Methods and models to determine perinatal status of Florida manatee carcasses

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Abstract

Differences in perinatal mortality can be indicators of differences in physiology, behavior, toxicology, population dynamics, and ecology of species and individuals. Extensive data collected under the Florida Manatee Carcass Recovery Program provide important information about manatee perinatal mortality. However, early age at death can be difficult to determine for often highly decomposed carcasses. Here, I provide quantitative methods to identify perinatal status for manatee carcasses. First, perinatal and nonperinatal mortality were defined based on physiological indicators. After review of necropsy reports, specific length classes became clear indicators of perinatal status: carcasses <82-cm long were always perinatal, and carcasses >160-cm long were always nonperinatal. Using data from carcasses 82–160 cm long of known perinatal status, Bayesian models quantify the relationship among age at death, carcass length, and carcass recovery month. The models predict the perinatal status of carcass 82–160 cm long when physiological indicators are unavailable. Overall, perinatal status could be determined for 98.2% of carcasses collected from 1978 to 2005. Initial examination of the fraction of perinatal mortalities within the entire carcass sample reveals interesting spatial and temporal patterns that warrant further analyses.

Key words: Florida manatee, *Trichechus manatus*, perinatal mortality, Bayesian analysis.

Perinatal mortalities, deaths occurring around the time of birth, can provide managers and scientists with valuable information on the overall condition of populations, particularly when attempting to identify effective conservation measures (Ono *et al.* 1987, Courtenay and Santow 1989, Escos and Alados 1991, Rothe *et al.* 1992, Lent and Davis 1993, Frank 1997, Beckmen *et al.* 2003, Linklater *et al.* 2004, Pojar and Bowden 2004, Gutierrez *et al.* 2005, Jarnemo and Liberg 2005, Jenkins and Barten 2005, Olson *et al.* 2005, Beier *et al.* 2006, Duncan and Holland 2006,

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Schwartz *et al.* 2006, Sepulveda *et al.* 2006). Perinatal mortality trends and patterns from data collected through the Florida Manatee Carcass Recovery Program could potentially assist in decisions about mitigating ongoing threats to this endangered species. However, in order to do so, all collected carcasses need to first be identified as perinatal or nonperinatal.

Since its inception in the mid 1970s, the Florida Manatee Carcass Recovery Program has collected and necropsied almost every reported manatee carcass (with the small exception of carcasses that were reported but lost before recovery). Technicians have recorded extensive physiological data for over 5,800 carcasses from 1974 through 2005 in the form of necropsy reports, including carcasses necropsied in Puerto Rico. However, the reports only contain data to determine perinatal status for a fraction of the carcasses small enough to have possibly been perinatal. Where status is not determined, either technicians did not record the data, or decomposition had removed any indicators of perinatal status (Schwarz 2004).

This paper develops methods to identify perinatal status for as many manatee carcasses as possible, either through direct physiological characteristics or models based on other predictors. First, I define the physiological indicators used to identify near-birth mortality based on features described in necropsy protocol documentation (Bonde *et al.* 1983). Second, physiological limitations almost certainly play a role in the possible lengths of perinatal and nonperinatal mortalities. Very small animals are usually premature and are unable to survive long outside the womb. Thus, they would never survive long enough to exhibit nonperinatal characteristics. On the other end of the spectrum, adult female size almost certainly limits the maximum length of newborns. So, carcasses above a certain length will have always lived at least through the perinatal phase. Therefore, after review of necropsy data to identify perinatal and nonperinatal carcasses, some assignments of perinatal status are developed based on reported minimum nonperinatal and maximum perinatal lengths. Next, I fit Bayesian models to the fraction of carcasses that were perinatal as a function of length and month of carcass recovery. When necropsy reports lacked the physiological data needed to identify perinatal status, the models and assumptions allow us to estimate the probability that a given carcass is perinatal based on length and recovery month. Last, to present examples of the utility of the models in exploring perinatal mortalities within the carcass sample, I provide a first glance at the fraction of perinatal carcasses by month, region, and year by region.

MATERIALS AND METHODS

Data and Perinatal Criteria

The Fish and Wildlife Research Institute under the Florida Fish and Wildlife Conservation Commission, a state agency, currently runs the Florida Manatee Carcass Recovery Program. They maintain all hard copies and digital versions of necropsy reports as well as an electronic database that provides some, but not all, of the necropsy information for each reported carcass since 1974. For this research, I limited the data to carcasses collected in the continental United States between 1978 and 2005. Years prior to 1978 were considered "pilot-study years" in which necropsy protocols were established. I also excluded animals that lived more than 90 d as captive rescues or lived all their lives in captivity. Given the above limitations, this study utilizes data from a total of 5,603 carcasses. To quantify the length *vs.* perinatal relationship, I

reviewed all necropsy reports for animals ≤175-cm long for information on perinatal status ($n = 1,459$).

In the past, carcasses <150 cm in length for which cause of death was not human related were given a special cause-of-death category in the carcass database: "perinatal (natural or undetermined)." The category was intended to identify animals that probably died of natural causes related to pregnancy or birth or early separation from their mothers (Bonde *et al.* 1983). However, that "perinatal" cause-of-death category makes potentially incorrect assumptions about the relationships among three variables: length, age, and cause of death. In addition, the category does not address variability in the age-length relationship. Therefore, a new definition of perinatal mortality is presented here that describes a very young life-history stage without making assumptions about cause of death.

Physiological evidence recorded in the necropsy reports allows many small carcasses to be assigned to one of two defined categories: died within about 2 wk of birth, including stillborns (perinatal) *vs.* died later in life (not perinatal). I classified a carcass as "known perinatal" if at least one of the following indicators was present in the necropsy report:

1. Consolidated or partially expanded lungs,
2. Presence of fetal folds,
3. Umbilical chord or placenta attached,
4. Meconium in the digestive system,
5. Open or partially healed umbilicus, and
6. Comments in the report state the animal was stillborn, newborn, premature, died at or shortly after birth, or was a fetus.

A carcass was classified as "known not perinatal" if at least one of the following indicators was present in the necropsy report:

1. Vegetation/ingesta/digesta in the lower intestine,
2. Epibiota such as barnacles, algae, warts, or fungus,
3. Permanent scars indicating completely healed wounds,
4. Healed broken bones,
5. Internal parasites such as nematodes or trematodes,
6. Comments in the report state the animal was not stillborn or did not die at or shortly after birth, and
7. At least two of the following indicators were reported: closed ductus arteriosis, healed umbilicus, milk or debris in the lower intestine, infection, closed urachus.

Using only the criteria above, not all necropsy reports provided enough physiological data to determine perinatal status. Either the data were not recorded during necropsy, or advanced decomposition had removed all physiological indicators. In order to determine perinatal status for such carcasses, I created models based on the data of carcasses for which perinatal status could be determined.

Models

After review of necropsy reports, I drew two initial conclusions about the relationship between length and age at death: carcasses shorter than the minimum reported nonperinatal length were assumed perinatal, and carcasses longer than the maximum reported perinatal length were considered nonperinatal. Results from the

models provide estimates of the probability that a manatee died perinatal for carcasses within the limited "length window" between those minimum and maximum lengths. I assumed all length measurements were accurate (did not account for uncertainty of length estimates), and I assumed the probability of necropsy reports containing physiological information sufficient for determining perinatal status was independent of perinatal status. Finally, I assumed carcass detection and reporting probabilities were the same for a given length regardless of perinatal status.

To determine the relationship among length, recovery month, and perinatal status, I used the carcass sample for which perinatal status could be determined solely from physiological indicators. I limited the sample to carcasses within the given length window and stratified the data by month of carcass recovery. Finally, I developed the models to predict the perinatal status of individual carcasses for which other physiological indicators were not available. I chose a Bayesian technique so the results would reflect the uncertainty in parameter estimates, unlike the traditional frequentist approach (Ellison 2004). The predictive models that incorporate such uncertainty ultimately have higher predictive power compared to frequentist analysis (Wade 2000).

Once the data were stratified by recovery month, a logistic regression was used to model the fraction of perinatal carcasses with respect to length:

$$\Pr(Y = 1) = \frac{1}{1 + e^{-(\beta_0 + \beta_1 \, length)}}, \tag{1}$$

where $Y = 1$, if an animal is perinatal and $Y = 0$, if the animal is not perinatal. Using animals of known perinatal status and length, $\Pr(Y = 1)$ is the binomial proportion of perinatal carcasses of a given length. Parameters β_0 and β_1 are unknown. The binomial nature of the data requires a likelihood function that is a multiple of the binomial pdf:

$$L = \prod_{i=1}^{k} \frac{N_i!}{(N_i - m_i)! m_i!} \alpha_i^{m_i} (1 - \alpha_i)^{N_i - m_i}, \tag{2}$$

where
  $i =$ length group
  $k =$ total number of length groups
  $\alpha_i = \Pr(Y = 1)$
  $N_i =$ total number of carcasses of known status within a length group
  $m_i =$ number of perinatal carcasses within a length group.

I gave the variables of interest, β_0 and β_1, uniform priors wide enough that posterior distributions of the parameters were not truncated at any prior boundaries ($\pi(\beta_0)$ and $\pi(\beta_1) \sim U(-100, 100)$). The wide uniform priors on β_0 and β_1 actually describe a bimodal prior distribution on each α_i with high, but equal, probabilities near zero and one. The prior is similar to a beta distribution with parameters equal to zero or 0.5, a widely accepted, noninformative prior used in binomial analyses (Gelman *et al.* 2004). Sample sizes >20 are relatively insensitive to such a prior (Van Dongen 2006). Sample sizes for this study ranged from 29 to 110, depending on month of carcass recovery (Table 1).

Table 1. Counts of carcasses by length group and month and the methods used to determine perinatal status.

Length	Month	Physiological indicators		<82 cm (assumed perinatal)	82–160 cm[b]		>160 cm (assumed not perinatal)	Unknown
		Perinatal	Not perinatal		Logistic regression (variable status)	Simple binomial (variable status)		
<82 cm	–	17	36	6				
82–160 cm	January	12	11					
	February	18	18		23			
	March	85	6		24			
	April	93	14		39			
	May	96	12		53			
	June	64	13			66		
	July	64	10			60		
	August	38	13			81		
	September	35	11			63		
	October	19	26			31		
	November	10	45			30		
	December	6	100			25		
160–175 cm	–				24		62	
>175 cm	–						4,040	
Unknown	–							104
Total		557	315	6	163	356	4,102	104

[a]Models and assumptions were used when perinatal status could not be determined by physiological indicators.
[b]Numbers in these columns represent the number of individual carcasses to which the models were applied for classification and do not represent the final status.

I used the program MTG (Metropolis within Gibbs, developed by Dr. Daniel Goodman, Montana State University) to determine posterior distributions of the unknown model variables (β_0 and β_1). MTG is a Markov chain semi-random walk simulation program that samples the joint posterior distribution, obtaining combinations of values of the parameters in frequencies proportional to their posterior probability. Posterior marginal properties (mode, mean, standard deviation, tail areas, histogram, *etc*.) for parameters of interest, as well as posterior correlations among parameters of interest, can be calculated from the posterior sample, which can be saved to a file for use in further simulations. I set my simulations for a rejection rate of about 0.7, a subsampling (thinning) of 1 in 5,000, and a burn-in period of 5,000, continuing for a subsample size of 20,000 for each inference (see Cowles and Carlin 1995). The resulting lag -1 autocorrelations were <0.08, and independent chains with different parameter starting values gave indistinguishable results. To verify convergence and stationarity within the final chains, I used the Heidelberger and Welch convergence diagnostic available from the CODA package in R using standard 10% increments and $P \leq 0.05$ (Heidelberger and Welch 1983, Plummer *et al.* 2006).

For some recovery months, length, beyond knowledge that it was in the length window for possible perinatal death, could not provide additional information about perinatal status (posterior distribution of β_1 centered on zero). In such cases, length data were pooled, and a simple Bayesian analysis of the binomial proportion of perinatal carcasses in a given month was performed. The likelihood is a binomial:

$$L = \frac{T!}{(T-s)!s!} p^s (1-p)^{T-s}, \tag{3}$$

where

> T = total number of carcasses of known status in a given month
> s = number of perinatal carcasses in a given month
> p = binomial proportion of perinatal carcasses in a given month.

Choosing a broad conjugate beta prior distribution of βeta$(1, 1)$ results in a beta posterior distribution for p (Robert 2001):

$$p \sim \beta\text{eta}(s + 1, T - s + 1). \tag{4}$$

To verify the simpler model as the more appropriate model for some months, deviance information criteria (DIC) were calculated to compare the goodness of fit of the logistic regression model with the binomial model (Gelman *et al.* 2004). Hierarchical models and more complex regression models with month as a fixed effect did not reduce the DICs by more than five for any given month, nor did such models increase posterior precision. I do not present those models here because, in this case, they added complexity with no substantive gain in information.

Application of Models

To demonstrate the use of the perinatal prediction models and assumptions, I determined the temporal and regional trends in fraction of perinatal carcasses using

carcass recovery data from 1978 to 2005 collected in the continental United States (excluding long-term captives). Regions were defined from the four subpopulations described in the ESA recovery plan for Florida manatees: Northwest, Southwest, Atlantic, and Upper St. John's River (U. S. Fish and Wildlife Service 2001). Although no one has yet quantified the annual timing of births for Florida manatees, informal compilations of evidence and studies on the reproductive biology of Florida manatees suggest full-term births occur from March through November (Hernandez *et al.* 1995, Marmontel 1995, O'Shea and Hartley 1995, Odell *et al.* 1995). Because the perinatal mortalities can indicate annual variability in survival and reproductive rates, I defined a year as 1 March to the last day of February, starting the year cycle at the beginning of the birthing phase. All carcasses of known perinatal status were tallied accordingly. Each carcass for which perinatal status was unknown was assigned a probability of being perinatal according to the model, and then the probabilities of aggregate numbers of perinatals were calculated by numerical integration, assuming any uncertainty in perinatal status was independent between individuals.

RESULTS

Applying Criteria to the Data

Of the 1,459 necropsy reports reviewed from 1978 to 2005, I could identify perinatal status of 872 carcasses based directly on the defined criteria (Table 1). Five hundred fifty-seven were classified as perinatal, and 315 were categorized as nonperinatal. During necropsy, technicians recorded more than one physiological indicator in the necropsy reports for 62% of the carcasses defined as perinatal and 45% of the nonperinatal carcasses. Of the carcasses with only one reported indicator, meconium was reported for 43% of the perinatal carcasses, and vegetation in the lower intestine was reported for 67% of the nonperinatal carcasses.

Of the remaining carcasses ≤175-cm long ($n = 587$), 27 of the necropsy reports contained contradictory data. No particular contradictory indicators were consistently recorded together, meaning the contradictions were probably due to misidentification of indicators rather than inappropriate criteria to define perinatal status. Given the small percentage of inconsistent cases, misidentification of indicators is probably infrequent and would affect the counts of perinatal and nonperinatal carcasses equally. I did not use such inconsistent cases in the model analyses. I instead grouped them with other carcasses of unknown perinatal status. For the remaining carcasses, necropsy reports did not contain the data needed to identify perinatal status ($n = 282$), or advanced decomposition prevented determination of perinatal status ($n = 278$).

The longest reported perinatal carcass was 160-cm long, and the shortest reported nonperinatal carcass was 82-cm long (Table 1). Looking at the subsample of carcasses of known perinatal status 82–160 cm long, perinatal carcasses were 122.8 (± 16.4 SD) cm long, and nonperinatal carcasses were 137 (± 15.0 SD) cm long. Given those length distributions, the 82-cm long nonperinatal carcass represents an outlier and is probably the minimum possible length for survival past the perinatal stage. Similarly, a 160-cm long perinatal carcass represents the maximum possible length for a perinatal carcass. Based on the assumption that carcasses <82-cm long are perinatal, and carcasses >160-cm long are nonperinatal, I could determine perinatal status for another 4,108 carcasses. All but six of those carcasses were classified nonperinatal (Table 1).
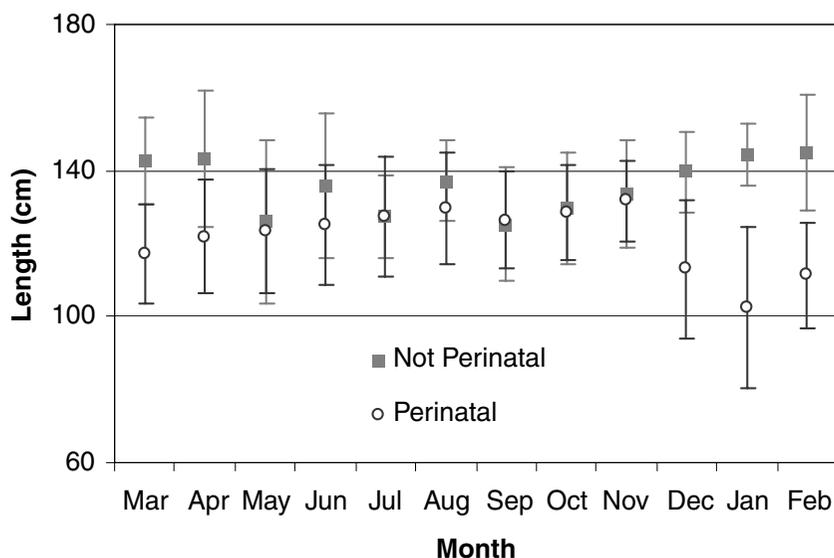
Figure 1. Length of perinatal and nonperinatal carcasses by recovery month using all data for which perinatal status is known. Carcasses were limited to the range 81–160 cm. Points are means, and bars represent one standard deviation.

Models

Using the carcasses for which perinatal status could be determined from physiological indictors, the lengths and counts of perinatal and nonperinatal carcasses were seasonal within the 82–160 cm window ($n = 755$, Fig. 1 and Table 1). For the months May through November, lengths did not differ between perinatal and nonperinatal carcasses. Perinatal carcasses were shorter whereas nonperinatal carcasses were slightly longer in December through April compared to other months. Counts of perinatal carcasses were highest in March through May whereas counts of nonperinatal carcasses in the selected size window are highest in November through January.

Final marginal posterior distributions of β_0 and β_1 were relatively normal and strongly negatively correlated (Table 2). The Bayesian logistic regression results indicate length provides additional information about perinatal status for months December through April ($\beta_1 \neq 0$). In general, perinatal probability increases for any given length as we move from December to April (Fig. 2). Inclusion of length data to predict perinatal status reduces the DIC between 19% and 63% compared to the model that excludes length. For months May through November, posterior distributions of β_1 center on zero, and length as a predictor does not reduce the DICs. Therefore, the probability an animal is perinatal when recovered in May through November can be simply estimated as the binomial proportion of perinatal carcasses 82–160 cm long in that month.

Application of Models

The models allow us to estimate perinatal status for an additional 519 carcasses 82–160 cm long: 163 using the logistic regression model for carcasses collected in

Table 2. Posterior distribution statistics by month for logistic regression parameters β_0 and β_1 modeling length *vs.* probability a carcass 81–160 cm long is perinatal, compared to a binomial model that excludes length as a predictive parameter.

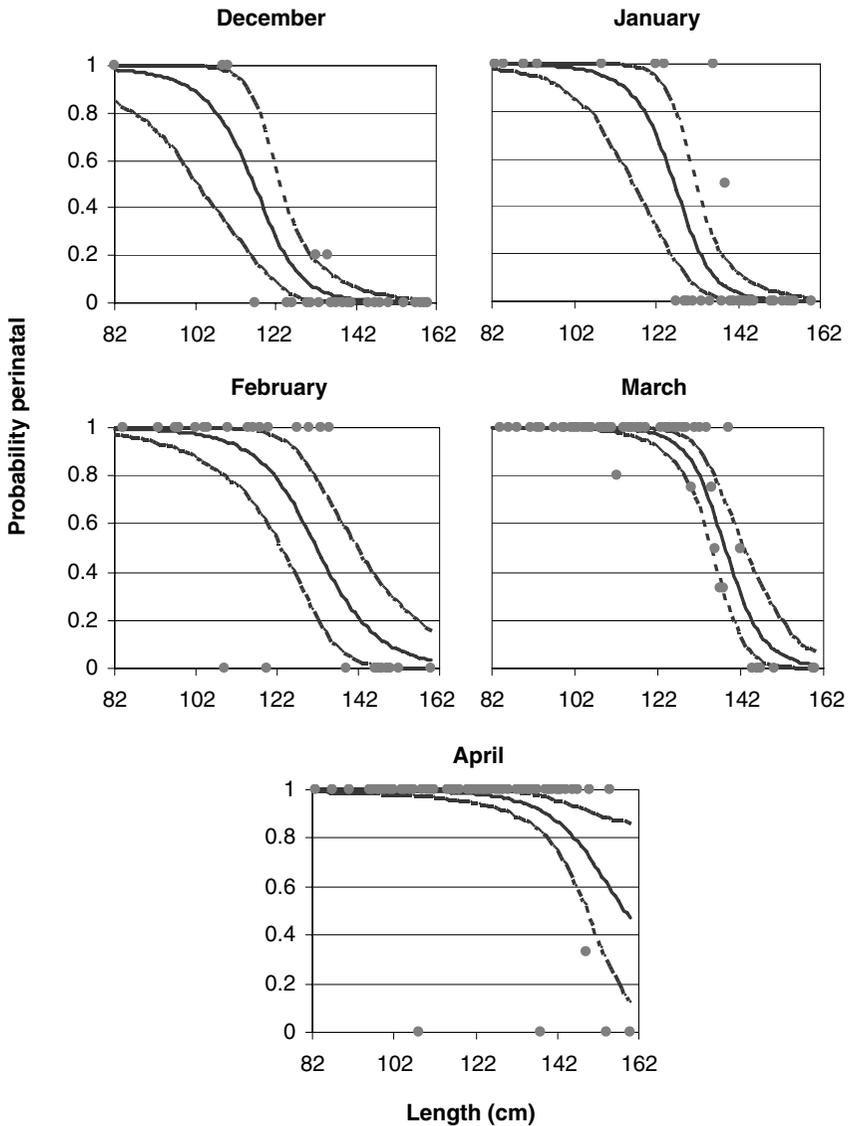| Month | Logistic regression | | | | | | | | Simple binomial | | |
| | β_0 | | | β_1 | | | $\beta_0 - \beta_1$ Correlation | DIC | $s+1$ | $T-s+1$ | DIC |
	Mode	Mean	SD	Mode	Mean	SD					
January	26.4	30.0	9.3	−0.21	−0.24	0.07	−0.997	20.8	13	37	56.0
February	16.3	19.5	6.3	−0.12	−0.15	0.05	−0.995	23.0	19	12	42.4
March	30.6	31.9	6.4	−0.22	−0.23	0.05	−0.998	48.4	86	19	97.4
April	17.1	19.0	6.1	−0.11	−0.12	0.04	−0.997	38.5	94	7	47.3
May	3.1	3.2	2.2	−0.009	−0.010	0.02	−0.990	87.4	97	15	85.8
June	6.9	7.4	2.9	−0.041	−0.044	0.02	−0.994	69.5	65	13	71.9
July	1.7	1.8	2.6	−0.0007	−0.0013	0.02	−0.993	73.9	65	14	71.9
August	6.8	7.6	4.1	−0.042	−0.047	0.03	−0.996	53.6	39	11	54.2
September	−1.6	−1.6	2.8	0.019	0.019	0.02	−0.994	66.9	36	14	65.7
October	1.1	1.2	3.8	−0.0040	−0.0046	0.03	−0.995	43.6	20	12	41.3
November	0.4	0.4	3.9	−0.010	−0.011	0.03	−0.995	46.7	11	27	44.5
December	19.1	22.4	8.0	−0.16	−0.19	0.06	−0.996	23.8	7	46	39.0

Figure 2. Logistic regression analysis on length *vs.* binomial proportion perinatal by month for months December through April. Points are fraction perinatal by length using carcasses of known perinatal status. Solid lines represent means of logistic regression curves using the posterior samples of β_0 and β_1. Dashed lines are lower and upper 95% posterior intervals.

December–April, and 356 using the binomial model for carcasses collected in May–November (Table 1). In all, perinatal status of 40% of all carcasses ≤160-cm long needed to be estimated from the models. Perinatal status could not be determined for 104 remaining carcasses (1.8% of the total sample) because length and physiological indicators both were not recorded for those animals.
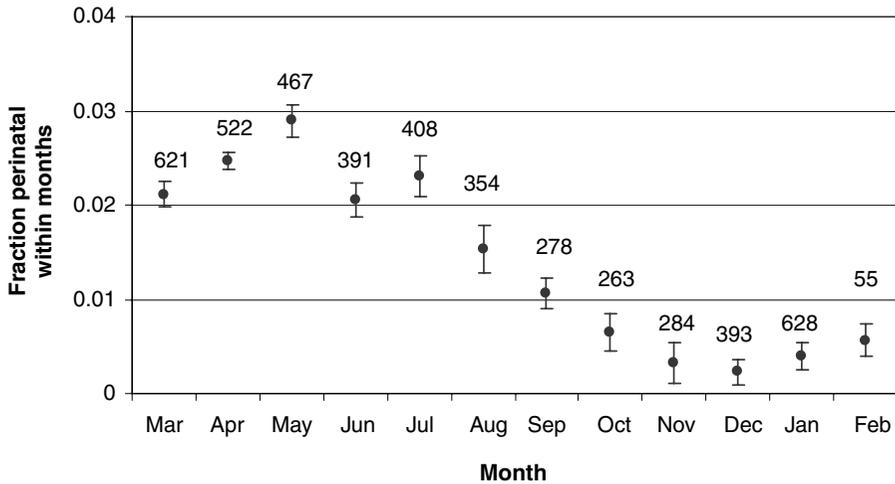
Figure 3. Fraction perinatal by month using the models to estimate perinatal status of animals 81–160 cm when status is unknown. Points are means. Bars represent minimum and maximum values. Numbers are total carcass counts by month, collected March 1978–March 2005.

The fraction of collected perinatal carcasses by month jumps dramatically between February and March (Fig. 3). Fractions peak in May and then gradually decline. Although the count of recovered carcasses is low in the Northwest region compared to the Atlantic and Southwest regions, the fraction of perinatal carcasses within the Northwest region is comparatively high (Fig. 4).

There were sufficient data to separate the Atlantic and Southwest regions by year and provide a first look at temporal trends. Counts in both regions have generally increased over the years (Fig. 5). In general, the annual fraction of perinatal carcasses collected in the Atlantic appears to have increased over the years, whereas fractions in the Southwest show little evidence of a temporal trend (Fig. 6). Comparing fractions with counts by year, the annual fraction of perinatal carcasses has increased with increased perinatal counts in the Atlantic, whereas annual perinatal fractions and counts in the Southwest do not appear to be correlated (Fig. 7).

DISCUSSION

Physiological Criteria

The criteria employed to define perinatal and nonperinatal carcasses in this analysis were based on data collected relatively consistently during Florida manatee necropsies over a 17-yr period. Nevertheless, lack of reporting accounted for 50% of the 560 carcasses <160-cm long for which perinatal status was undetermined from physiological indicators. Although the necropsy process has improved dramatically over the years, necropsy technicians still need to ensure they are consistently recording physiological characteristics that can identify perinatal mortalities.
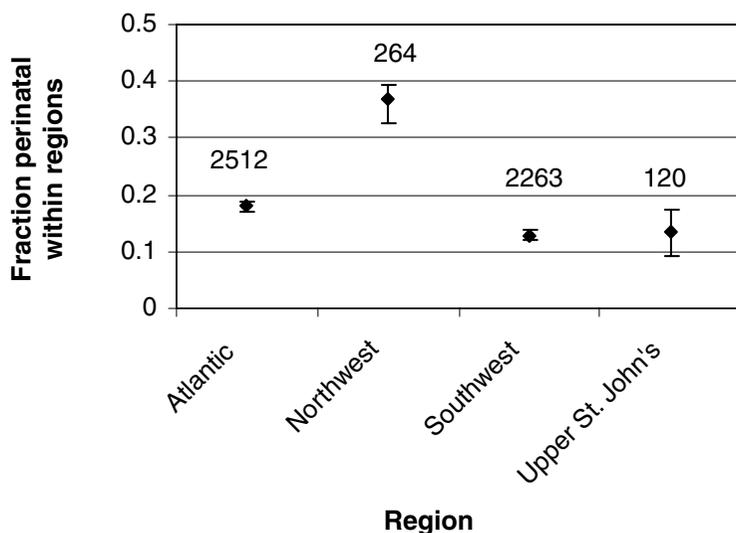
Figure 4. Fraction perinatal by region using the models to estimate perinatal status of animals 81–160 cm when status is unknown. Points are means, and bars represent minimum and maximum values. Numbers are total carcass counts by region collected March 1978– March 2005.

Additional physiological indicators could be used to determine early age at death:

1. Size of ductus arteriosis *vs.* size of pulmonary artery,
2. Open/closed foramen ovule,
3. Presence/absence of fetal hair,
4. Flexibility of skeleton,
5. Phase of healing of urachus: open, closed but probe patent, closed, median umbilical ligament; and
6. Phase of healing of umbilical region:
   a. External umbilicus: open, scabbed, completely healed; and
   b. Umbilical vessels: open, closed but probe patent, closed, medial umbilical ligament.

The age at which such physiological changes occur would have to be established, and such physiological indicators would need to be recorded consistently in necropsy reports.

Models

Manatee biology most likely limits the carcass lengths at which perinatal mortalities occur. *In utero* fetuses from the carcass recovery data provide additional support to such an assertion. Assuming a modal conception date near 1 June, fetuses start to reach 82-cm long around 6 mo after conception (Schwarz, unpublished data). With a gestation period of 12 mo, perinatal mortalities <82 cm-long could be as much as 6-mo premature, and such premature animals are probably too underdeveloped to survive past the perinatal stage. Fetus data also provide additional support for a
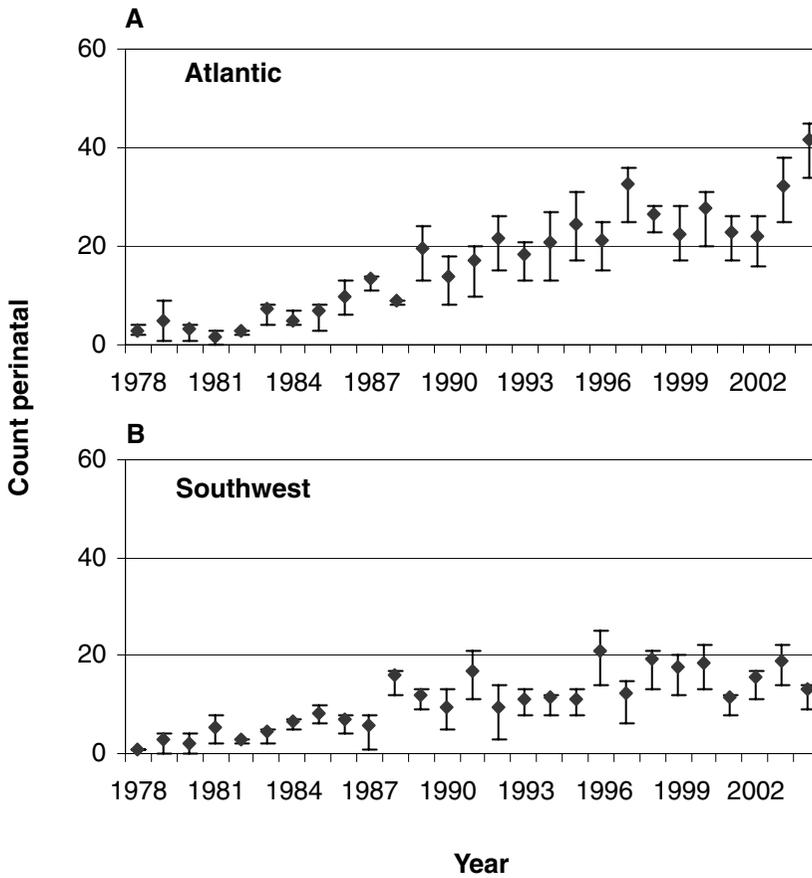
Figure 5. Counts of perinatal carcasses within the (A) Atlantic and (B) Southwest regions by year using the models to estimate perinatal status of animals 81–160 cm when status is unknown. Points are means, and bars represent minimum and maximum values. A year runs from 1 March to 1 March.

maximum length limit on perinatal mortalities. Within the carcass data set of 158 confirmed pregnancies, only four females were pregnant with fetuses >140-cm long, and three of those females died from birthing complications (Schwarz, unpublished data). Adult female size almost certainly limits the maximum length of newborns to near 160 cm. Therefore, the conclusion that perinatal status is uncertain only in the length window 82–160 cm is almost certainly biologically based.

The parameters chosen to predict perinatal status emphasize manatee physiology and biology. Length by month is an indicator of physiological growth, and month itself is connected to seasonal timing of births and deaths. Births and mating occur primarily in the spring and summer months. In addition, the definition of perinatal includes stillborns and fetuses, so the overall shorter lengths of perinatal carcasses in winter (December through February) within the 82–160 cm length window probably represent premature births. Smaller manatees are particularly sensitive to cold stress, perhaps due to a large surface-to-volume ratio and/or a lower percent fat by body
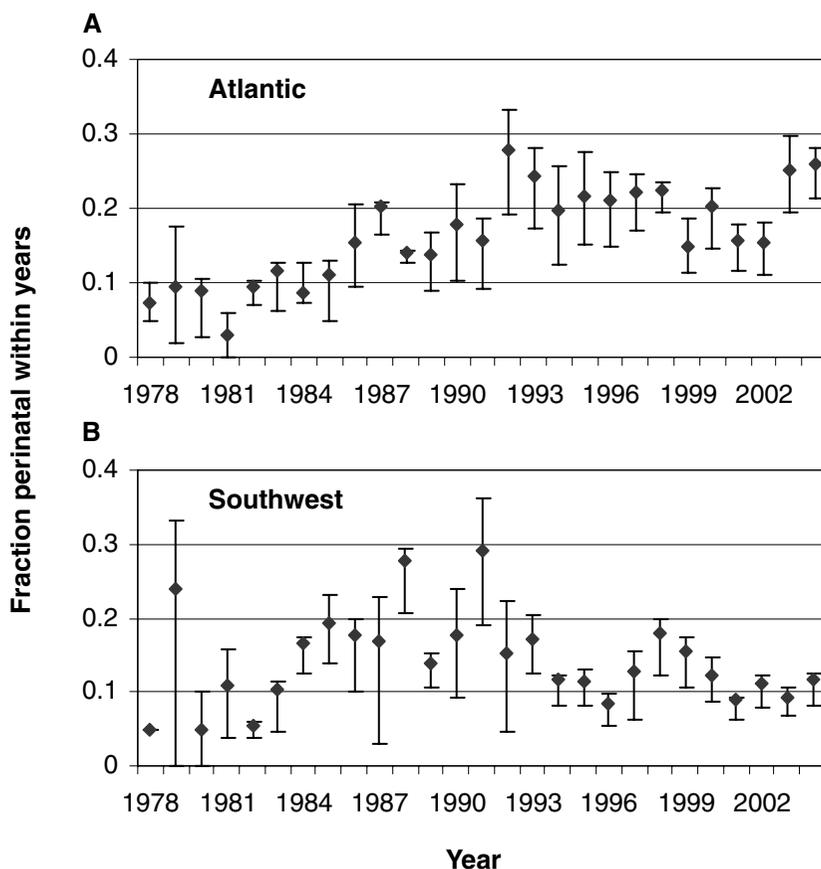
Figure 6. Fraction perinatal within the (A) Atlantic and (B) Southwest regions by year using the models to estimate perinatal status of animals 81–160 cm when status is unknown. Points are means, and bars represent minimum and maximum values. A year runs from 1 March to 1 March.

mass (Buergelt *et al.* 1984, O'Shea *et al.* 1985, Ackerman *et al.* 1995, Ortiz and Worthy 2004). Therefore, with the exception of very small animals corresponding to premature births, animals <160-cm long dying in winter may be "runts" that were born the previous summer and then experience increased mortality due to cold stress compared to larger individuals of the same age. Length is a poor indicator of perinatal death for 7 of the 9 mo when animals are most likely born, probably because of normal variability in length at birth and growth rates. Results of the models reflect the seasonal variability in both the proportion of carcasses that are perinatal and the relationship between length and time since birth within the length group of 82–160 cm.

Application of Models

Because we can now determine or probabilistically assign the perinatal status of all but 1.8% of the carcass sample, we can begin to explore trends and patterns in
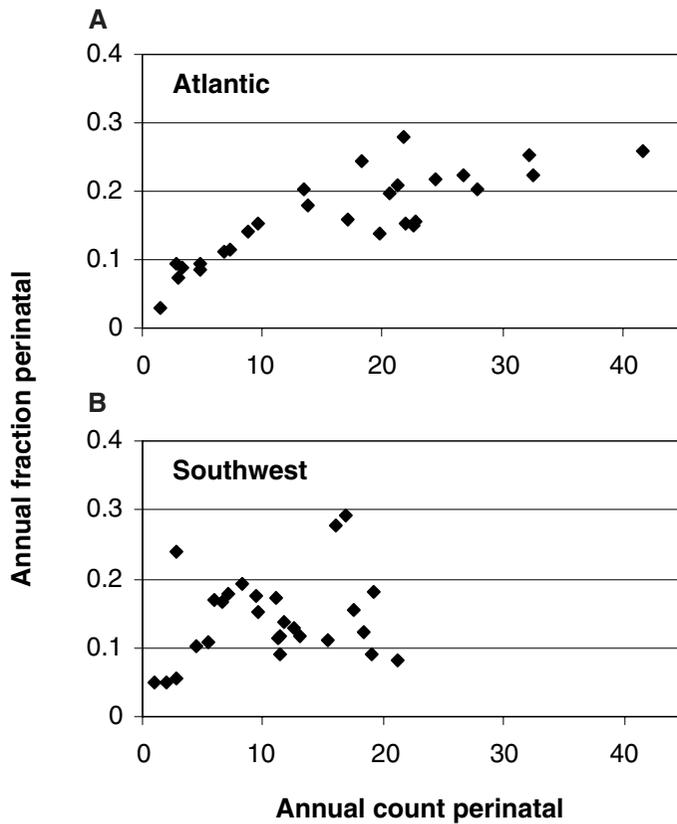
Figure 7. Relationship between annual fractions and counts of perinatal carcasses within the (A) Atlantic and (B) Southwest regions using the models to estimate perinatal status of animals 81–160 cm when status is unknown. Points are means. Error bars have been removed to show general relationship.

perinatal mortality within the carcass sample. A conclusive analysis of the apparent patterns presented here, as well as further exploration of other potential patterns, is warranted but is beyond the scope of this paper. More importantly, to utilize the perinatal data in management decisions, we would often need to determine the reasons for any detected trends or patterns that will require information beyond the scope of the necropsy data. Comparisons of both counts and fractions indicate potentially complex regional differences and trends in newborn mortality rates, pregnancy rates, population sizes, and nonnewborn mortality rates.

For example, we find a high fraction of perinatal mortalities in the Northwest compared to other regions. Adult survival rates are high, but conditional reproductive rates (which measure the probability a female produces a calf and the calf survives to winter given the female did not reproduce the previous year) are low for this region (Kendall *et al.* 2004, Langtimm *et al.* 2004). If pregnancy rates are the same by region, a low conditional reproductive rate could indicate high newborn mortality in the Northwest compared to other regions. High newborn mortality in combination

with high effective recruitment to the adult class and low adult mortality could lead to a higher fraction of perinatal carcasses.

The cause of the apparent connection between counts and fractions of perinatal carcasses by year in the Atlantic may be equally complex. Increased counts of perinatal carcasses over time could indicate increases in the population size, newborn mortality rates, and/or pregnancy rates. Increased fractions of perinatal carcasses over time could indicate a decrease in nonnewborn mortality, an increase in newborn mortality rates, and/or an increase in pregnancy rates.

Overall, the carcass recovery program relies almost solely on the public to detect and report carcasses, so new analyses exploring population-level trends will need to address potential differences or changes in sampling effort over space and time. The detection rates and overall detection probabilities of both perinatal and nonperinatal carcasses may be different between regions or may have changed over time.

Future Directions

Lastly, we need to understand the connection between such patterns and trends with environmental fluctuations, behavioral traits, and management actions. For instance, are regional differences in perinatal counts and fractions associated with resource competition, particularly access to warm water in winter? We can also now explore the association of counts and fractions of perinatal deaths with major mortality events such as severe cold weather, hurricanes, and red tides. Do such events affect perinatal mortality either directly or perhaps indirectly through lower fitness of mothers? More in-depth analyses could also explore the role of timing and severity of such events on manatee survival. With the new techniques to determine carcass perinatal status presented in this research, we can begin to explore many such questions with the carcass recovery data.

LITERATURE CITED

ACKERMAN, B. B., S. D. WRIGHT, R. K. BONDE, D. K. ODELL AND D. J. BANOWETZ. 1995. Trends and patterns in mortality of manatees in Florida, 1974–1992. Pages 223–258 *in* T. J. O'Shea, B. B. Ackerman and H. F. Percival, eds. Population biology of the Florida manatee. National Biological Service Information and Technology Report 1. Washington, DC.

BECKMEN, K. B., J. E. BLAKE, G. M. YLITALO, J. L. STOTT AND T. M. O'HARA. 2003. Organochlorine contaminant exposure and associations with hematological and humoral immune functional assays with dam age as a factor in free-ranging northern fur seal pups (*Callorhinus ursinus*). Marine Pollution Bulletin 46:594–606.

BEIER, P., M. R. VAUGHAN, M. J. CONROY AND H. QUIGLEY. 2006. Evaluating scientific inferences about the Florida panther. Journal of Wildlife Management 70:236–245.

BONDE, R. K., T. J. O'SHEA AND C. A. BECK. 1983. A manual of procedures for the salvage and necropsy of carcasses of the West Indian manatee (*Trichechus manatus*). Report from the USGS/FISC Sirenia Project, 2201 NW 40th Terrace, Gainesville, FL 32605-3574. 175 pp.

BUERGELT, C. D., R. K. BONDE, C. A. BECK AND T. J. OSHEA. 1984. Pathologic findings in manatees in Florida. Journal of the American Veterinary Medical Association 185:1331–1334.

COURTENAY, J., AND G. SANTOW. 1989. Mortality of wild and captive chimpanzees. Folia Primatologica 52:167–177.

COWLES, M. K., AND B. P. CARLIN. 1995. Markov chain Monte Carlo diagnostics: A comparative review. Journal of the American Statistical Society 91:883–904.

DUNCAN, K. M., AND K. N. HOLLAND. 2006. Habitat use, growth rates and dispersal patterns of juvenile scalloped hammerhead sharks *Sphyrna lewini* in a nursery habitat. Marine Ecology-Progress Series 312:211–221.

ELLISON, A. M. 2004. Bayesian inference in ecology. Ecology Letters 7:509–520.

ESCOS, J., AND C. L. ALADOS. 1991. Influence of weather and population characteristics of free-ranging Spanish ibex in the Sierra de Cazorlay Segura and in the eastern Sierra Nevada. Mammalia 55:67–78.

FRANK, L. G. 1997. Evolution of genital masculinization: Why do female hyaenas have such a large 'penis'? Trends in Ecology & Evolution 12:58–62.

GELMAN, A., J. B. CARLIN, H. S. STERN AND D. B. RUBIN. 2004. Bayesian data analysis. 2nd edition. Chapman and Hall/CRC, Boca Raton, FL.

GUTIERREZ, C., J. A. CORBERA, M. C. JUSTE, F. DORESTE AND I. MORALES. 2005. An outbreak of abortions and high neonatal mortality associated with *Trypanosoma evansi* infection in dromedary camels in the Canary Islands. Veterinary Parasitology 130:163–168.

HEIDELBERGER, P., AND P. D. WELCH. 1983. Simulation run length control in the presence of an initial transient. Operations Research (INFORMS) 31:1109–1144.

HERNANDEZ, P., J. E. REYNOLDS, III, H. MARSH AND M. MARMONTEL. 1995. Age and seasonality in spermatogenesis of Florida manatees. Pages 84–97 *in* T. J. O'Shea, B. B. Ackerman and H. F. Percival, eds. Population biology of the Florida manatee. National Biological Service Information and Technology Report 1, Washington, DC.

JARNEMO, A., AND O. LIBERG. 2005. Red fox removal and roe deer fawn survival—A 14-year study. Journal of Wildlife Management 69:1090–1098.

JENKINS, K. J., AND N. L. BARTEN. 2005. Demography and decline of the Mentasta caribou herd in Alaska. Canadian Journal of Zoology 83:1174–1188.

KENDALL, W. L., C. A. LANGTIMM, C. A. BECK AND M. C. RUNGE. 2004. Capture-recapture analysis for estimating manatee reproductive rates. Marine Mammal Science 20:424–437.

LANGTIMM, C. A., C. A. BECK, H. H. EDWARDS, K. J. FICK-CHILD, B. B. ACKERMAN, S. L. BARTON AND W. C. HARTLEY. 2004. Survival estimates for Florida manatees from the photo-identification of individuals. Marine Mammal Science 20:438–463.

LENT, P. C., AND W. J. DAVIS. 1993. Variables influencing survival in four generations of captive-born muskoxen. Rangifer 13:37–142.

LINKLATER, W. L., E. Z. CAMERON, E. O. MINOT AND K. J. STAFFORD. 2004. Feral horse demography and population growth in the Kaimanawa Ranges, New Zealand. Wildlife Research 31:19–128.

MARMONTEL, M. 1995. Age and reproduction in female Florida manatees. Pages 98–119 *in* T. J. O'Shea, B. B. Ackerman and H. F. Percival, eds. Population biology of the Florida manatee. National Biological Service Information and Technology Report 1, Washington, DC.

ODELL, D. K., G. D. BOSSART, M. T. LOWE AND T. D. HOPKINS. 1995. Reproduction of the West Indian manatee in captivity. Pages 192–193 *in* T. J. O'Shea, B. B. Ackerman

and H. F. Percival, eds. Population biology of the Florida manatee. National Biological Service Information and Technology Report 1, Washington, DC.

OLSON, K. A., T. K. FULLER, G. B. SCHALLER, B. LHAGVASUREN AND D. ODONKHUU. 2005. Reproduction, neonatal weights, and first-year survival of Mongolian gazelles (*Procapra gutturosa*). Journal of Zoology 265:227–233.

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.

ORTIZ, R. M., AND G. A. J. WORTHY. 2004. Could lower body fat mass contribute to cold-water susceptibility in calves of the West Indian manatee (*Trichechus manatus*)? Marine Mammal Science 20:176–183.

O'SHEA, T. J., AND W. C. HARTLEY. 1995. Reproduction and early-age survival of manatees at Blue Spring, Upper St. John's River, Florida. Pages 157–170 *in* T. J. O'Shea, B. B. Ackerman and H. F. Percival, eds. Population biology of the Florida manatee. National Biological Service Information and Technology Report 1, Washington, DC.

O'SHEA, T. J., C. A. BECK, R. K. BONDE, H. I. KOCHMAN AND D. K. ODELL. 1985. An analysis of manatee mortality patterns in Florida, 1976–81. Journal of Wildlife Management 49:1–11.

PLUMMER, M., N. BEST, K. COWLES AND K. VINES. 2006. CODA: Convergence diagnosis and output analysis for MCMC. R News 6:7–11.

POJAR, T. M., AND D. C. BOWDEN. 2004. Neonatal mule deer fawn survival in west-central Colorado. Journal of Wildlife Management 68:550–560.

ROBERT, C. P. 2001. The Bayesian choice. Springer Texts, New York, NY.

ROTHE, H., K. DARMS AND A. KOENIG. 1992. Sex-ratio and mortality in a laboratory colony of the common marmoset (*Callithrix jacchus*). Laboratory Animals 26:88–99.

SCHWARTZ, C. C., M. A. HAROLDSON, G. C. WHITE, R. B. HARRIS, S. CHERRY, K. A. KEATING, D. MOODY AND C. SERVHEEN. 2006. Temporal, spatial, and environmental influences on the demographics of grizzly bears in the Greater Yellowstone Ecosystem. Wildlife Monographs:1–68.

SCHWARZ, L. K. 2004. Report on the review of Florida manatee necropsy data and restructuring of the carcass recovery database. Report presented to the United States Marine Mammal Commission, 4340 East West Highway, Suite 905, Bethesda, MD 20814. 55 pp.

SEPULVEDA, M. S., F. DEL PIERO, J. J. WIEBE, H. R. RAUSCHENBERGER AND T. S. GROSS. 2006. Necropsy findings in American alligator late-stage embryos and hatchlings from north central Florida lakes contaminated with organochlorine pesticides. Journal of Wildlife Diseases 42:56–73.

U. S. FISH AND WILDLIFE SERVICE. 2001. Florida manatee recovery plan, (*Trichechus manatus latirostris*), third revision. U. S. Fish and Wildlife Service, Atlanta, GA.

VAN DONGEN, S. 2006. Prior specification in Bayesian statistics: Three cautionary tales. Journal of Theoretical Biology 242:90–100.

WADE, P. R. 2000. Bayesian methods in conservation biology. Conservation Biology 14:1308–1316.