknown#
Pup/immature						
1968–1979	2	4	21	2	9	115
1980–1984	1	3	10	9	16	157
1985–1989	1	0	0	7	15	67
1990–1994	34	0	13	1	9	105
1995–1999	42	9	11	0	7	186
Subadult						
1968–1979	2	24	19	11	18	51
1980–1984	3	10	3	14	9	63
1985–1989	5	8	3	13	5	36
1990–1994	13	11	1	7	5	29
1995–1999	30	6	1	2	11	62
Adult						
1968–1979	12	31	21	14	21	156
1980–1984	5	27	13	26	21	184
1985–1989	12	18	6	23	23	81
1990–1994	45	29	17	8	11	115
1995–1999	64	56	4	11	34	229
Old adult						
1968–1979	3	6	8	3	6	35
1980–1984	5	9	1	2	5	39
1985–1989	13	8	5	3	3	23
1990–1994	24	5	7	4	2	27
1995–1999	25	12	0	4	9	58

† Causes of death include protozoans, bacterial infection, acanthocephalan peritonitis, coccidioidesimmitis, emaciation, and other.
‡ Shark bites are classified as ''certain'' or ''probable.''
§ Causes of death include dependent pups and immatures with trauma, lacerations, and females with mating wounds.
‖ Causes of death include entanglement in fishing gear, oil spills, gunshot, and research fatalities.
¶ Causes of death include dead pups, trauma of uncertain origin, and miscellaneous trauma.
# Causes of death include uncertain, with no trauma apparent, dependent pups and immatures, and unknown.

into five broad categories (Table 1), plus an unknown category that included carcasses for which no cause could be determined (usually due to the carcass being in an advanced stage of decomposition). We restricted our analyses to the last 19 years of data (1981–1999), excluding the earlier years because of the small number of records with a known cause of death. In total, our data set included information from 2242 carcasses, 60% of which (1357) died for unknown reasons. Data were pooled for both sexes.

Formulating the demographic model

The demographic model consisted of 500 age-based Leslie matrices representing different parameter values for key vital rates. Key vital rates used in these model (see Siniff and Ralls 1991) included age-specific fecundity, weaning success, and survival by age: juvenile (post weaning, 6–24 months), subadult (age 2–3 years), adult (age 4–12 years), and old adult (age 13–20 years). The first row of our population matrices consists of age-specific reproduction, defined as the age-specific probability of producing a 1-year-old female offspring. This was calculated as the product of fecundity, weaning success rate, and the square root of the annual juvenile survival rate (i.e., to account for survival from 6 months to 1 year); throughout the paper, we refer to these products as reproductive rates. We assumed that 20 years was the maximum attainable life span of otters in this population. Although all of our models are age-based, we report results in terms of the stage-specific survival and reproductive parameters for juvenile, subadult, adult, and old-adult otters that are used to formulate the age-specific elements of the population matrices.

To look for general patterns in the importance of different demographic rates and age groups, we first formulated 500 Leslie matrices, setting vital rates for each matrix as uniform random variables selected to represent the range of plausible values for these vital rates as determined by a review of the relevant literature (Table 2). To account for uncertainty in the ''true'' value of specific vital rates and our lack of information about their variance, we set the upper and lower bounds for each rate to encompass the full range of reported

values, and then increased this range by 10% above and below reported values. For each replicate matrix, we calculated the discrete annual rate of population increase (λ) and the associated reproductive value ($\mathbf{R}_x$) vector. Reproductive values represent the extent to which individuals of a particular age contribute to the ancestry of future generations (Caswell 2001).

An analytical elasticity analysis was performed to predict the effects of proportional changes in each of the stage-specific reproductive or survival rates on λ. Following Caswell (2001), elasticities were calculated for each age-specific matrix transition element and a single elasticity value for each age was calculated as the sum of the age-specific elasticities for all ages comprising that age class. For all of these statistics, we examined the distribution of results over the 500 replicate matrices.

Using mortality data in life-stage simulation analysis

We sought to quantify the effect of observed variation in individual mortality sources on population growth. In doing so, we took an approach that accounts for both the mean and the temporal variance in the importance of each mortality factor, using the observed year-to-year variation in relative numbers of carcasses as a representation of the variation in actual mortality rates. First, we characterized the mean and variance in the number of deaths observed each year, for each of the i age classes and for each of the j mortality sources. Accordingly, we separately tabulated the number of carcasses for each of the four stage classes i ($i = 1, 2. . . I$) dying from each of the seven primary causes j ($j = 1, 2. . . J$), and for each category we estimated the mean and variance in number of deaths over all years of data.

We calculated 500 sets of demographic schedules by varying the expected number of deaths at each stage. Variance estimates were based on year-to-year variations in the number of observed carcasses collected for each stage i and mortality source j, with the assumption that the necropsy data represented the true population. This treatment of the data assumes that although there is not an equal probability of observing deaths from each source of mortality, the year-to-year variance in mortality represents the actual variance in nature. In addition, the use of the observed death variance assumes that there have not been dramatic changes in population sizes during the sampling period, that mortality is not compensatory, and that there is not significant covariance between age-specific sources of mortality.

Using a single projection matrix, vital rates for each stage were varied to solve for expected stage-specific mortalities that were consistent with the range of observed stage-specific mortalities (carcasses). These runs were used to derive the mean and variance of the expected distribution of stage-specific vital rates. To create each set of simulated rates, we assumed that the distribution for each stage- and cause-specific death rate could be adequately described using negative binomial distributions, and drew a random number from each death distribution according to its estimated mean and variance. We then used these numbers of simulated dead otters to estimate total survival rates for each age class. For each set of rates, the annual age-specific survival rate for age i otters, s_i, was calculated as

$$s_i = 1 - (1 - \bar{s}_i)\frac{\sum m_{ij}}{\sum \bar{m}_{ij}} \quad (1)$$

where $\bar{s}_i$ is the mean survival rate (set to our ''intermediate'' survival values in Table 2), m_{ij} is the simulated number of dead otters of age i and cause j for a particular replicate, and $\bar{m}_{ij}$ is the mean of each dead otter distribution. Both summations in Eq. 1 are over the j mortality causes. The formulation results in a mean mortality rate for each age class that is equal to the $\bar{s}_i$ values used. We removed simulation results that yielded age-specific s_i values outside the plausible range (Table 2) and recorded 500 replicate simulation results. Less than 1% of our simulations fell out of this plausible range for s_i, and these values occurred at the extreme tails of a normal distribution for s_i. Finally, we accounted for variation in age-specific fecundity rates by selecting age-specific fecundity rates as uniform random variables from within the range of possible values (Table 2).

Having generated 500 sets of demographic schedules, we constructed a population matrix from each and calculated the associated population growth rates (λ). We estimated the contribution of each source of mortality to variation in λ using the following multiple linear regression model:

$$\lambda = \alpha + \beta_1(m_{1,1}) + \beta_2(m_{1,2}) + \beta_2(m_{1,3}) \cdots + \beta_n(m_{IJ}). \quad (2)$$

Using this model, we calculated the partial coefficients of determination ($r^2_{y1\cdot 2\ldots n}$) for each of the n age-specific mortality causes (Neter et al. 1996), which were then summed across age classes for each source of mortality. In our multiple regression model, the partial coefficient of determination represents the relative amount of variance in our response variable that is explained by each independent variable, after controlling for variance due to all other independent variables (Neter et al. 1996). In particular, this metric provides an indication of the sensitivity of λ to variation in a given source of mortality. For all summary statistics, we report the mean values with one standard deviation.

Putting life-stage simulation analysis into practice: projecting effects of conservation strategies

To demonstrate the potential relevance of this approach for prioritizing conservation actions, we next used our model to examine the sensitivity of λ to specific increases or decreases in each source of mortality.

TABLE 2. Summary of published estimates for southern sea otter vital rates, and the range of values used for the current analyses.

Source, by sex and age	Sexual maturity age (yr)	Annual birth rate	Pup survival
Siniff and Ralls (1991)			
Female	3	0.90–0.94	0.46–0.58
Male	6		
Riedman et al. (1994)			
Mother age 3–6 yr	3	0.90	0.40
Mother age 7–10 yr	3		0.75
Mother age 11–14 yr	3	1.07‖	1.00
Eberhardt and Schneider (1994)			
Female	4–5	0.890–0.898	
Jameson and Johnson (1993)			
Female	3	0.87–0.90	
This study, minimum values			
Mother age 3 yr	3	0.60	
Mother age 4–6 yr	3	0.90	0.40¶
Mother age 7–10 yr	3	1.05‖	0.70
Mother age 11–20 yr	3		0.80
This study, maximum values			
Mother age 3 yr	3	0.60	
Mother age 4–6 yr	3	0.90	0.70¶
Mother age 7–10 yr	3	1.05‖	0.95
Mother age 11–20 yr	3		1.00
This study, intermediate values			
Mother age 3 yr	3	0.60	
Mother age 4–6 yr	3	0.90	0.55¶
Mother age 7–10 yr	3	1.05‖	0.83
Mother age 11–20 yr	3		0.90

Note: Pup survival is measured as weaning success; age categories are: juvenile 0–1 yr; subabult 2–3 yr; adult 4–12 yr; old adult 13–20 yr.

† Data for juveniles and subadults pooled. The low value in the range is presented as mean ± 1 SD.

‡ Data for adults and old adults pooled.

§ Lambda (λ) for females and males pooled (rate of population increase).

‖ Pooled for mother age 7–20 yr.

¶ Pooled for mother age 3–6 yr.

Using our 500 sets of demographic rates as a starting point, we varied the number of deaths from each mortality source from 0 to 200% of values used in the original sets of simulated values, while holding other causes of mortality constant at observed rates. For each modified set of demographic rates, we constructed a new population matrix and calculated the predicted population growth rate.

Assessing sensitivity of model to unrecovered carcasses

All analyses just described are based on data obtained from beach-cast carcasses. If a particular mortality source affects the probability of a carcass reaching the beach, then the pattern of mortality in unrecovered carcasses may differ from that in recovered carcasses. To assess the degree to which our results regarding the relative importance of each mortality source might be susceptible to such a bias, we evaluated the sensitivity of λ to unrecovered carcasses, relative to the sensitivity of λ to known sources of mortality. Ideally, such an analysis would use direct estimates of the number of unrecovered (and hence, unseen) carcasses. Due to the impossibility of measuring the number of unrecovered carcasses, our approach instead was to fit a mortality distribution model to the 1982–1999 population data.

To implement this approach, we drew on several approaches for estimating sea otter demographic rates. First, we wanted to account for the change in demographic parameters associated with the period of population increase (5% annual increase between 1984 and 1994) and population decline (3% annual decline between 1995 and 1999) (see Fig. 1). To do so, we used the method of Tinker et al. (2000), which provides an updated version of the hazard model used by Siniff and Ralls (1991). The Tinker et al. (2000) approach uses a maximum likelihood technique, in conjunction with a time series of carcass age structure data, to fit modifying functions (following methods described in Doak and Morris [1999], Monson et al. [2000]) that adjust age-specific survival rates to reflect changes over the

TABLE 2. Extended.

Juvenile survival	Subadult survival	Adult survival	Old adult survival	λ
0.75 ± 0.145 to 0.80†		0.89 ± 0.088 to 0.91‡		1.05§
0.85 ± 0.179 to 0.88†		0.52 ± 0.167 to 0.61‡		
0.50	0.70	0.84	0.80	0.92
0.90	0.95	0.98	0.98	1.17
0.70	0.83	0.91	0.89	1.05

period of population decline. We initialized the simulation by multiplying the estimated population size in 1982 (as measured by the 1982 range-wide count plus 5%, to account for animals missed by observers; Estes and Jameson 1988) by the stationary age distribution associated with the hazard matrix model. The simulation was then run for each year between 1982 and 1999, using the hazard model to project population growth (and thus carcass production) for the 1982–1993 period and the Tinker et al. model for the 1994–1999 period. The resulting age-specific distributions of expected carcasses were compared to the distributions of carcasses actually obtained. The difference between expected and observed carcasses was used to estimate the annual number of unrecovered carcasses between 1982 and 1999. Finally, we repeated the life-stage simulation analysis previously described, with ''unrecovered'' included as a separate mortality source. We compared the sensitivity of λ to unrecovered carcasses, relative to the sensitivity of λ to recovered carcasses with known or unknown cause of death, using one-way ANOVA.

RESULTS

Use of the intermediate values of reported demographic rates for sea otters (Table 2) to parameterize a Leslie matrix resulted in an expected rate of annual growth of ~5% per year (λ = 1.05). Selecting all rates as random variables between the lower and upper allowable values resulted in a distribution of growth rates between λ = 0.94 and λ = 1.15 (95% confidence to the mean = 1.00, 1.09; Fig. 2). These rates of growth correspond fairly well to minimum and maximum values reported in the literature for southern sea otters (Riedman et al. 1994). The vector of reproductive values was relatively consistent for all matrix replications (Fig. 3), with maximum values occurring for adult females 6–8 years old and then declining rapidly for older animals. The elasticities obtained for southern sea otters indicate that the population is far more sensitive

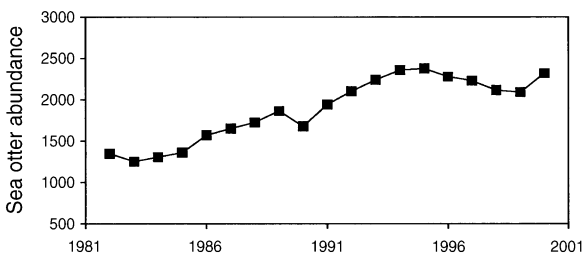

FIG. 1. Abundance estimates of sea otters from 1982 to 2000. These data represent counts made during annual spring surveys; additional inconsistent surveys were made before 1982.

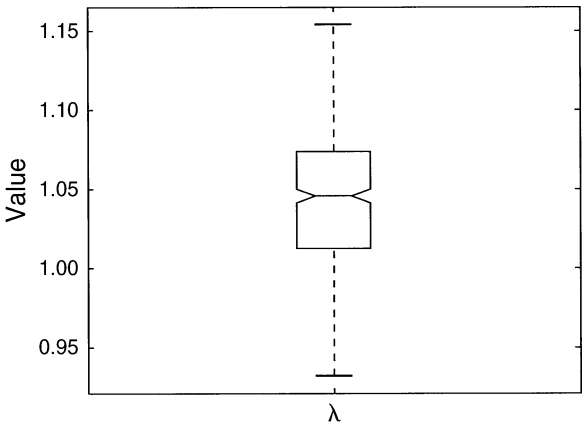

FIG. 2. Box and whisker plot showing the distribution of annual rates of population growth (λ) for replicate Leslie matrices. For each replicate matrix, vital rates were selected as uniform random variables from within the range of possible values (Table 1). The upper and lower boundaries of the box indicate the upper and lower quartiles; the middle line indicates the median value (the notch delineates the 95% confidence interval for the median); and the whiskers span the range of all observed values (median = 1.042, lower and upper quartiles = 1.012–1.076, range = 0.9235–1.165, mean = 1.045, 1 SD = 0.043, 95% CL = 1.001, 1.089).

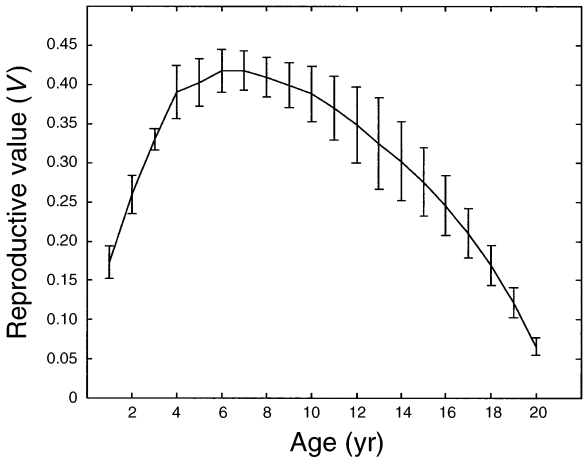

FIG. 3. Reproductive value, V (mean ± 1 SD), for replicate Leslie matrices. For each replicate matrix, vital rates were selected as uniform random variables from within the range of possible values.

(sensu Caswell 2001) to changes in survival than in growth or reproduction, and more sensitive to adult survival than to subadult or juvenile survival (Fig. 4).

Our multiple regression analysis of the sensitivity of λ to each mortality source shows that most of the variation in the values of λ from our simulated matrixes is explained by unknown causes (61.6 ± 4.89%). Considering only known causes, disease and emaciation had the greatest impact on λ, accounting for ~21% of the variance (Fig. 5). Although our elasticity analysis shows that changes in adult survival most strongly influence λ, the death assemblage data indicate that the cause of mortality with the greatest potential effect on λ (disease) occurs in juveniles. Reducing mortalities due to disease to 50% of their current mean values would be sufficient to increase the annual rate of population growth by ~1.5% (Fig. 6). In contrast, eliminating all mortality due to gun shots would have a negligible effect on population viability. Surprisingly, shark bites and trauma together explained only 14% of the variation in λ.

Finally, we used matrix model projections to analyze the potential biases associated with unrecovered carcasses by calculating expected population growth over the period 1982–1999. The simulated population trajectory closely approximated observed population growth (Fig. 7) and indicated that, on average, only 46 ± 9.3% of mortalities are retrieved as beach-cast carcasses, although this percentage varies considerably from year to year (Fig. 7). This unrecovered category may account for a substantial percentage of the fluctuations in λ (~35% of total variation). Indeed, our sensitivity analysis suggests that λ was more sensitive to these unrecovered carcasses, considered as a single group, than to recovered carcasses with either known or unknown causes of death ($F = 34.12$, df = 2, 72, $P < 0.001$).

DISCUSSION

Our analyses provide a novel approach to using death assemblages to evaluate hypotheses about population declines and to suggest recovery strategies. In particular, focusing management efforts on minimizing the transmission of newly emerging diseases could have a relatively large impact on reversing the population decline. To the extent that several of the diseases currently

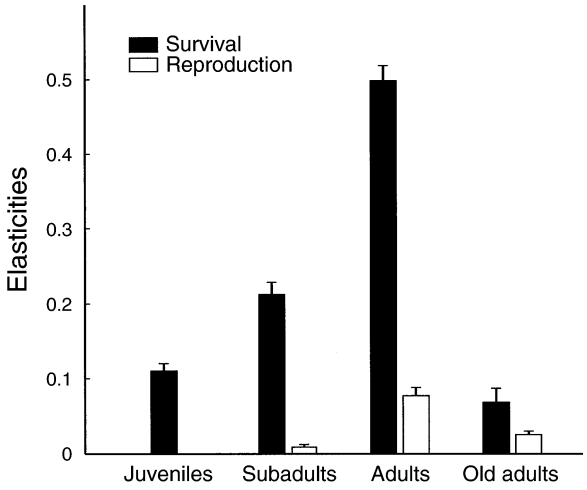

FIG. 4. Elasticities of λ (mean + 1 SD) to changes in age-specific survival and reproduction values for all entries into the population projection matrix. Mean values were calculated for replicate matrices in which vital rates were selected as uniform random variables from within the range of possible values.

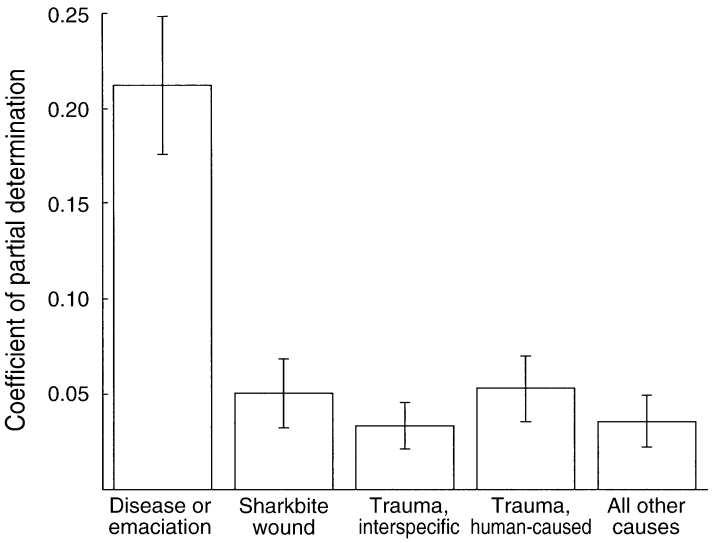

FIG. 5. Sensitivity of λ to each source of mortality, as measured by the percentage variance in λ explained by the focal mortality agent after accounting for variance from all other sources (estimated as $r^2_{y1\cdot 2\ldots n}$).

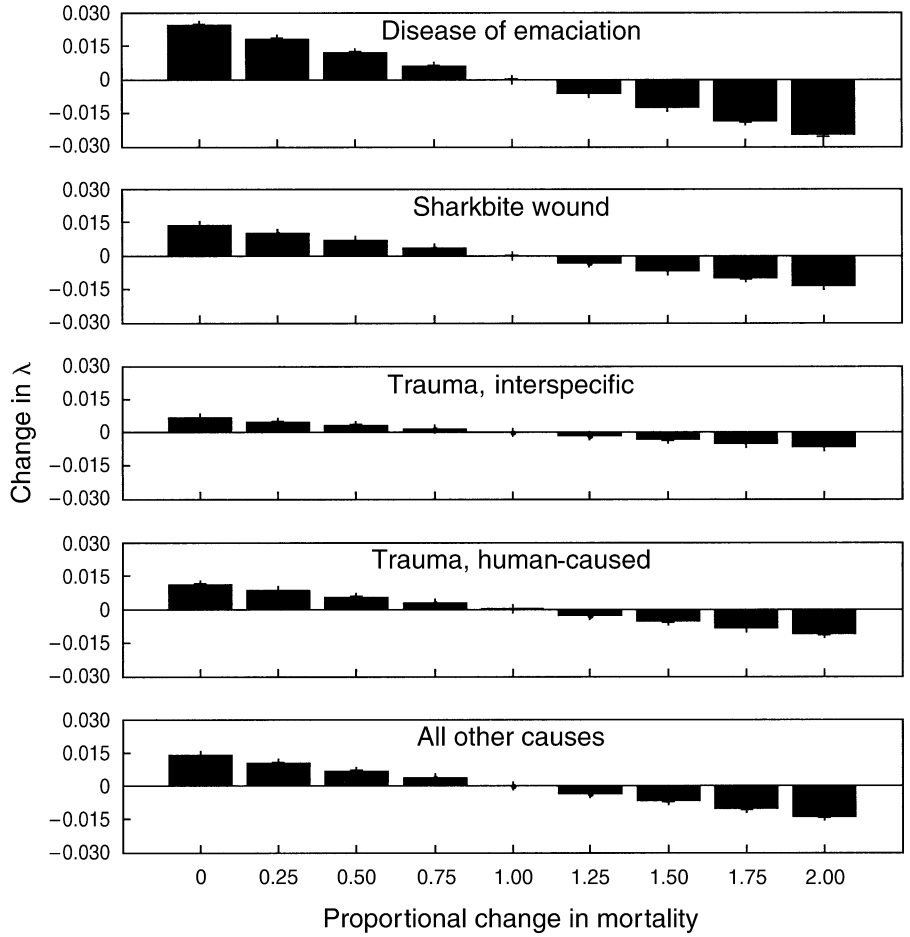

FIG. 6. Effect on λ of systematically varying the level of mortality (0–200% baseline level) for one focal cause, while allowing mortality from all other causes to vary randomly.

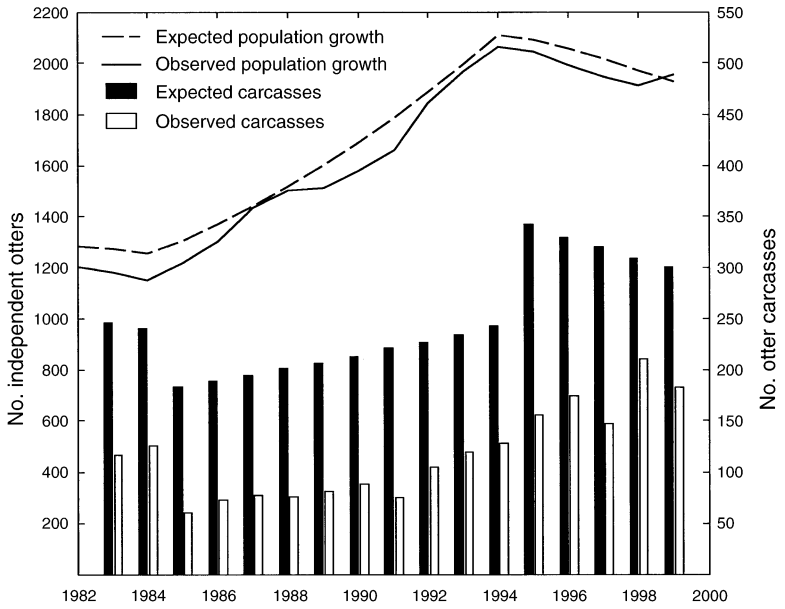

FIG. 7. Expected vs. observed population growth trajectories for southern sea otters between 1982 and 1999 and expected vs. observed carcass production.

killing sea otters appear to have terrestrial and anthropogenic origins (Lafferty and Gerber 2002), enforcement of existing laws and regulations may be effective in reducing their impacts on sea otter. For example, State Fish and Game and Water Quality Control Board codes make it illegal to place or allow to be placed into marine waters any substance or material that is harmful to fish and wildlife, or that impairs beneficial uses of State waters.

Just as importantly, our findings emphasize the uncertainties in linking any source of mortality to effects on population growth. Even for this extremely well-studied population, we can only roughly link variation in changing mortality causes with effect on population dynamics, due to uncertainty in both demographic and cause-of-death data. Although this poor predictive certainty may seem disappointing, by clarifying the degree of certainty and the assumptions behind any management inferences, our approach can yield more careful and well-thought-out decision making and use of research resources.

Are insights from these models obvious?

What does this approach tell us that may not have been qualitatively obvious from a simple examination of the necropsy and stranding data? Our modeling results for sea otters are consistent with observed mortality patterns: the largest proportion of known deaths was due to disease and starvation; of these, disease may be more manageable. In particular, our elasticity analysis shows that changes in adult survival most strongly influence λ, yet the death assemblage data indicate that disease, the primary cause of mortality with the greatest potential effect on λ, occurs mainly in juveniles. These results agree with those of Lafferty and Gerber (2002) and Miller et al. (2002). However, they conflict with the findings of Estes et al. (2003), who report no differences in the overall proportion of deaths from infectious disease in the salvaged carcasses between periods of population growth and decline. Thus, the results from our detailed modeling are not surprising, but are necessary to understand how the interplay between elasticity patterns and total number of deaths in particular age categories result in variable levels of importance for different mortality causes.

Sources of uncertainty

Although our analyses help to elucidate the importance of mortality and pathology data in understanding population trends, they also help to highlight and clarify uncertainty about possible management actions. One source of uncertainty comes from the uncertainty inherent in the demographic estimates. Another arises from the high proportion of carcasses for which cause of death is unknown. A third and far more significant source of uncertainty stems from the unrecovered carcasses. In the best of all worlds, the carcass record is perfectly representative of actual mortality patterns. How far astray might this assumption be, and what might be done to account for this uncertainty in further studies? First, it should be noted that most variation in λ is explained by the ''unknown'' category of mortality. This category includes both highly decomposed animals (not necropsied) and animals that receive necropsies but for which the cause of death cannot be determined. Our analysis relies on the assumption that ''unknown'' mortalities could be represented based on the distribution for known causes of mortality. If the

''unknown'' category is not represented in documented causes of mortality, the overall pattern that we describe (for example, the importance of disease and shark bites for λ) would be weakened. Second, our sensitivity analysis suggests that variation in the unrecovered carcass class may account for more variation in λ than all of the known causes combined. Again, this is not problematic if the unrecovered carcasses truly represent a random subset of all dead animals. However, additional information on potential biases in mortality associated with unrecovered carcasses is needed before strong conclusions can be made based on the results presented in this paper. For example, there is some suggestion that animals that drown in fishing nets may not be recovered because they sink rather than float to the surface (J. A. Ames, California Department of Fish and Game, *personal communication*). The only reliable way to measure or account for such biases is to collect longitudinal data on survival and mortality from marked individuals. Thus, although analysis of carcasses may be useful in understanding mortality patterns, continued research on the living population is critical.

Pathology data are themselves imprecise. Our analyses rely primarily on pathology data derived from gross examination of carcasses and assignment of a presumptive cause of death. A data set is now being developed with ~500 fresh-dead sea otters examined by veterinary pathologists. This death assemblage has the potential to provide more precise disease-specific and trauma-type information. Additional modeling work should be conducted as these data become available. For example, with adequate data, one could examine the effect of changes in state structure of individuals. Because immune state could be affected by inbreeding depression or pollution, this approach could make a large difference in the final management assessment. Even so, distinguishing the proximate vs. ultimate causes of mortality in wild populations will always be difficult. By assuming that there is no compensatory mortality in our modeling, we have assumed that all primary pathologies were the true ultimate causes of death. One goal for future work on this and other species is a careful analysis of patterns of variation and covariation in death causes to make better inferences about the primary drivers of death vs. more opportunistic causes of death. Although some such patterns are well known in ecology (e.g., muskrats, Allee et al. [1949]; fish, He and Kitchell [1990], He et al. [1993]; harbor seals, Ross et al. [1996]; Steller sea lions, NMFS [1994]), these examples have not been considered in the context of the analysis presented in the current study.

Implications for conservation and management

Perhaps the greatest benefit to be gained by understanding the role of mortality sources for southern otters is that it will help to focus attention on the sources of mortality that can and cannot be controlled in recovery efforts. In light of our results and the real-world potential for implementing putative management options, where should conservation efforts be focused? Such decisions should derive from contributions to λ, on the one hand, and the practicality of implementation, on the other. For instance, eliminating disease-related mortality might provide significant improvements in λ, but also could require the expenditure of huge amounts of capital to control terrestrial sources of pathogens and pollutants. Conversely, reducing gunshot mortalities might be relatively simple to address, but would have a much less significant impact on λ. The apparent role of shark predation in limiting the growth of sea otters raises the question of whether it is practical to cull sharks, which are increasingly the focus of conservation attention themselves, in order to conserve sea otters.

A second important benefit to the simulation approach that we present is the quantification of uncertainty in the data, and of the potential impacts of this uncertainty on implied trends. Although highlighting uncertainty may, at first blush, appear to be a drawback, it is in fact a much needed and often overlooked component of wildlife management, and may suggest fruitful areas for further investigation. For example, incidental losses to entanglement in fishing gear might be relatively straightforward to address (e.g., through fishery regulations), but the contribution to λ from this source remains uncertain due to potential biases in carcass recovery and diagnostic problems in the carcass record (i.e., drowning is difficult to diagnose). If all of the unrecovered carcasses represented mortalities due to drowning, the impact of entanglement in fisheries on λ could exceed that of disease and starvation (Fig. 7). Thus the quantification of uncertainty in this case provides a caution against prematurely discounting specific mortality sources.

In light of the combination of uncertainty and practical constraints, what should be done to improve recovery prospects for southern sea otters? First, if mortality due to emaciation is a result of a reduction in carrying capacity, little action could be taken to increase the population's growth rate if it is indeed approaching carrying capacity. However, in light of the uncertainty just described, action can be taken to specifically buffer sea otters from some diseases, although other independent events may alter the disease landscape. Some of the diseases that cause the most mortality in otters are accidental and, seemingly, newly emerged (Thomas and Cole 1996, Lafferty and Gerber 2002, Miller et al. 2002). In some cases, humans may have influenced the spread and emergence of these new diseases. Improved control of sewage disposal into coastal areas may decrease the prevalence of protozoal and bacterial infections and, perhaps, reduce bioaccumulation of contaminants (which could improve otter immune defenses). Fungal spores that cause coccidiomycosis (valley fever) may be reduced by changes

in agricultural and construction practices. These results may be used as a decision framework to weigh the costs and benefits associated with particular management actions. For example, it would be possible to reverse the declining trend of sea otters by reducing shark predation by 75%, by reducing all human-caused trauma by 75%, or by reducing both shark predation and human-caused trauma by 40% (Fig. 6).

Application to other populations

The method that we present in this paper could be fruitfully applied to other case studies for which mortality and pathology data are available. Southern sea otters represent an interesting example for developing this approach; however, the same approach would be much more difficult to implement in other parts of the sea otter's range (e.g., Aleutian Islands) because few carcasses are recovered. Compared to terrestrial mammals, one of the unusual problems with sea otters is that carcasses may be recovered in different areas than where the animals lived and died. This creates the potential for differential recovery by cause of death, unless all deaths have the same probability of transfer from sea to land. The same problem holds for virtually all marine birds and mammals. Thus, the approach described in this paper would work best for cases in which death assemblage data truly represent a snapshot of causes of death (i.e., recoverability is not biased for age classes or cause). A review of the published literature suggests several species that would be amenable to this approach (e.g., manatees, Bonde [2000]; bottlenose dolphins, Ewing et al. [2002], Eguchi [2002]; asteroids, Thorpe and Spencer [2000]; gastropods and bivalves, Inoue et al. [2002], Warwick and Light [2002], Gonzalez et al. [2001]; beetles, Connor [1988]; pronghorn, Lubinski and O'Brien [2001]). In addition, there are other candidates that could be examined from the fossil record (e.g., clams, Green et al. [1984]; tetrapods, Rogers et al. [2001]; ungulates, Berger et al. [2001]), which could shed light on demographic correlates of observed extinction patterns.

Although our sea otter case study focused on sensitivity to particular mortality sources, our approach could be expanded to encompass other factors such as spatial population structure. Evaluations of the efficacy of management actions for endangered species will benefit by using a quantitative framework that explicitly links mortality data to factors driving extinction risk.

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