



Harbor seal pup dispersal and individual morphology, hematology, and contaminant factors affecting survival

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ABSTRACT

Understanding the factors affecting individual harbor seal (*Phoca vitulina*) survival is essential for determining population level health risks. We estimated postweaning dispersal, and modeled the effects of morphology, hematology, and blubber contaminants on the survival of recently weaned harbor seal pups using a mark recapture framework. We deployed satellite transmitters on apparently healthy pups captured in San Francisco Bay (SFB, $n = 19$) and Tomales Bay (TB, $n = 7$), and pups released after rehabilitation that stranded along the central California coast preweaning ($n = 21$). Dispersal distances were further than previously reported for harbor seal pups (maximum = 802 km) which has implications for understanding risks to this vulnerable age class. We found differences in body condition, serum immunoglobulin and thyroxine (T₄) concentrations, white blood cell count, and blubber organohalogen contamination (OH) among the three groups. Overall, increased T₄, decreased OH, and increased mass were associated with greater survival probabilities; whereas, among stranded seals, greater mass gain, shorter time in rehabilitation, and admission to rehabilitation earlier in the season were associated with greater survival probabilities. Attention to these latter factors may improve the success of rehabilitation efforts. For wild pups, reduction of legacy contaminants and direct causes of mortality, such as ship strike, may enhance pup survival.

Key words: harbor seal, *Phoca vitulina*, contaminants, survival, dispersal, stranding, telemetry, postweaning, juvenile, health.

Understanding population health requires data about the impacts of risk factors on vital rates such as survival. Juvenile survival is of

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particular interest because of its variability and its influence on future productivity (Eberhardt and Siniff 1977). In pinnipeds, juvenile survival has been linked to genetics, protein reserves, climate perturbations such as El Nino, thermal stress, sex, immunity, contaminant concentrations, and size or mass at weaning (Ono *et al.* 1987; Coltman *et al.* 1998; Craig and Ragen 1999; Hall *et al.* 2001, 2002, 2009; Harding *et al.* 2005; Bennett *et al.* 2007; Baker 2008). Mass at weaning has in turn been linked to maternal mass, maternal experience, and prey availability during lactation (Lunn *et al.* 1994, Bowen *et al.* 2001, McMahon and Burton 2005). Some factors, such as mass at weaning, may vary annually and be affected by changes in prey availability or climate (McMahon and Burton 2005). While the studies of juvenile survival referenced above investigated the effect of environmental covariates on maternal and pup body condition and physiology, fewer studies have assessed the effects of contaminants and disease.

The role of organohalogen contaminants in marine mammal survival is increasingly being investigated: earlier studies focused on the effects of contaminants on immunity and reproduction and only indirectly on survival, but recently direct associations with survival probability have been considered. Experimental exposure studies have demonstrated effects of contaminants on immunity and reproduction in captive harbor seals (*Phoca vitulina*; Reijnders 1980, Ross *et al.* 1995, de Swart *et al.* 1996); PCBs, PBDEs, and DDE were associated with an increase in leukocyte count in wild harbor seals (Neale *et al.* 2005); and effects of contaminants on the function of harbor seal immune cells have been demonstrated *in vitro* (Neale *et al.* 2002, Levin *et al.* 2005, Hammond *et al.* 2005). Blubber concentrations of PBDEs in weaned gray seal (*Halichoerus grypus*) pups were associated with first year survival probability (Hall *et al.* 2009).

Organohalogen contaminants can also act as endocrine disruptors and affect growth and metabolism through alterations to circulating thyroid hormones (Hall *et al.* 1998). In gray seals, blood PBDE concentrations were positively associated with total serum levels of thyroxine (T₄) and triiodothyronine (T₃), when time since weaning and body condition were taken into account (Hall *et al.* 2003). It has also been suggested that contaminant exposure increases the likelihood of disease and mortality in sea lions and cetaceans (De Guise *et al.* 1995, Jepson *et al.* 2005, Ylitalo *et al.* 2005).

Harbor seals are good candidates for understanding regional impacts of contaminant exposure on marine mammals, because their tissue contaminant concentrations vary with location reflecting spatially limited foraging areas (Ross *et al.* 2004, Greig *et al.* 2011). Harbor seals are distributed coastally throughout the Northern Hemisphere and although they are relatively abundant throughout their range, local declines have been reported in Alaska (Small *et al.* 2008, Womble *et al.* 2010) and Scotland (Lonergan *et al.* 2013). Robust estimates of local survival, and knowledge of local factors influencing survival, therefore are important for understanding regional risks to population stability and growth.

Harbor seal survival rates were historically estimated from mortality rates and life tables with annual adult survival estimated at approximately 80% (Bigg 1969, Boulva and McLaren 1979). Studies of a Scottish

population, using natural markings and mark recapture models, estimated adult harbor seal survival rates as between 92% and 98% (Mackey *et al.* 2008, Cordes and Thompson 2014). In central California, a recent radio telemetry study estimated adult female survival at 90% (Manugian *et al.* 2017). Juvenile survival is more variable with estimates of pre-weaning survival ranging from 69% to 83% (Boulva and McLaren 1979, Steiger *et al.* 1989) and postweaning first year survival ranging from 35% to 80% (Bigg 1969, Reijnders 1978). One difficulty in estimating postweaning survival in harbor seals is that they disperse from their natal beaches soon after weaning, and it is not known where they go or if and when they return to these same beaches. Radio tags or other marks that require searching a large geographic area to relocate the animal, therefore, are of limited use for this age class (Lander *et al.* 2002). Satellite telemetry is better suited to tracking animals whose destination is not known because the location data can be acquired remotely.

After passage of the Marine Mammal Protection Act in 1972 in the United States, harbor seal numbers along the California coast increased rapidly before stabilizing in the 1990s (Sydeman and Allen 1999, Carretta *et al.* 2007). Despite coastal increases, harbor seal numbers within San Francisco Bay (SFB) have remained relatively stable (Grigg *et al.* 2004, Manugian *et al.* 2017). Factors hypothesized to limit harbor seal numbers in SFB have included oceanographic features and changes in prey availability, increased predation, limited haul-out space (Sydeman and Allen 1999), and anthropogenic pollutants (Kopec and Harvey 1995, Neale *et al.* 2005, Brookens *et al.* 2007). To date, the factors controlling population numbers within SFB remain poorly understood and the variables affecting juvenile survival are not known.

To explore the relative importance of contaminant exposure, compared with other variables, in affecting juvenile survival of harbor seals in SFB, we used satellite telemetry to monitor survival of weaned seal pups from SFB, weaned seal pups from Tomales Bay (TB), and seals released from The Marine Mammal Center (TMMC), a hospital for the rehabilitation of stranded marine mammals. San Francisco Bay is a large estuary and port that has been dramatically modified by human occupation and activity (Conomos *et al.* 1985) whereas TB, located to the north and bordered by Point Reyes National Seashore, is a semienclosed, relatively undeveloped, seasonal estuary (Kimbrow *et al.* 2009). Each year, stranded newborn harbor seals from San Luis Obispo to Mendocino counties (an area including SFB and TB) are admitted to TMMC for rehabilitation and release. Seal pups from TB and TMMC were chosen because they had different blubber contaminant levels from SFB seals: pups from SFB and TB had different contaminant profiles, whereas pups from TMMC had lesser blubber contaminant concentrations at the time of release from rehabilitation because they ingested less, if any, contaminated maternal seal milk (Greig *et al.* 2011). Factors unique to the rehabilitation setting (for example, time spent in rehabilitation) were evaluated to determine if any variables affecting survival beyond contaminant exposure could guide rehabilitation practices. In addition to the factors affecting survival, the use of satellite telemetry allowed us to record postweaning harbor seal pup dispersal.

METHODS

Satellite tags (Spot5, Wildlife Computers, Redmond, WA) were deployed on three groups of seals born in 2008: recently weaned, wild-caught pups from SFB (37.93°N, 122.42°W) and TB (38.22°N, 122.96°W), and rehabilitated pups from TMMC once they achieved weaning mass. Rehabilitated seals were released at three locations within the TMMC response area depending on logistics: Chimney Rock (37.99°N, 122.96°W), Fitzgerald Marine Reserve (37.52°N, 122.51°W), and Cypress Point (36.58°N, 121.97°W).

Animals were captured and handled as described in Greig *et al.* (2014). At the time of tag deployment, pups were weighed, length and girth were recorded, and blubber depth was measured by ultrasound. Blood was drawn to measure leukocyte count (WBC), total immunoglobulin G concentrations (IgG), total thyroxine (T₄), and total triiodothyronine (T₃) as described in Greig *et al.* (2010), and a blubber sample was collected for contaminant analysis. Blubber was collected using a sterile 8 mm dermal biopsy punch (Miltex, Inc., York, PA), wrapped in solvent rinsed teflon, and stored at -80°C until analysis. A satellite-linked transmitter was glued to the pelage on the top of each individual's head using loctite 422 (Loctite Corp., Hartford, CT).

Satellite tags weighed 49 g, measured 48 × 42 × 14 mm, and had temperature and wet/dry sensors. Each tag contained a battery capable of transmitting data 30,000 times: the tags were programmed to transmit a maximum of 100 transmissions on alternate days to extend the battery life for 10 mo at which time seals would be expected to molt and tags would be shed. The tags transmitted location, temperature, and percentage time the tag was dry (as a proxy for time spent ashore) to the Argos system of orbiting satellites. The system used satellite locations and changes in the frequency recorded for each message, caused by the motion of the satellite relative to the tag (Doppler effect), to calculate the location of each tagged seal (CLS 2008). Argos assigned a location class (LC) to each location estimate and provided error estimates; LC 3 (<250 m), LC 2 (250–500 m), LC 1 (500–1,500 m), and LC 0 (>1,500 m) with no estimates of accuracy available for LC A or B (CLS 2008).

Sample Analysis

Total WBCs/μL were determined for blood samples collected as described in Greig *et al.* (2010). Blubber samples were analyzed for organohalogen pollutants (OH) using the methods described in Greig *et al.* (2011), and contaminant concentrations in ng/g lipid weight, summed by major contaminant classes, were used as individual covariates in the survival model. The contaminant classes were polychlorinated biphenyls (PCB), dichlorodiphenyltrichloroethane and its metabolites (DDT), polybrominated diphenylethers (PBDE), chlordanes (CHLD), and hexachlorocyclohexanes (HCH). All five contaminant classes were summed for a sixth covariate of overall organohalogen contamination (OH).

A protein A enzyme linked immunosorbent assay (ELISA) was used to quantify total IgG after Ross *et al.* (1993). Concentration in mg/mL was calculated from absorbance using a standard curve generated for each ELISA plate using dog reference serum with a known amount of IgG (Bethyl Laboratories, Montgomery, TX). Intraassay coefficient of variation (CV) was <6.0%, interassay CV was 7.9%.

Blood concentrations in nmol/L of total thyroxine (T4) and total triiodothyronine (T3) were determined by enzyme-linked immunosorbent assay (ELISA, Fortress Diagnostics, Belfast, U.K.). All samples were assayed in duplicate with controls and standards included in all assay runs. Total T4 intraassay coefficient of variation (CV) was 10.4%. Total T3 intraassay CV was 7.6% (Hall and Thomas 2007).

Data Analysis

Morphology covariates and blood variables (WBC, IgG, T3, and T4) were analyzed by group using analysis of variance (ANOVA) and Tukey's honest significant difference method. The residuals from a linear regression of mass (kg) on length (cm), $\text{mass} = -21.90 + 0.47 \times \text{length}$ ($F = 47.86$, $df = 45$, $R^2 = 0.52$, $P < 0.005$), were used as an index of body condition. Because contaminant concentrations have been associated with health parameters in other studies (Hall *et al.* 2003, Neale *et al.* 2005), linear regression and generalized linear models were used to evaluate whether similar associations were present in this study and might confound the survival analyses. Specifically, we tested whether contaminant concentrations were predictive of blood thyroid hormone levels as in gray seals (Hall *et al.* 2003). We also tested for associations among the blood and morphology variables and OH. Contaminant concentrations were log transformed to achieve normality. Statistical analyses were performed using the R programming language (R Development Core Team 2009).

The satellite tags could only communicate successfully with the satellites when the tag antenna was out of the water, and the tag did not float if detached from the animal. If an animal was dead and floating with its tag still attached, the antenna would be underwater and unable to transmit because the head is not the buoyant part of the carcass. Thus, when transmissions were no longer received, this was interpreted as indicating either death of the animal or failure of the tag. Tag failure could result from physical damage, electrical malfunction, or detachment of the tag from the animal. While it was not possible to estimate the probability of failure of the tags in this study, all the tags came from one manufacturing batch and there was no reason to expect differential deployment of unreliable tags on one group of seals over another. Tag loss from detachment was estimated assuming that the time to detachment would be normally distributed, as described in Hanson *et al.* (2013). The models used an initial estimate of 210 d for mean time to detachment; a value that was refined during model fitting. This high initial value was used to begin model fitting because there was often a local minimum, where low mean detachment times confused mortality and detachment, and trapped the model fitting algorithm at sets of parameters that described the data less well. The tag loss estimate was then used to right

sensor the data set prior to estimating seal survival in MARK. Tags that continued to transmit but were dry 100% of the time (*i.e.*, on shore) and never returned to the water, were assumed to have come ashore with an animal that was dead or dying or to have detached from a live animal while it was onshore. We treated these tags as no longer transmitting (*i.e.*, mortality) for all analyses. Tags that were consistently wet, *i.e.*, transmitting from an animal that was not returning to shore each day, were not observed. Because we expected tag life span and attachment to be the same for all groups, differences in tag duration reflected differences in pup survival.

To characterize seal movements and dispersal, the highest quality location data from each transmission day were identified (11% were LC 3, 25% were LC 2, 18% were LC 0, 13% were LC A, and 2% were LC B) and then the distance between these points measured. The spherical law of cosines was used to calculate the great circle distance between consecutive location estimates: $\text{Distance (km)} = \arccos[\sin(\text{lat}_1) \times \sin(\text{lat}_2) + \cos(\text{lat}_1) \times \cos(\text{lat}_2) \times \cos(\text{lon}_2 - \text{lon}_1)] \times R$, where R is the mean radius of the earth (6,371 km) and latitude and longitude are in radians. The distances were then summed to estimate distance travelled and divided by the length of time the tag transmitted to estimate a dispersal distance per day. Distance traveled and dispersal distance per day were minimums because (1) the path the seal took between two points was not known, and (2) a high quality transmission was not always received every other day so sometimes the gap between locations was 4 or 6 d. Additionally, the error in the location data may be greater than provided by ARGOS as found for elephant seals and several otariid species (Costa *et al.* 2010).

Distance traveled and dispersal distance per day were used to summarize how far and how quickly each seal went from its capture/release site, however, with a maximum of one location every other day, the data are quite coarse. Additionally, if seals traveled further initially, this covariate would be biased toward a greater dispersal distance per day among the seals that stopped transmitting earlier. Distance from deployment location to the last location received was also calculated. For animals tagged in SFB that exited the bay, distance was first calculated from the capture location to the mouth of the bay and then to the last location received to avoid crossing land. This distance from deployment is biased because travel was not always unidirectional away from the capture/release location.

Program MARK was used to model the effect of individual covariates on the probability of survival using a Cormack Jolly Seber live resighting framework (White and Burnham 1999). Each location received was considered to be a recapture, and binary encounter data summarized for each seal on a weekly basis as still transmitting (1) or no transmissions received (0). The final data set included the encounter data, a group covariate based on location (SF, TB, or TMMC), and the individual covariates. Because sample size did not support adding all the covariates into a single model set, several model sets with different individual covariates were assembled: model sets 1 through 4 were used to determine the best blood, morphology, and contaminant covariates to include in

model set 5. Model set 5 was used to model relative survival between the three groups of seals with the most appropriate individual covariates, and model set 6 was specific to the rehabilitated group of seals.

MARK used maximum likelihood to estimate a survival probability (Φ) that best fit the encounter data. Recapture probability (p) was set to 1 because the satellite tags provided 100% recapture rates until they stopped transmitting. Akaike's information criterion, corrected for small sample size (AICc), was calculated: this criterion balanced the fit of the model with the precision lost when additional parameters were estimated (Cooch and White 2009) and was used to compare models within a set of candidate models. Models with a $\Delta\text{AICc} < 2$ when compared with the model with the minimum AICc were considered strongly supported by the data, and models with a ΔAICc between two and four were considered to be supported by the data (Burnham and Anderson 2002). Median \hat{c} was used to test for goodness of fit.

RESULTS

Wild harbor seals were caught near the end of pupping season in May (SFB) and June (TB) 2008. Stranded harbor seals were admitted between 25 March and 19 May 2008. Seals in rehabilitation spent between 33 and 104 d in rehabilitation, gaining from 2.5 to 18 kg of mass during that time.

Morphology and Blood Variables

The body condition index was the only morphology covariate that differed among the seal locations. The TMMC pups had significantly poorer body condition than the SFB pups (ANOVA, Tukey's adjusted $P = 0.036$, Table 1). There were differences by group for the blood variables (Table 1): Immunoglobulins were significantly increased in TMMC pups compared with SFB (ANOVA, Tukey's adjusted $P < 0.005$) and TB (ANOVA, Tukey's adjusted $P = 0.039$) pups. Leukocyte count was significantly greater in TMMC pups than SFB pups (ANOVA, Tukey's adjusted $P = 0.004$). Thyroxine levels varied by group with T4 levels significantly greater in TB than SFB (ANOVA, Tukey's adjusted $P = 0.008$) and TMMC (ANOVA, Tukey's adjusted $P < 0.005$) pups. There were no differences in T3 among groups (ANOVA, $P = 0.113$, $df = 2$).

Associations Between Contaminants and the Other Variables

Contaminant concentrations were significantly less in TMMC pups (ANOVA, Tukey's adjusted $P < 0.005$) and TB pups (ANOVA, Tukey's adjusted $P = 0.025$) than the pups from SFB (Table 1). When the data were controlled for location, length was the only variable related to contaminant concentration with shorter animals associated with greater OH levels (glm, $t = -2.486$, $P = 0.017$, Table 2, Fig. 1). Contaminant concentrations were not predictive of T4 or T3 for any of the three seal groups (linear regression, $P > 0.05$).

Table 1. Mean and range for covariate values by group (SF = San Francisco, TB = Tomales Bay and TMMC = The Marine Mammal Center). The geometric mean is shown for contaminant concentrations (ng/g lipid weight).

	SF	TB	TMMC
Sample size	19	7	21
Morphology			
Mass (kg)	19 (13–27)	20 (16–25)	18 (13–25)
Length (cm)	85 (72–100)	88 (83–95)	86 (79–96)
Girth (cm)	72 (61–86)	74 (65–83)	69 (58–93)
Blubber depth (mm)	19 (14–25)	19 (14–23)	18 (13–22)
Body condition	1.0 (–3.0–7.5)	0.1 (–1.9–1.9)	–1.0 (–5.4–2.8) ^a
Sex (male, female)	9, 10	5, 2	9, 12
Blood			
IgG (mg/mL)	24 (20–33)	26 (21–29)	29 (26–32) ^a
WBC (/μL)	7.4 (4.3–11.1) ^b	8.3 (4.9–13.6) ^{a, b}	10.1 (6.2–15.0) ^a
T4 (nmol/L)	28 (10–58)	46 (18–62)	19 (7–40) ^a
T3 (nmol/L)	0.86 (0.53–2.32)	0.61 (0.39–0.94)	0.88 (0.52–1.46)
Contaminants			
PCB	9,777 ^a (2,594–30,075)	1,794 (668–10,627)	1,148 (229–7,528)
DDT	7,179 (2,738–24,436)	3,897 (992–17,380)	1,616 ^a (318–5,885)
PBDE	1,053 ^a (360–2,874)	192 (40–1,117)	154 (61–876)
CHLD	373 (125–883)	252 (126–817)	99 ^a (47–461)
HCH	27 (12–57)	43 ^a (20–70)	24 (17–42)
OH	18,601 ^a (5,836–57,855)	6,302 ^b (1,952–27,880)	3,160 (672–14,599)

Note: IgG = serum immunoglobulin, WBC = total white blood cell count, T4 = serum total thyroxine, T3 = serum total triiodothyronine, PCB = polychlorinated biphenyls, DDT = summed dichlorodiphenyltrichloroethane and its metabolites, PBDE = polybrominated diphenylethers, CHLD = chlordanes, HCH = hexachlorocyclohexanes, and OH = the five contaminant classes summed. Super-script letters represent significant differences among groups.

Table 2. Results for the significant generalized linear model of summed organohalogen contaminants (OH) controlling for group (SF = San Francisco, TB = Tomales Bay and TMMC = The Marine Mammal Center)

Dependent variable	Model parameter	Estimate	SE	<i>t</i>	<i>P</i>
OH	intercept	13.853	1.626	8.52	<0.005
	location_TB	–0.929	0.319	–2.91	0.006
	location_TMMC	–1.706	0.226	–7.554	<0.005
	length	–0.047	0.019	–2.486	0.017

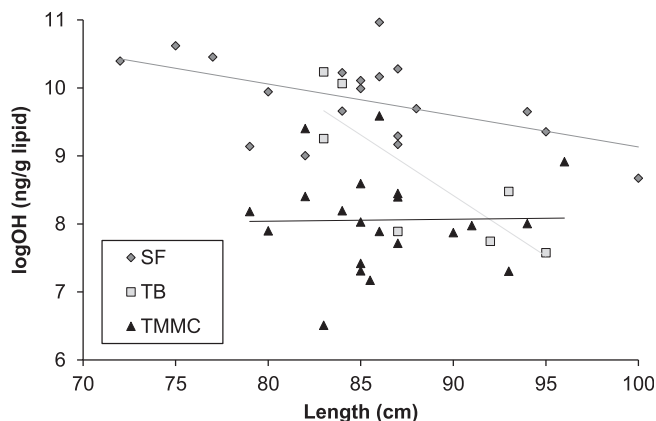


Figure 1. Relationship between standard length and summed organohalogen contaminants (OH) for the three groups: San Francisco Bay [$\log\text{OH} = (\text{length}) \times (-0.05) + 13.76$, $F = 6.117$, $\text{df} = 17$, $R^2 = 0.2646$, $P = 0.024$], Tomales Bay [$\log\text{OH} = (\text{length}) \times (-0.05) + 24.45$, $F = 10.3$, $\text{df} = 5$, $R^2 = 0.6733$, $P = 0.023$], and The Marine Mammal Center (not significant).

Satellite Telemetry

Transmissions from satellite tags deployed on seals from SFB ($n = 19$), TB ($n = 7$), and TMMC ($n = 21$) were received for eight months. Pups from TB survived longer than the other groups: at 16 wk, 5 of 7 TB pups were still transmitting, as opposed to 5 of 19 SFB pups and 2 of 21 TMMC pups (Fig. 2). One pup was recovered dead within San

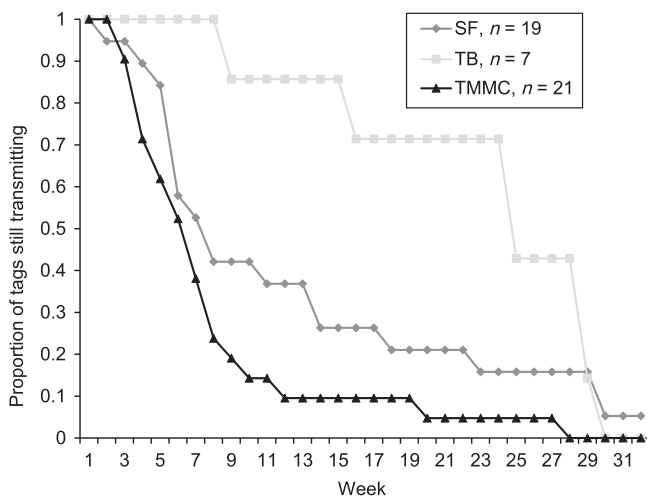


Figure 2. Proportion of tags still transmitting by week post deployment separated by location of deployment: SF = San Francisco Bay, TB = Tomales Bay, and TMMC = The Marine Mammal Center. Tags that were permanently ashore were considered to be no longer transmitting.

Francisco Bay 5 d after deployment with a fractured skull and humerus. The injury was consistent with collision with a high speed vessel and the animal otherwise had no lesions and a thick blubber layer (34 mm). Three tags presumed to have come ashore with dead or dying seals or to have detached from a live animal while it was onshore (*i.e.*, transmitted constantly from the same location) transmitted for 388, 403, and 417 d, confirming that the tags were capable of transmitting for over a year. Attempts to find and locate these tags were not successful.

Distance from deployment to final transmission ranged from 5 km to 802 km with the furthest location reached by a pup from SFB that stopped transmitting just south of Playas de Rosarito, Mexico (32.14°N, 117.08°W). Distance travelled ranged from 5 to 2,530 km with the shortest distance covered by the pup hit by a boat in SFB which transmitted for 4 d and the furthest distance covered by an SFB pup that transmitted for 232 d and stopped transmitting offshore of Coos Bay, Oregon (43.34°N, 124.45°W) 637 km from the capture location (Fig. 3). Dispersal distance per day varied from 0.5 to 49 km/d. Of those animals that transmitted for less than 6 wk, 17% traveled over 20 km/d (24% of TMMC pups and 16% of SFB pups, Fig. 4).

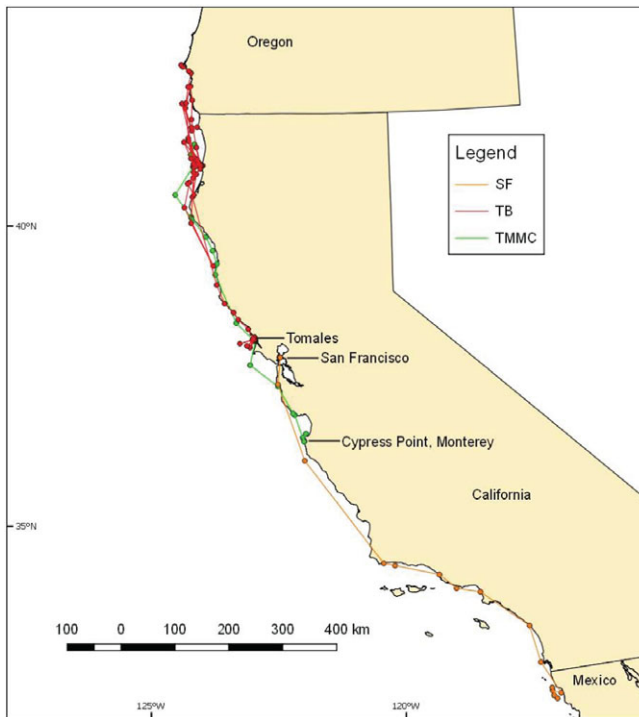


Figure 3. Map of the tracks taken by seals that traveled the greatest distance from their capture or release location for each of the three groups of seals. SF = San Francisco Bay, TB = Tomales Bay, and TMMC = The Marine Mammal Center. The TMMC seal that traveled the furthest was released from Cypress Point, Monterey.

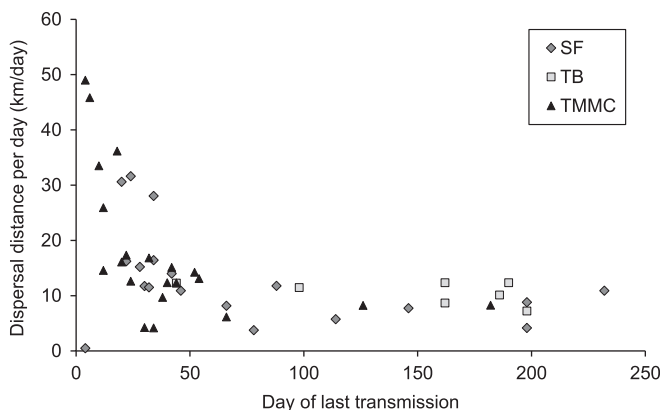


Figure 4. The relationship between total tag deployment time and dispersal distance per day for satellite tagged seal pups.

Using the TMMC and a combined SFB + TB model set to estimate tag loss, two exponential curves best fit the data and yielded a normal tag loss curve with a mean of 201 d and SD = 20 d (Fig. 5). Subtracting two standard deviations from the mean resulted in 161 d (23 wk) of satellite data before effects from tag loss were predicted to affect survival estimates, therefore we right censored the encounter data to 23 wk to model the individual covariate effects on survival. The effect of tag loss is more evident on the survival curve for the wild-caught seals because so few of the TMMC animals transmitted long enough for tag loss to become an issue (Fig. 5).

Correlates of Survival

Model set 1 (group and time)—The first model set contained location (SF, TMMC, TB) as a group covariate. The best model identified by MARK included a location effect on survival (Table 3). The $\{\Phi(g)\}$ model was a good fit to the data with a median \hat{c} of 1.15 (indicating minimal over dispersion of the data). This \hat{c} was used in all the model sets to convert the AICc to QAICc. There was no support for a model with time dependent survival (*i.e.*, a model with different survival probabilities in different weeks). There was support for grouping SFB and TMMC together in terms of their estimated survival probabilities, but this model would not be appropriate for model sets with contaminant concentration as an individual covariate because of the known difference in contaminant concentration between those two groups (Table 3).

Model set 2 (contaminant classes)—This model set included the location group covariate and the lipid weight summed contaminants for the various contaminant classes (PCB, DDT, PBDE, CHLD, and HCH) as individual covariates. There was support for all the models with

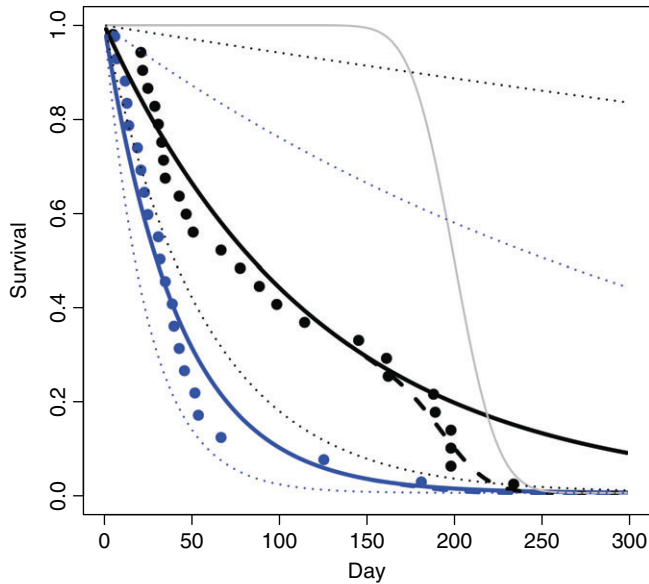


Figure 5. The last day of satellite tag transmission for each seal (circles) modeled by two exponential curves (solid lines) with 95% confidence intervals (dotted lines). Data and estimates are in black for the wild (SF and TB) seals and blue for the rehabilitated (TMMC) seals. The gray line represents tag survival based on a normal distribution. The dashed lines display the effect of tag loss on the two survival curves.

Table 3. Model selection of apparent survival probability (Φ) for model set 1: group and time.

Model	QAICc	Δ QAICc	QAICc weights	Model likelihood	No. parameters	Deviance
$\{\Phi(g)\}$	222.45	0.000	0.538	1.000	3	49.658
$\{\Phi(\text{SF and TMMC v TB})\}$	222.95	0.501	0.419	0.779	2	52.184
$\{\Phi(\text{SF and TB v TMMC})\}$	227.55	5.102	0.042	0.078	2	56.786
$\{\Phi(\cdot)\}$	233.43	10.983	0.002	0.004	1	64.684
$\{\Phi(t)\}$	254.10	31.655	0.000	0.000	22	41.095
$\{\Phi(g \times t)\}$	320.74	98.291	0.000	0.000	66	0.000

Note: t = time, g = group (SF = San Francisco, TB = Tomales Bay, and TMMC = The Marine Mammal Center). \hat{c} adjustment = 1.15. Highest ranked model highlighted in bold.

contaminant covariates (Δ QAICc < 2) although the model with location only $\{\Phi(g)\}$ was still the best model (Table 4).

Model set 3 (morphology and sex)—Location was again retained as a group covariate and mass, length, girth, body condition, blubber depth, and sex were the individual covariates. The best models included length,

Table 4. Model selection of apparent survival probability (Φ) for model set 2: lipid weight contaminant classes.

Model	QAICc	Δ QAICc	QAICc weights	Model likelihood	No. parameters	Deviance
$\{\Phi(g)\}$	220.57	0.000	0.226	1.000	3	214.517
$\{\Phi(g + \log\text{PBDE})\}$	221.04	0.473	0.179	0.789	4	212.955
$\{\Phi(g + \log\text{HCH})\}$	221.11	0.544	0.172	0.762	4	213.026
$\{\Phi(g + \log\text{CHLD})\}$	221.67	1.104	0.130	0.576	4	213.586
$\{\Phi(g + \log\text{PCB})\}$	222.06	1.491	0.107	0.475	4	213.973
$\{\Phi(g \times \log\text{HCH})\}$	222.25	1.683	0.098	0.431	6	210.070
$\{\Phi(g + \log\text{DDT})\}$	222.46	1.894	0.088	0.388	4	214.376

Note: g = group (SF, TB, and TMMC), PCB = polychlorinated biphenyls, DDT = summed dichlorodiphenyltrichloroethane and its metabolites, PBDE = polybrominated diphenylethers, CHLD = chlordanes, and HCH = hexachloro-cyclohexanes. \hat{c} adjustment = 1.15. Highest ranked model highlighted in bold.

Table 5. Model selection of apparent survival probability (Φ) for model set 3: morphology and sex.

Model	QAICc	Δ QAICc	QAICc weights	Model likelihood	No. parameters	Deviance
$\{\Phi(g + \text{length})\}$	219.90	0.000	0.191	1.000	4	211.810
$\{\Phi(g + \text{mass})\}$	220.44	0.544	0.145	0.762	4	212.354
$\{\Phi(g + \text{sex} + \text{length})\}$	220.46	0.567	0.144	0.753	5	210.334
$\{\Phi(g)\}$	220.57	0.672	0.136	0.715	3	214.517
$\{\Phi(g + \text{sex})\}$	220.83	0.935	0.120	0.627	4	212.745
$\{\Phi(g + \text{sex} + \text{mass})\}$	221.16	1.267	0.101	0.531	5	211.034
$\{\Phi(g + \text{bd})\}$	222.23	2.330	0.060	0.312	4	214.140
$\{\Phi(g + \text{cond})\}$	222.45	2.557	0.053	0.278	4	214.368
$\{\Phi(g + \text{girth})\}$	222.56	2.662	0.050	0.264	4	214.472

Note: g = group (SF, TB, and TMMC), bd = blubber depth, cond = the body condition index. \hat{c} adjustment = 1.15. Highest ranked model highlighted in bold.

mass, and sex plus length (Table 5). Increased length, increased mass, and female pups were each associated with increased survival probability.

Model set 4 (blood variables)—This model set contained the location group covariate and T4, T3, WBC, and IgG as individual covariates. The best model was $\{\Phi(g + T4)\}$ with decreased T4 associated with decreased probability of survival (Table 6).

Model set 5 (top covariates from previous model sets)—Based on the results from model sets 1 through 4, this model set contained the group covariate and OH, mass, and T4 as individual covariates. Total OH was used for the contaminant covariate because all contaminant classes were similarly supported, and mass was used instead of length to avoid confounding because length was associated with contaminant

Table 6. Model selection of apparent survival probability (Φ) for model set 4: blood variables.

Model	QAICc	Δ QAICc	QAICc weights	Model likelihood	No. parameters	Deviance
$\{\Phi(g + T4)\}$	217.45	0.000	0.481	1.000	4	209.366
$\{\Phi(g + T4 + T3)\}$	219.49	2.033	0.174	0.362	5	209.356
$\{\Phi(g + T3)\}$	220.04	2.587	0.132	0.274	4	211.953
$\{\Phi(g)\}$	220.57	3.116	0.101	0.211	3	214.517
$\{\Phi(g + wbc)\}$	221.49	4.040	0.064	0.133	4	213.406
$\{\Phi(g + IgG)\}$	222.04	4.589	0.048	0.101	4	213.955

Note: g = group (SF, TB, and TMMC), $T4$ = total thyroxine, $T3$ = triiodothyronine, WBC = white blood cell count, IgG = total immunoglobulin. \hat{c} adjustment = 1.15. Highest ranked model highlighted in bold.

Table 7. Model selection of apparent survival probability (Φ) for model set 5 incorporating top covariates from previous models.

Model	QAICc	Δ QAICc	QAICc weights	Model likelihood	No. parameters	Deviance
$\{\Phi(g + T4)\}$	248.8571	0	0.37137	1	4	240.7709
$\{\Phi(g + T4 + \text{mass})\}$	250.1334	1.2763	0.19618	0.5283	5	240.0038
$\{\Phi(g + T4 + \log\text{OH})\}$	250.298	1.4409	0.18068	0.4865	5	240.1684
$\{\Phi(g + T4 + \text{mass} + \log\text{OH})\}$	251.8598	3.0027	0.08275	0.2228	6	239.678
$\{\Phi(g + \text{mass})\}$	252.2936	3.4365	0.06662	0.1794	4	244.2074
$\{\Phi(g)\}$	252.7458	3.8887	0.05314	0.1431	3	246.6942
$\{\Phi(g + \text{mass} + \log\text{OH})\}$	254.2608	5.4037	0.02491	0.0671	5	244.1312
$\{\Phi(g + \log\text{OH})\}$	254.3069	5.4498	0.02434	0.0655	4	246.2207

Note: g = group (SF, TB, and TMMC), $T4$ = total thyroxine, OH = PCB+DDT+PBDE+HCH+CHLD, Φ = survival.

concentration. The best model was $\{\Phi(g + T4)\}$, but there was also strong support for $\{\Phi(g + T4 + \log\text{OH})\}$ and $\{\Phi(g + T4 + \text{mass})\}$ (Table 7). Increased OH, and decreased $T4$ were associated with decreased survival probability (Fig. 6). The weekly survival estimates from the top model yielded a 23 wk estimated survival probability of 0.127 (95% CI 0.035–0.285) for SFB, 0.062 (95% CI 0.010–0.189) for TMMC, and 0.524 (95% CI 0.071–0.859) for TB (see Table 8 for weekly survival estimates).

Model set 6 (TMMC, rehabilitation covariates)—This model set contained only the encounter data from TMMC pups and incorporated mass, $T4$, OH, and individual covariates that were specific to the animals in rehabilitation, such as date of admission (in Julian days), number of days

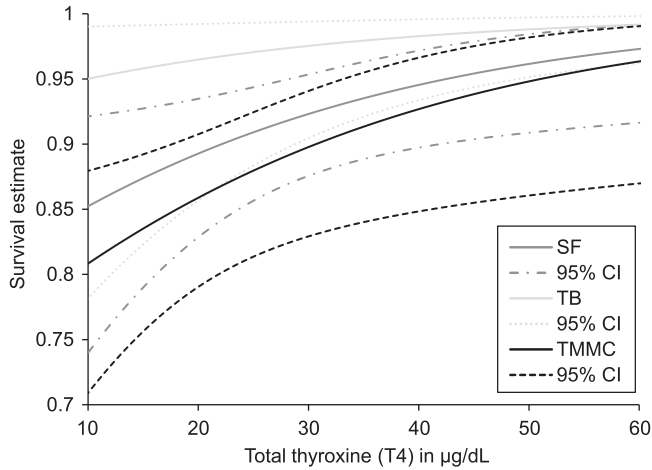


Figure 6. Association between survival probability estimates with circulating T4 (nmol/L) at the time of capture/release from the best supported model: $\{\Phi(g + T4)\}$.

Table 8. Weekly survival estimate, standard error, and 95% confidence interval from the top model: $\{\Phi(g + T4)\}$.

	Estimate	SE	LCL	UCL
SF	0.914	0.021	0.864	0.947
TMMC	0.886	0.028	0.820	0.930
TB	0.972	0.020	0.891	0.993

in rehabilitation, change in mass from admission to release, and mass gain per day. The best model was $\{\Phi(\text{mass})\}$ with increased mass at the time of release associated with increased survival (Fig. 7) and there was equal support for the positive association with T4 (Table 9). There was also support for the rehabilitation specific covariates: greater total mass gain, a shorter time in rehabilitation, and admission to rehabilitation earlier in the season were all associated with an increase in survival probability.

DISCUSSION

In postweaning harbor seals, increased 6 mo survival in seals from different locations with different health histories was associated with increased mass, increased T4, and decreased blubber organohalogen concentration. Factors associated with growth and metabolism (T4) and morphology (mass) were more important than those associated with inflammation (WBC and IgG). This may simply confirm that the animals were clinically healthy at the time of sampling and tag deployment, and, had any of the seals been sick (*i.e.*, had markedly different WBC and

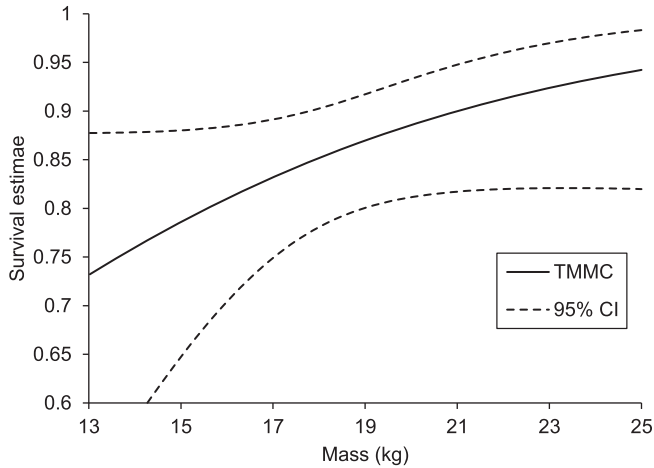


Figure 7. Association between the survival probability estimate and mass (kg) at the time of release from rehabilitation from the best supported TMMC model: $\{\Phi(\text{mass})\}$.

Table 9. Model selection of apparent survival probability (Φ) for model set 6 investigating individual covariates specific to rehabilitated seal pups (TMMC).

Model	QAICc	Δ QAICc	QAICc weights	Model likelihood	No. parameters	Deviance
$\{\Phi(\text{mass})\}$	101.79	0.000	0.220	1.000	2	97.704
$\{\Phi(\text{T4})\}$	102.08	0.287	0.190	0.866	2	97.991
$\{\Phi(\cdot)\}$	102.42	0.625	0.161	0.732	1	100.387
$\{\Phi(\text{change in mass})\}$	102.80	1.013	0.133	0.603	2	98.717
$\{\Phi(\text{days in rehab})\}$	103.47	1.679	0.095	0.432	2	99.383
$\{\Phi(\text{admit date})\}$	103.73	1.942	0.083	0.379	2	99.646
$\{\Phi(\log\text{OH})\}$	104.38	2.593	0.060	0.273	2	100.297
$\{\Phi(\text{mass per day})\}$	104.46	2.666	0.058	0.264	2	100.370

Note: T4 = total thyroxine, adjustment = 1.15.

IgG levels), these health factors might have been more strongly associated with survival probability.

Although WBC and IgG levels were increased in the TMMC pups, there was only moderate support for the association of these immune parameters with the survival estimates. It is possible that there is a threshold effect for these parameters as was suggested by Neale *et al.* (2005). Neale *et al.* (2005) reported a significant positive relationship between WBC and whole blood polychlorinated biphenyls (PCB), though that was driven by three data points with a WBC greater than 17,000/ μL , a level greater than found in the seals in this study (maximum = 15,000/ μL). Tabuchi *et al.* (2006) found decreased circulating T4 and an increase in thyroid receptors in the blubber among harbor seal pups with increased PCB concentrations in the blubber. Our study

supports that link between decreased T4 and increased contaminant levels and adds an association with decreased survival probability.

In contrast to studies of seals in the United Kingdom (Hall *et al.* 2003, Hall and Thomas 2007), we did not identify statistically significant associations between contaminant classes and circulating levels of T3 or T4. The contaminant profiles of harbor seals in the United Kingdom studies differed from those in the seals we sampled. For example, concentrations of the endocrine disrupting pesticide lindane (the γ HCH isomer) were greater in the UK seals than in seals from central California (Hall and Thomas 2007, see Greig *et al.* 2011 for the isomeric details of each contaminant class). Furthermore, seals in the United Kingdom varied in age, and for pups, corrections were made for the number of days between each pup's weaning and sampling. In wild harbor and gray seal pups, T4 levels decline from birth to weaning (Haulena *et al.* 1998, Hall *et al.* 1998). Without knowing the exact age of the pups in this study, or the thyroid dynamics in rehabilitating seals, it was not possible to interpret the interactions between blubber contaminants, thyroid levels, growth, and metabolism. However, the relationship between length and contaminant concentration indicated that these interactions may be worth investigating further.

Whereas the survival curve of the pups released from rehabilitation was similar to that of the wild-caught pups from San Francisco, survival probability was associated with different factors, with OH more important in SFB than in rehabilitated seals. This was consistent with the higher level of contamination in the SFB weaned pups compared with the rehabilitated seals that did not suckle contaminated milk. Mass at the time of release was the most important correlate of survival in the rehabilitated pups. Wild harbor seal pups on Sable Island, Nova Scotia, took 4–6 wk before they were able to forage effectively, and during this time lost approximately half their fat stores, but only about 20% of their total body mass (Muelbert *et al.* 2003). Two harbor seal pups that restranded after release from rehabilitation in 2007 both lost significant mass rapidly: one lost 21% of its body mass in 16 d and the other lost 32% in 37 d (TMMC unpublished data), suggesting that, compared with the wild pups in Muelbert *et al.* (2003), they were depleting more of their fat stores and/or failing to add muscle. Lander *et al.* (2002) reported two similar instances of mass loss in rehabilitated seal pups postrelease. Our rehabilitating seals that gained more weight in shorter periods of time increased their chance of survival: this more closely mimics what occurs naturally with pups weaning 3–5 wk after birth. In addition, Lander *et al.* (2002) reported that rehabilitated harbor seal pups spent significantly more time in the water than wild pups, and Gaydos *et al.* (2012) reported that, during their first 34 d postrelease, the mean daily rate of travel for rehabilitated pups was greater than for wild pups. Pups raised in captivity may take longer than wild pups to forage effectively and an extra reserve of body fat may be required to carry them through the time they need to adjust to a new environment and learn foraging skills.

Survival rates among the rehabilitated and wild-caught pups were less than those estimated by Lander *et al.* (2002). Survival rate differences may represent annual variation or may be related to the release masses

of the harbor seals; the rehabilitated pups in Lander *et al.* (2002) were not released until they were 20 kg (*i.e.*, heavier than many of the pups in this study). Lander *et al.* (2002) additionally suggested that difference in pelage quality between rehabilitated and wild-caught seals may have resulted in differential tag loss. Our study does not address this directly: although one rehabilitated seal retained its tag as long as any of the wild-caught seals, the others stopped transmitting well before tag loss was expected to occur. In this study, if a tag stopped transmitting, that animal was considered dead, so if the tag was retained on the animal, but failed for a different reason than animal mortality (*e.g.*, battery failure or antenna loss), our study would have overestimated the probability of mortality.

This study is the first to show the extent of harbor seal pup dispersal along the west coast of the United States and Mexico. Harbor seal pups travelled as far as Oregon and Mexico, well beyond the area generally assumed on the basis of genetic differences (Lamont *et al.* 1996). Recent increases in harbor seal pup numbers born in Mexico as well as an extralimital sighting of a weaned harbor seal pup on Guadalupe Island, Mexico, raise further questions about the movements of these seals from postweaning to recruitment (Lubinsky-Jinich *et al.* 2017, Orr *et al.* 2018). Whether these pups return to their natal beaches before recruiting to the population as reproductive adults is not known. Behavioral differences between rehabilitated and wild harbor seal pups have been reported with rehabilitated pups traveling further from the release site than wild pups (Gaydos *et al.* 2012). This pattern was not evident in our data set in which both rehabilitated and wild seals quickly dispersed. Although the harbor seal population in California was considered stable at the time of this study (Carretta *et al.* 2007), these dispersal patterns have management implications should juvenile mortality from habitat factors become a concern for this population, as these pups travel well outside of current marine protected areas.

In summary, T4, mass, and contaminant concentrations were all associated with survival probability; and harbor seal pups that gained the most mass in rehabilitation had an increased probability of surviving their first 6 mo. In terms of conservation and management, the most important anthropogenic factor affecting survival, organohalogen contamination, has been addressed in the United States with bans on production and use of PCBs, DDT, CHLs, and HCHs since the 1970s (Goldberg 1991) and more recently on most PBDEs in the 2000s (Betts 2008). Despite bans and clean-up efforts, these lipophilic contaminants are still present and exerting effects in the marine environment and it will be important to monitor the effects of these as well as new, replacement compounds. This study illuminates another factor decreasing harbor seal pup survival in San Francisco Bay with the first report of a probable boat strike; the risk to harbor seal pups in San Francisco Bay from vessel traffic merits further investigation. Among stranded pups in rehabilitation, the best strategy to increase the probability of postrelease survival appears to be increasing pup mass prior to release. Survival correlates like T4 may be related to the ecosystem and prey availability, and

further study on prey variability and patterns of dispersal will be useful for understanding harbor seal population dynamics in California.

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LITERATURE CITED

- Baker, J. D. 2008. Variation in the relationship between offspring size and survival provides insight into causes of mortality in Hawaiian monk seals. *Endangered Species Research* 5:55–64.
- Bennett, K. A., J. R. Speakman, S. E. Moss, P. Pomeroy and M. A. Fedak. 2007. Effects of mass and body composition on fasting fuel utilisation in grey seal pups (*Halichoerus grypus* Fabricius): An experimental study using supplementary feeding. *Journal of Experimental Biology* 210:3043–3053.
- Betts, K. S. 2008. New thinking on flame retardants. *Environmental Health Perspectives* 116:A210–A213.
- Bigg, M. A. 1969. The harbor seal in British Columbia. *Bulletin of the Fisheries Research Board of Canada* 172. 33 pp.
- Boulva, J., and I. A. McLaren. 1979. Biology of the harbor seal, *Phoca vitulina*, in Eastern Canada. *Bulletin of the Fisheries Research Board of Canada* 200. 24 pp.
- Bowen, W. D., S. J. Iverson, D. J. Boness and O. T. Oftedal. 2001. Foraging effort, food intake and lactation performance depend on maternal mass in a small phocid seal. *Functional Ecology* 15:325–334.
- Brookens, T., J. Harvey and T. O'Hara. 2007. Trace element concentrations in the Pacific harbor seal (*Phoca vitulina richardii*) in central and northern California. *Science of the Total Environment* 372:676–692.
- Burnham, K. P., and D. R. Anderson. 2002. *Model selection and multimodel inference: A practical information-theoretic approach*. 2nd edition. Springer, New York, NY.
- Carretta, J. V., K. A. Forney, M. S. Lowry, J. Barlow, J. Baker, B. Hanson and M. M. Muto. 2007. U.S. Pacific marine mammal stock assessments: 2007. U.S. Department of Commerce, NOAA-TM-NWFS-SWFSC-414. 316 pp.
- Coltman, D. W., W. D. Bowen and J. M. Wright. 1998. Birth weight and neonatal survival of harbour seal pups are positively correlated with genetic variation measured by microsatellites. *Proceedings of the Royal Society B: Biological Sciences* 265:803–809.
- Conomos, T. J., R. E. Smith and J. W. Gartner. 1985. Environmental setting of San Francisco Bay. *Hydrobiologia* 129(1):1–12.
- Cooch, E., and G. White. 2009. Program MARK “A Gentle Introduction”. Available at <http://www.phidot.org/software/mark/docs/book/>.
- Cordes, L. S., and P. M. Thompson. 2014. Mark-recapture modeling accounting for state uncertainty provides concurrent estimates of survival and fecundity in a protected harbor seal population. *Marine Mammal Science* 30: 691–705.

- Costa, D. P., P. W. Robinson, J. P. Y. Arnould, *et al.* 2010. Accuracy of ARGOS locations of pinnipeds at-sea estimated using Fastloc GPS. *PLoS ONE* 5(1): e8677.
- CLS. 2008. Argos user's manual. Available at <http://www.argos-system.org/manual/>.
- Craig, M. P., and T. J. Ragen. 1999. Body size, survival, and decline of juvenile Hawaiian monk seals, *Monachus schauinslandi*. *Marine Mammal Science* 15:786–809.
- De Guise, S., D. Martineau, P. Beland and M. Fournier. 1995. Possible mechanisms of action of environmental contaminants on St. Lawrence beluga whales (*Delphinapterus leucas*). *Environmental Health Perspectives* 103 Supplement 4:73–77.
- De Swart, R. L., P. S. Ross, J. G. Vos and A. D. Osterhaus. 1996. Impaired immunity in harbour seals (*Phoca vitulina*) exposed to bioaccumulated environmental contaminants: Review of a long-term feeding study. *Environmental Health Perspectives* 104:823–828.
- Eberhardt, L. L., and D. B. Siniff. 1977. Population dynamics and marine mammal management policies. *Journal of the Fisheries Research Board of Canada* 34:183–190.
- Gaydos, J. K., L. I. Vilchis, M. M. Lance, *et al.* 2012. Postrelease movement of rehabilitated harbor seal (*Phoca vitulina*) pups compared with cohort-matched wild seal pups. *Marine Mammal Science* 29:E282–E294.
- Goldberg, E. D. 1991. Halogenated hydrocarbons: Past, present and near-future problems. *Science of The Total Environment* 100:17–28.
- Greig, D. J., F. M. D. Gulland, C. A. Rios and A. J. Hall. 2010. Hematology and serum chemistry in stranded and wild-caught harbor seals in central California: Reference intervals, predictors of survival, and parameters affecting blood variables. *Journal of Wildlife Diseases* 46:1172–1184.
- Greig, D. J., G. M. Ylitalo, E. A. Wheeler, *et al.* 2011. Geography and stage of development affect persistent organic pollutants in stranded and wild-caught harbor seal pups from central California. *Science of the Total Environment* 409:3537–3547.
- Greig, D. J., F. M. D. Gulland, W. A. Smith, *et al.* 2014. Surveillance for zoonotic and selected pathogens in harbor seals (*Phoca vitulina*) from central California. *Diseases of Aquatic Organisms* 111:93–106.
- Grigg, E. K., S. G. Allen, D. E. Green and H. Markowitz. 2004. Harbor seal, *Phoca vitulina richardii*, population trends in the San Francisco Bay estuary, 1970–2002. *California Fish and Game* 90:51–70.
- Hall, A. J., N. J. L. Green, K. C. Jones, P. P. Pomeroy and J. Harwood. 1998. Thyroid hormones as biomarkers in grey seals. *Marine Pollution Bulletin* 36:424–428.
- Hall, A. J., B. J. McConnell and R. J. Barker. 2001. Factors affecting first-year survival in grey seals and their implications for life history strategy. *Journal of Animal Ecology* 70:138–149.
- Hall, A. J., B. J. McConnell and R. J. Barker. 2002. The effect of total immunoglobulin levels, mass and condition on the first-year survival of grey seal pups. *Functional Ecology* 16:462–474.
- Hall, A. J., O. I. Kalantzi and G. O. Thomas. 2003. Polybrominated diphenyl ethers (PBDEs) in grey seals during their first year of life—are they thyroid hormone endocrine disrupters? *Environmental Pollution* 126:29–37.
- Hall, A. J., and G. O. Thomas. 2007. Polychlorinated biphenyls, DDT, polybrominated diphenyl ethers, and organic pesticides in United Kingdom harbor seals (*Phoca vitulina*)—mixed exposures and thyroid homeostasis. *Environmental Toxicology and Chemistry* 26:851–861.

- Hall, A. J., G. O. Thomas and B. J. McConnell. 2009. Exposure to Persistent organic pollutants and first-year survival probability in gray seal pups. *Environmental Science and Technology* 43:6364–6369.
- Hammond, J. A., A. J. Hall, and E. A. Dyrinda. 2005. Comparison of polychlorinated biphenyl (PCB) induced effects on innate immune functions in harbour and grey seals. *Aquatic Toxicology* 74:126–138.
- Hanson N., D. Thompson, C. Duck, S. Moss, M. Lonergan. 2013. Pup mortality in a rapidly declining harbour seal (*Phoca vitulina*) population. *PLoS ONE* 8(11):e80727.
- Harding, K. C., M. Fujiwara, Y. Axberg and T. Harrkonen. 2005. Mass-dependent energetics and survival in harbour seal pups. *Functional Ecology* 19:129–135.
- Haulena, M., D. J. St. Aubin and P. J. Duignan. 1998. Thyroid hormone dynamics during the nursing period in harbour seals, *Phoca vitulina*. *Canadian Journal of Zoology* 76:48–55.
- Jepson, P. D., P. M. Bennett, R. Deaville, C. R. Allchin, J. R. Baker and R. J. Law. 2005. Relationships between polychlorinated biphenyls and health status in harbor porpoises (*Phocoena phocoena*) stranded in the United Kingdom. *Environmental Toxicology and Chemistry* 24:238–248.
- Kimbro, D.K., J. Largier, and E. D. Grosholz. 2009. Coastal oceanographic processes influence the growth and size of a key estuarine species, the Olympia oyster. *Limnology and Oceanography* 54:1425–1437.
- Kopec, A. D., and J. T. Harvey. 1995. Toxic pollutants, health indices, and population dynamics of harbor seals in San Francisco Bay, 1989–1992. Moss Landing Marine Laboratories, Technical Publication 96-4. 138 pp.
- Lamont, M. M., J. T. Vida, J. T. Harvey, *et al.* 1996. Genetic substructure of the Pacific harbor seal (*Phoca vitulina richardsi*) off Washington, Oregon, and California. *Marine Mammal Science* 12:402–413.
- Lander, M. E., J. T. Harvey, K. D. Hanni and L. E. Morgan. 2002. Behavior, movements, and apparent survival of rehabilitated and free-ranging harbor seal pups. *Journal of Wildlife Diseases* 66:19–28.
- Levin, M., S. De Guise and P. S. Ross. 2005. Association between lymphocyte proliferation and polychlorinated biphenyls in free-ranging harbor seal (*Phoca vitulina*) pups from British Columbia, Canada. *Environmental Toxicology and Chemistry* 24:1247–1252.
- Lonergan, M., C. Duck, S. Moss, C. Morris and D. Thompson. 2013. Rescaling of aerial survey data with information from small numbers of telemetry tags to estimate the size of a declining harbour seal population. *Aquatic Conservation: Marine and Freshwater Ecosystems* 23:135–144.
- Lubinsky-Jinich, D., Y. Schramm and G. Heckel. 2017. The Pacific harbor seal's (*Phoca vitulina richardii*) breeding colonies in Mexico: Abundance and distribution. *Aquatic Mammals* 43:73–81.
- Lunn, N. J., I. L. Boyd and J. P. Croxall. 1994. Reproductive performance of female Antarctic fur seals: The influence of age, breeding experience, environmental variation and individual quality. *Journal of Animal Ecology* 63: 827–840.
- Mackey, B. L., J. W. Durban, S. J. Middlemas and P. M. Thompson. 2008. A Bayesian estimate of harbour seal survival using sparse photo-identification data. *Journal of Zoology* 274:18–27.
- Manugian, S. C., D. J. Greig, D. Lee, B. H. Becker, S. Allen, M. S. Lowry and J. T. Harvey. 2017. Survival probabilities and movements in central California. *Marine Mammal Science* 33:154–171.
- McMahon, C. R., and H. R. Burton. 2005. Climate change and seal survival: Evidence for environmentally mediated changes in elephant seal, *Mirounga*

- leonina*, pup survival. Proceedings of the Royal Society B: Biological Sciences 272:923–928.
- Muelbert, M. M., W. D. Bowen and S. J. Iverson. 2003. Weaning mass affects changes in body composition and food intake in harbour seal pups during the first month of independence. Physiological and Biochemical Zoology 76:418–427.
- Neale, J. C., J. A. Van De Water, J. T. Harvey, R. S. Tjeerdema and M. E. Gershwin. 2002. Proliferative responses of harbor seal (*Phoca vitulina*) T lymphocytes to model marine pollutants. Developmental Immunology 9:215–221.
- Neale, J. C. C., F. M. D. Gulland, K. R. Schmelzer, *et al.* 2005. Contaminant loads and hematological correlates in the harbor seal (*Phoca vitulina*) of San Francisco Bay, California. Journal of Toxicology and Environmental Health, Part A 68:617–633.
- Ono, K. A., D. J. Boness and O. T. Oftedal. 1987. The effect of a natural environmental disturbance on maternal investment and pup behavior in the California sea lion. Behavioral Ecology and Sociobiology 21:109–118.
- Orr, A. J., T. A. Norris, C. Gálvez, A. C. Deming and F. R. Elorriaga-Verplancken. 2018. First extralimital record of an Eastern Pacific harbor seal (*Phoca vitulina richardii*) pup at Guadalupe Island, Mexico. Aquatic Mammals 44:279–284.
- R Development Core Team. 2009. R: A language and environment for statistical computing. R Foundation for Statistical Computing, Vienna, Austria.
- Reijnders, P. J. H. 1978. Recruitment in the harbour seal (*Phoca vitulina*) population in the Dutch Wadden Sea. Netherlands Journal of Sea Research 12:164–179.
- Reijnders, P. J. H. 1980. Organochlorine and heavy metal residues in harbour seals from the Wadden Sea and their possible effects on reproduction. Netherlands Journal of Sea Research 14:30–65.
- Ross, P., B. Pohajdak, W. Bowen and R. Addison. 1993. Immune function in free-ranging harbor seal (*Phoca vitulina*) mothers and their pups during lactation. Journal of Wildlife Diseases 29:21–29.
- Ross, P. S., R. L. D. Swart, P. J. H. Reijnders, H. V. Loveren, J. G. Vos and A. D. M. E. Osterhaus. 1995. Contaminant-related suppression of delayed-type hypersensitivity and antibody responses in harbor seals fed herring from the Baltic Sea. Environmental Health Perspectives 103:162–167.
- Ross, P. S., S. J. Jeffries, M. B. Yunker, R. F. Addison, M. G. Ikonomou and J. C. Calambokidis. 2004. Harbor seals (*Phoca vitulina*) in British Columbia, Canada, and Washington State, USA, reveal a combination of local and global polychlorinated biphenyl, dioxin, and furan signals. Environmental Toxicology and Chemistry 23:157–165.
- Small, R. J., P. L. Boveng, G. V. Byrd and D. E. Withrow. 2008. Harbor seal population decline in the Aleutian Archipelago. Marine Mammal Science 24:845–863.
- Steiger, G. H., J. Calambokidis, J. C. Cabbage, D. E. Skilling, A. W. Smith and D. H. Gribble. 1989. Mortality of harbor seal pups at different sites in the inland waters of Washington. Journal of Wildlife Diseases 25:319–328.
- Sydeman, W. J., and S. G. Allen. 1999. Pinniped population dynamics in Central California: Correlations with sea surface temperature and upwelling indices. Marine Mammal Science 15:446–461.
- Tabuchi, M., N. Veldhoen, N. Dangerfield, S. Jeffries, C. C. Helbing and P. S. Ross. 2006. PCB-related alteration of thyroid hormones and thyroid hormone receptor gene expression in free-ranging harbor seals (*Phoca vitulina*). Environmental Health Perspectives 114:1024–1031.
- White, G. C., and K. P. Burnham. 1999. Program MARK: Survival estimation from populations of marked animals. Bird Study 46:120–138.
- Womble, J. N., G. W. Pendleton, E. A. Mathews, G. M. Blundell, N. M. Bool and S. M. Gende. 2010. Harbor seal (*Phoca vitulina richardii*) decline

continues in the rapidly changing landscape of Glacier Bay National Park, Alaska 1992–2008. *Marine Mammal Science* 26:686–697.

Ylitalo, G. M., J. E. Stein, T. Hom, *et al.* 2005. The role of organochlorines in cancer-associated mortality in California sea lions (*Zalophus californianus*). *Marine Pollution Bulletin* 50:30–39.

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