Alaska
Fisheries Science
Center

National Marine
Fisheries Service

U.S DEPARTMENT OF COMMERCE

AFSC PROCESSED REPORT 2008-02

2008 Status Review and Extinction Risk Assessment of Cook Inlet Belugas (*Delphinapterus leucas*)

April 2008

This document should be cited as follows:

Hobbs, R. C., K. E. W. Shelden, D. J. Rugh, and S. A. Norman. 2008. 2008 status review and extinction risk assessment of Cook Inlet belugas (*Delphinapterus leucas)*. AFSC Processed Rep. 2008-02, 116 p. Alaska Fish. Sci. Cent., NOAA, Natl. Mar. Fish. Serv., 7600 Sand Point Way NE, Seattle WA 98115.

2008 STATUS REVIEW AND EXTINCTION RISK ASSESSMENT OF COOK INLET BELUGAS (*DELPHINAPTERUS LEUCAS*)

By:

Roderick C. Hobbs, Kim E. W. Shelden, David J. Rugh, and Stephanie A. Norman

With contributions from:

R. Angliss, K. Brix, K. Burek, K. Goetz, B. Mahoney, G. O’Corry-Crowe, C. Sims, B. Smith, and D. Vos

National Oceanic and Atmospheric Administration
National Marine Fisheries Service
Alaska Fisheries Science Center
National Marine Mammal Laboratory
7600 Sand Point Way N.E.
Seattle, WA 98115-6349

April 2008

Cover photo: Belugas (*Delphinapterus leucas*) in the Susitna River delta, Cook Inlet, Alaska, June 1994. Photographer: J. Waite (AFSC-NMML).

CONTENTS

EXECUTIVE SUMMARY

Background

After completion (in 2005) of the Draft Conservation Plan for Cook Inlet belugas (*Delphinapterus leucas*) under the Marine Mammal Protection Act (MMPA); the National Marine Fisheries Service (NMFS) recommended that a Status Review be conducted to incorporate new scientific findings available since the publication of a scientific review in 2000 in the journal *Marine Fisheries Review* 62 (3). NMFS formally initiated this Status Review on March 29, 2006 to determine if Cook Inlet belugas should be listed under the U.S. Endangered Species Act (ESA). On April 20, 2006, NMFS received a petition from Trustees for Alaska to list Cook Inlet belugas as endangered under the ESA. After reviewing the information contained in the petition, as well as other scientific information readily available, NMFS determined the petitioned action may be warranted. Within 12 months of the date of the petition, NMFS was required to make one of the following findings:

1) the petitioned action is not warranted;
2) the petitioned action is warranted and the Secretary of Commerce will publish in the *Federal Register* (FR) a proposed regulation to implement the action pursuant to 50 CFR 424.16; or
3) the petitioned action is warranted, but
    A) the immediate proposal and timely promulgation of a regulation to implement the petitioned action is precluded because of other pending proposals to list, delist, or reclassify species; and
    B) expeditious progress is being made to list, delist, or reclassify qualified species, in which case such findings shall be promptly published in the FR.

The Status Review published in November 2006 provided a summary of the best available science to aid NMFS managers in this process. Based on the findings from the Status Review and consideration of the factors affecting this species, NMFS concluded Cook Inlet belugas constituted a distinct population segment (DPS) that was in danger of extinction throughout its range. NMFS issued a proposed rule to list the Cook Inlet beluga DPS as an endangered species on April 20, 2007. Since completion of the 2006 Status Review and extinction assessment of Cook Inlet belugas, NMFS has completed analyses of the 2006 and 2007 aerial survey data and generated abundance estimates for those years. In November 2007, NMFS initiated a review of the science presented in the 2006 Status Review by a panel of independent experts through the Center for Independent Experts (CIE). This *AFSC Processed Report* includes these findings and an update of the best available science obtained since publication of the 2006 Status Review in response to the CIE review and public comments.

Status of Cook Inlet Belugas

Temporal Changes in Distribution

Since the mid-1990s, 96% to 100% of the observed Cook Inlet belugas have congregated in the upper Inlet in shallow areas near river mouths—they were only occasionally found in the central or southern portions of the Inlet during the summer months. It is unknown if this contracted distribution is a result of changing habitat, prey concentration, predator avoidance, or a more acute reduction of the population into all but a small number of preferred habitat areas. This concentration of belugas in the northernmost portion of Cook Inlet appears to be a fairly consistent pattern from June to October. Data from tagged whales (14 tags between July and March 2000-03) show that belugas use the upper Inlet intensively between summer and late autumn, but during winter months they also disperse to mid-Inlet offshore waters. Tagged whales and extensive surveys both within Cook Inlet and in the Gulf of Alaska indicate that belugas do not have a seasonal migration in and out of the Inlet. Yakutat Bay is the only location outside of Cook Inlet where there is a known, persistent population of several belugas. It is not clear from available data whether this group is isolated from Cook Inlet.

Population Size and Trend

NMFS began comprehensive, systematic aerial surveys of the beluga population in Cook Inlet in 1993. Unlike previous efforts, these surveys included the upper, middle, and lower sections of the Inlet. These surveys documented a decline in abundance of nearly 50% between 1994 and

1998, from an estimate of 653 whales to 347 whales. Although this rapid decline stopped after hunting was regulated in 1998, beluga numbers have not increased.

Data analyses indicated that the documented decline in beluga abundance from 1994 to 1998 is adequately explained by the estimated mortalities from the Native subsistence hunt for the same period. With the very limited hunt between 1999 and 2007 (0 to 2 whales per year), NMFS anticipated that the population would begin to recover at a growth rate of 2% to 6% per year. However, a Bayesian analysis including the 2007 estimate of abundance indicates that there is a probability of less than 4% that the growth rate is above 2%, and a probability of 77% or more that the population will decline further. The best available data at this time indicate that the Cook Inlet beluga population is not growing as expected despite the limits on subsistence hunting.

Determination of Distinct Population Segment

NMFS established Cook Inlet belugas as a distinct population segment (DPS) and therefore, a species as defined under Section 3(15) of the ESA on June 22, 2000. At the time, the Cook Inlet stock had been designated as depleted under the MMPA (May 31, 2000) and included all belugas in waters of the Gulf of Alaska north of 58º N latitude (including, but not limited to, Cook Inlet, Kamishak Bay, Chinitna Bay, Tuxedni Bay, Prince William Sound, Yakutat Bay, Shelikof Strait, and off Kodiak Island and freshwater tributaries to these waters). The population of belugas in Cook Inlet is discrete from other Alaskan and Russian beluga populations in the Arctic. Physically, these whales are isolated from other populations by the Alaska Peninsula. Despite extensive, dedicated marine mammal survey effort, the lack of sightings along the southern side of the Alaska Peninsula and Aleutian Islands chain suggests that the Cook Inlet population does not disperse into the Bering Sea. Behaviorally, belugas show strong maternally-driven site-fidelity to summering areas, suggesting opportunity for intermixing may only occur during winter migrations. However, the available data suggest that belugas remain in Cook Inlet year-round and do not undertake extensive migrations. Furthermore, the genetic characteristics of this population differ markedly from the other four beluga populations that occur off western and northern Alaska. Given the site-fidelity of beluga populations, it is unlikely that immigrants from other Arctic beluga populations would repopulate Cook Inlet in the foreseeable future if the Cook Inlet beluga population goes extinct. As it is the only population found in subarctic waters east of the Alaska Peninsula, the result would be a significant loss in the range of the taxon.

Risk Assessment

Risk Factors

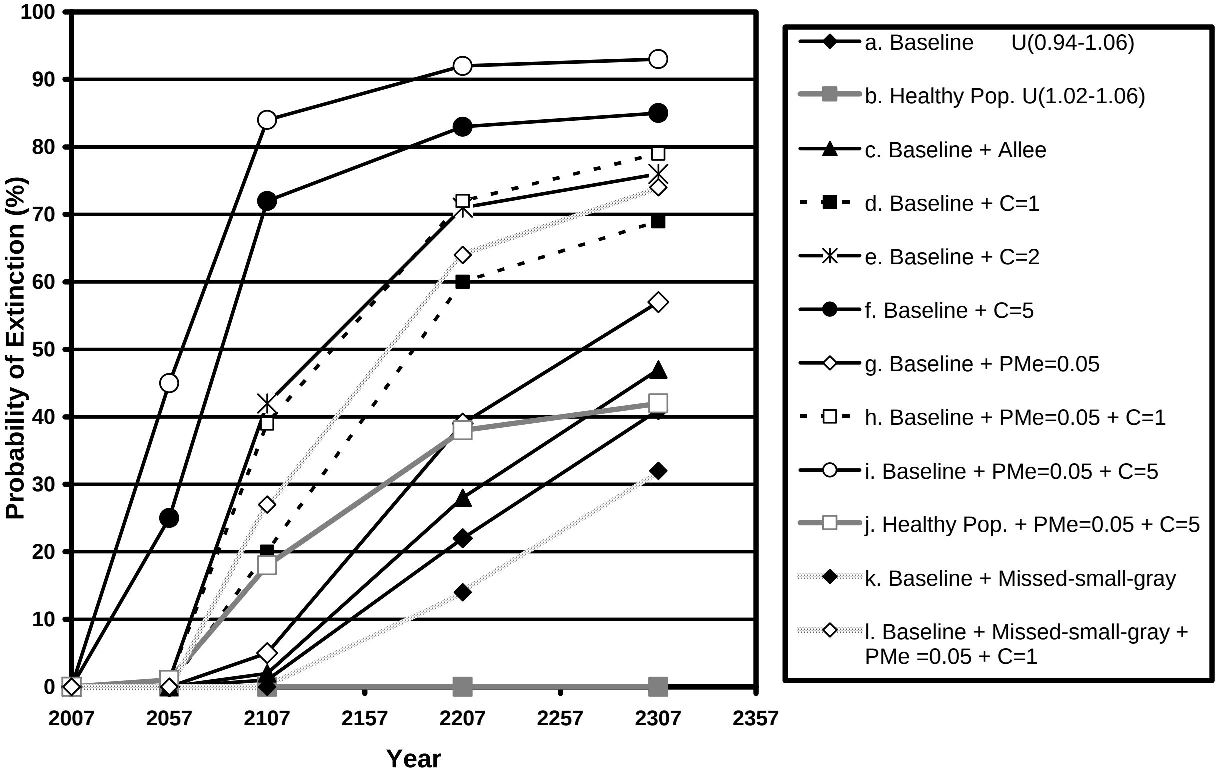
The ESA defines an endangered species as any species in danger of extinction throughout all or a significant portion of its range, and a threatened species as any species likely to become endangered within the foreseeable future. Section 4(b)(1)(a) of the ESA requires that determinations of whether a species is threatened or endangered be based solely on the best scientific and commercial data available, after taking into account those efforts, if any, being made to protect the species. The Secretary shall determine whether any species is endangered or threatened because of any of the following factors listed under Section 4(a)(1) of the ESA:

A) The present or threatened destruction, modification, or curtailment of habitat or range;
B) Overutilization for commercial, recreational, scientific, or educational purposes;
C) Disease or predation;
D) The inadequacy of existing regulatory mechanisms; or
E) Other natural or manmade factors affecting its continued existence.

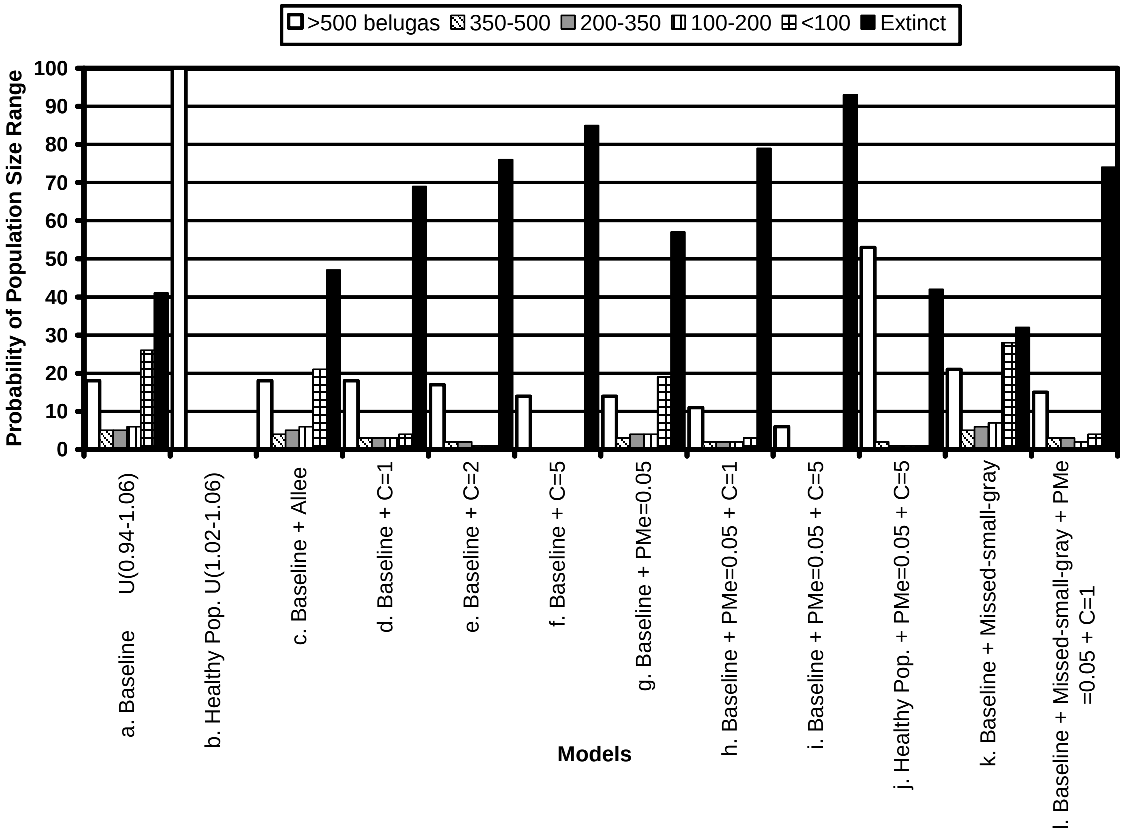
There are a number of behavioral and ecological characteristics that put Cook Inlet belugas at considerable risk of extinction. These include but are not limited to the following: 1) life history characteristics such as slow population growth rate; 2) distorted age, size or stage structure of the population, and reduced reproductive success; 3) strong depensatory or Allee effects; 4) habitat specificity or site fidelity; and 5) habitat sensitivity. The genetic and spatial isolation of the Cook Inlet beluga population and strong site-fidelity greatly increases the risk of inbreeding and expression of deleterious genes should this population decline further in number. At reduced numbers and with contraction of their range, this population is far more vulnerable to losses due to stranding, predation, or disease. Cook Inlet belugas rely heavily on several fish prey species that are available only seasonally and are also of considerable commercial interest. Disturbances that cause belugas to temporarily or permanently abandon summer feeding areas could reduce their ability to survive through the winter months.

Population Viability Analysis

A detailed population viability analysis model, including immature and mature stages of both sexes, developed for the Cook Inlet beluga population viability analysis (PVA) in the 2006 Status Review was rerun and expanded to include new data from 2006 and 2007 and address issues raised during the review process, in particular the possibility that small, gray calves and juveniles are undercounted in aerial surveys. This model focused on the behavior of a declining population at sizes less than 500 belugas. Small population effects, demographic stochasticity, Allee effects, predation mortality, and unusual mortality events were modeled explicitly. The modeled Allee effect and predation mortality produced thresholds of population size below which the population could not recover; extinction occurred more or less rapidly depending on the height of the population size threshold. This threshold was particularly pronounced when predation (*C*) was set at two mortalities or greater per year causing a visible break point below which there was little likelihood of the population avoiding extinction. The probability of extinction within 100 years ranged from 1% to 42%, and within 300 years ranged from 41% to 79% in the models that were considered to have parameters most representative of the Cook Inlet beluga population (ES-Fig.1, models a, c-e, g-h). What was thought to be the most realistic model (ES-Fig. 1, model h), with an average of one predation mortality per year and a 5% annual probability of an unusual mortality event killing 20% of the population, resulted in a 1% probability of extinction in 50 years, 39% probability of extinction in 100 years and 79% probability of extinction in 300 years. Models with five predation mortalities per year (ES-Fig. 1, models f, i, j) showed that the extinction probability was sensitive to changes or underestimation of this parameter and that the population at its current size of 375 would be near the threshold population size (200 animals) for this model, even if the population was otherwise healthy but suffered occasional unusual mortality events. The model with no threshold effects (i.e., Allee or predation) resulted in a 77% probability of decline and 41% probability of extinction within 300 years (ES-Fig. 1, model a). Even with this most optimistic scenario, with no harvest after 2007, the probability that the population would be larger than 500 animals in 2307 was only 18% (ES-Fig. 2, model a). The model with no threshold effects but with over half of the small gray animals under the age of 10 years being missed in the survey, resulted in a 32% probability of extinction in 300 years (ES-Fig. 1, model k) and this same model with an average of one predation mortality per year and a 5% annual probability of an unusual mortality event killing 20% of the population, resulted in a 27% probability of extinction in 100 years and 74% probability of extinction in 300 years (ES-Fig. 1, model k). These results from variations of the model with some small gray animals being missed are comparable to the equivalent models (ES-Fig. 1, models a, h) where it is assumed that all animals are accounted for by corrections during the analysis, indicating that the model results are robust in regards to this assumption.



ES-Figure 1. Probability of extinction by year for the Cook Inlet beluga population resulting from each population viability analysis model. Models using the same parameters are the same line style, color, and symbol type with open symbols indicating the inclusion of the unusual mortality event parameter P_{Me} set at a 5% annual probability of a 20% mortality. The constant mortality effect parameter (C) was set at 1, 2 or 5 whales per year. U = uniform distribution (of the annual growth multiplier). The Baseline model allowed declining and increasing annual growth while the Healthy Population model allowed only increasing annual growth. The Missed-small-gray allowed over half of the animals under the age of 10 years to be missed during each survey.



ES-Figure 2. Probability of the Cook Inlet beluga population size resulting from population viability analysis outcomes after 300 years. Note that in all cases the majority of outcomes are either extinct or > 500 animals. The Baseline Model allowed declining and increasing annual growth while the Healthy Population model allowed only increasing annual growth. U = uniform distribution (of the growth multiplier), C = constant mortality effect parameter (e.g., predation) set at 1, 2, or 5 belugas, P_{Me} = unusual mortality event parameter set at 5% annual probability of 20% mortality. The missed-small-gray allowed over half of the animals under the age of 10 years to be missed during each survey.

Conclusions of the Status Review

- The contraction of the range of this population northward into the upper Inlet makes it far more vulnerable to catastrophic events which have the potential to kill a significant fraction of the population.
- The population is not growing at 2% to 6% per year as had been anticipated since the cessation of unregulated hunting.
- The population is discrete and unique with respect to the species, and if it should fail to survive, it is highly unlikely that Cook Inlet would be repopulated with belugas. This would result in a permanent loss of a significant portion of their range.
- The importance of seasonal anadromous fish runs in Cook Inlet to belugas is evident. The bulk of their annual nutrition is acquired during the summer months.
- Belugas in Cook Inlet are unique in Alaska given their summer habitat is in close proximity to the largest urban area in the state.
- While the impact of disease and parasitism on this population has not been quantified, this population is at greater risk because of its small size and limited range such that a novel disease would spread easily through this population.
- The PVA shows a 39% probability of extinction in 100 years and 79% probability of extinction in 300 years (for the model assuming one predation mortality per year and a 5% annual probability of an unusual mortality event killing 20% of the population). It is likely that the Cook Inlet beluga population will continue to decline or go extinct over the next 300 years unless factors determining its growth and survival are altered in its favor.

1. INTRODUCTION

1.1. Scope and Intent of the Status Review Update

Following the 2006 review of the status of the beluga (*Delphinapterus leucas*) population in Cook Inlet, Alaska (Hobbs et al. 2006), the National Marine Fisheries Service (NMFS) proposed listing the Cook Inlet beluga population as an endangered distinct population segment (DPS) under the U.S. Endangered Species Act (ESA) on April 20, 2007. NMFS continued to gather available scientific data and complete the 2006 and 2007 abundance estimates. In November 2007, NMFS hosted an independent review of the science presented in the 2006 Status Review supplemented with information available before October 2007, by a panel of experts through the Center for Independent Experts (CIE). This *AFSC Processed Report* includes a review of data that have become available since the 2006 review, the findings of the CIE review, and updated models of extinction scenarios for the Cook Inlet beluga DPS. The Introduction is reproduced here as it appears in the 2006 assessment (Hobbs et al. 2006), with the addition of actions that occurred in 2007.

1.2. History of the Status of Cook Inlet Belugas

1.2.1. Candidate Species Listing—1988

Status Reviews are prepared by NMFS for marine species that are being considered for listing as a "Species of Concern" (69 FR 19975, April 15, 2004), Candidate Species (50 CFR 424.02), or that are already listed as endangered or threatened under the ESA (5 USC 1533). On August 31, 1988, NMFS announced the creation of a list of Candidate Species being considered by the Secretary of Commerce (NMFS is an agency within the Department of Commerce) for listing as threatened or endangered species under the ESA. A Candidate Species is a species that the U.S. Fish and Wildlife Service (USFWS) or NMFS is considering listing as endangered or threatened but which has not yet been the subject of a proposed rule. Candidate Species are afforded no protection under the ESA, but § 4(b)(3)(C)(iii) of the Act requires the agencies to monitor the status of certain candidate taxa “to prevent their extinction while awaiting listing” (58 FR 51146, September 30,1993).

Belugas found in Cook Inlet, Alaska, and infrequently in waters east of the Alaska Peninsula (Laidre et al. 2000), were included on the 1988 List of Candidate Vertebrate and Invertebrate Marine Species (53 FR 33516, August 31, 1988). The decision to list Cook Inlet belugas as a Candidate Species was based on information summarized in a species account (Hazard 1988) that was part of a larger compendium on selected marine mammal species in Alaska published in early 1988 (Lentfer 1988). This compendium was distributed to the NMFS and USFWS where it was used to develop or update research and management plans for species under their jurisdiction (MMC 1989:173). At the time, the limited available research suggested belugas in Cook Inlet made up a small population numbering less than 500 animals that was isolated from all other beluga populations in Alaska waters. On September 15, 1988, the NMFS office in Anchorage, Alaska (NMFS Alaska Region Office) prepared a review (Morris 1988) of all available information on Cook Inlet belugas including priorities and recommendations for research that would be needed to sustain the population at a stable level.

Cook Inlet belugas remained on the Candidate List when it was revised on June 11, 1991 (56 FR 26797). Aerial surveys were conducted on 8 and 10 June (Shelden 1994) and 18-21 June (NMFS 1992) in 1991 to determine the size of the population. The highest uncorrected count for these surveys was less than 250 animals. The status report prepared by the NMFS Alaska Region Office (NMFS 1992) again included recommendations for research to determine trends, genetic status, winter distribution and life history parameters. Abundance surveys and tissue sampling began in 1992, while other studies such as ship-based oceanographic sampling, tagging studies, and acoustic monitoring have occurred when funding allowed since 1994. When the candidate list was revised on July 14, 1997 (62 FR 37560, December 18, 1997), it was noted that Cook Inlet belugas continued to be listed and that research had been initiated as a result of the 1991 listing.

1.2.2. Status Review—1998-2002

Prompted by a sharp decline in the estimated abundance of Cook Inlet belugas between 1994 (653 animals) and 1998 (347 animals), a reduction of nearly 50% (Hobbs et al. 2000a), NMFS initiated a Status Review of the population on November 19, 1998 (63 FR 64228). The comment period on the Status Review, which began at the same time that workshops were convened to review beluga populations throughout Alaska, extended from November 19, 1998 through January 19, 1999. The workshops were held by the Alaska Beluga Whale Committee (November 16-17, 1998) and the Alaska Scientific Review Group (November 18-20, 1998), a body established under the MMPA to provide scientific advice regarding marine mammals to NMFS and the USFWS.

NMFS received two petitions in March 1999 to list Cook Inlet belugas as endangered under the ESA. One petition (brought by Joel Blatchford, a Native Alaskan beluga hunter; the Alaska Center for the Environment, the Alaska Community Action on Toxics, the Alaska Wildlife Alliance, the Center of Biological Diversity, the Center for Marine Conservation, the National Audubon Society, and the Trustees for Alaska) requested an emergency listing under Section 4(b)(7) of the ESA and the designation of critical habitat. Both petitions (the second brought by the Animal Welfare Institute) requested immediate promulgation of regulations to govern the subsistence hunt. NMFS determined that the petitions contained substantial scientific or commercial information indicating that the petitioned actions may be warranted (64 FR 17347, April 9, 1999). To ensure that the Status Review was comprehensive and based on the best available scientific information, NMFS sponsored a workshop on March 8-9, 1999 in Anchorage that reviewed relevant scientific information on this population. At this workshop, NMFS received additional public comments and recommendations. The abstracts of presentations from this workshop (Moore et al. 1999) were subsequently published in a special issue of *Marine Fisheries Review* 62(3).

1.2.3. MMPA Subsistence Hunt Management—1999-2007

In 1999, a temporary legislative moratorium on hunting Cook Inlet belugas by Native Americans was enacted (Pub. L. No. 106-31, Section 3022, 113 Stat. 57, 100, May 21, 1999). This legislation resulted in no hunt in 1999 and 2000, though hunters voluntarily suspended the hunt in spring 1999. Following the "depleted" determination under the MMPA, NMFS proposed regulations limiting the hunt of belugas in Cook Inlet, Alaska, on October 4, 2000 (65 FR 59164). While these regulations were undergoing public comment, the moratorium was made permanent in December 2000 (Pub. L. No. 106-553). The only exclusion to the moratorium is through a co-management agreement between NMFS and Alaska Native organizations (ANO). NMFS has since promulgated regulations for the taking of Cook Inlet belugas by Alaska Natives for the years 2001-2004 (69 FR 17973, April 6, 2004). A Final Environmental Impact Statement (EIS) was released with the final proposed regulations in July 2003 (68 FR 55604, September 26, 2003). Proposed long-term harvest regulations through a period which should see population recovery are currently under review (71 FR 8268, February 16, 2006) and discussed in this document (see Section 3.5). Preparation of a Supplemental EIS reviewing these long-term harvest regulations is underway (72 FR 73798, December 28, 2007).

1.2.4. NMFS MMPA Depleted Decision and ESA Not Warranted Decision—1999-2000

Following these reviews and taking into account the best information available at that time, NMFS proposed designating the Cook Inlet population of belugas as "depleted" under the MMPA on October 19, 1999 (64 FR 56298) and conducted a public hearing on November 22, 1999. NMFS issued a final rule on May 31, 2000 (65 FR 34590) designating these belugas as Depleted based on its determination that the abundance estimate was below the Optimum Sustainable Population (OSP) level. At the time, the Cook Inlet stock included all belugas in waters of the Gulf of Alaska north of 58º N latitude (including, but not limited to, Cook Inlet, Kamishak Bay, Chinitna Bay, Tuxedni Bay, Prince William Sound, Yakutat Bay, Shelikof Strait, and off Kodiak Island and freshwater tributaries to these waters). On June 22, 2000, NMFS also determined that Cook Inlet belugas were not in danger of extinction nor likely to become so in the foreseeable future. Therefore, NMFS determined that listing this population under the ESA was not warranted at the time (65 FR 38778). However, NMFS remained concerned about the status of the Cook Inlet beluga population and continued to include the population on the list of Candidate Species under the ESA. During this petition review, NMFS established Cook Inlet belugas as a DPS and therefore, a species as defined under Section 3(15) of the ESA (65 FR 121, June 22, 2000).

1.2.5. Court Challenge to ESA Not Warranted Decision—2001

The decision not to list can be challenged in court under the citizen suit provision of the ESA (16 U.S.C. § 1540(g)). In their suit (Cook Inlet Beluga, et al. v. Daley, No. 00-1017 D.C.), the petitioners argued that NMFS had acted in an "arbitrary and capricious" manner by not listing Cook Inlet belugas under the ESA. On August 20, 2001, U.S. District Court Judge James Robertson ruled that the Agency had acted within the scope of its legal authority, adequately explained its decision, based its decision on facts in the record, and considered the relevant factors and, therefore, upheld the decision not to list.

1.2.6. Species of Concern—2004

On April 15, 2004, NMFS moved Cook Inlet belugas from the Candidate Species list to the newly created Species of Concern list (64 FR 19975). This list is limited to species under NMFS jurisdiction and does not apply to the regulatory practices of the USFWS. NMFS uses the term "Species of Concern" to identify species about which NMFS has some concerns regarding status and threats but for which insufficient information is available to indicate a need to list the species under the ESA. This may include species for which NMFS has determined, following a biological Status Review, that listing under the ESA is "not warranted," pursuant to ESA Section 4(b)(3)(B)(i) but for which significant concerns or uncertainties remain regarding their status and/or threats, as is the case for Cook Inlet belugas. NMFS may conduct ESA Status Reviews on each Species of Concern as agency resources permit.

1.2.7. Conservation Plan—2005

On March 16, 2005, NMFS completed a draft Conservation Plan for Cook Inlet belugas as required under the MMPA. The comment period for the plan closed June 27, 2005 (70 FR 30697). A final version of the plan is currently under review at the NMFS Alaska Regional Office (NMFS 2005).

1.2.8. Status Review—2006

NMFS formally initiated a Status Review on March 29, 2006 (71 FR 14836) to aid NMFS managers in determining if Cook Inlet belugas should be listed under the ESA. This review, published as an *AFSC Processed Report* in November 2006 (Hobbs et al. 2006) concluded:
1) The contraction of the range of the population northward into the upper Inlet made it far more vulnerable to catastrophic events with the potential to kill a significant fraction of the population; 2) The population was not growing at 2% to 6% per year as had been anticipated since the cessation of unregulated hunting; 3) The population was discrete and unique with respect to the species, and if it should fail to survive, it was highly unlikely that Cook Inlet would be repopulated with belugas; resulting in a permanent loss of a significant portion of the range for the beluga species; 4) The importance of seasonal anadromous fish runs in Cook Inlet to belugas was evident and that the bulk of their annual nutrition was acquired during the summer months; and 5) The population viability analysis (PVA) model showed a 26% probability of extinction in 100 years and 68% probability of extinction in 300 years (for the model assuming one predation mortality per year and a 5% annual probability of an unusual mortality event killing 20% of the population). Based on the best available science at the time, the Cook Inlet beluga population was likely to continue to decline or go extinct over the next 300 years unless factors determining its growth and survival were altered in its favor.

1.2.9. Proposed Rule to List—2007

On April 20, 2006, NMFS received a petition from Trustees for Alaska to list Cook Inlet beluga as endangered under the ESA. After reviewing the information contained in the petition as well as other scientific information readily available, NMFS determined that the petition presented substantial scientific information indicating that the petitioned action may be warranted (71 FR 44614, August 7, 2006). Within 12 months of the date of the petition, NMFS must make one of the following findings: 1) The petitioned action is not warranted; 2) the petitioned action is warranted, in which case the Secretary shall promptly publish in the *Federal Register* a proposed regulation to implement the action pursuant to 50 CFR 424.16; or 3) the petitioned action is warranted, but A) the immediate proposal and timely promulgation of a regulation to implement the petitioned action is precluded because of other pending proposals to list, delist, or reclassify species, and B) expeditious progress is being made to list, delist, or reclassify qualified species, in which case such findings shall be promptly published in the *Federal Register* (71 FR 44614). Based on the findings from the Status Review and consideration of the factors affecting this species, NMFS concluded Cook Inlet belugas constituted a DPS that was in danger of extinction throughout its range. NMFS issued a proposed rule to list the Cook Inlet beluga DPS as an endangered species on April 20, 2007 (72 FR 19854). Public hearings and public comments on the proposed listing were held through August 3, 2007.

1.2.10. Center for Independent Experts Review—2007

In August 2007, NMFS scientists at the National Marine Mammal Laboratory (NMML) of the Alaska Fisheries Science Center (AFSC) requested an independent review of scientific documents, analysis and the resulting conclusions which supported the proposed listing of Cook Inlet belugas as endangered under the U.S. Endangered Species Act. This included a review of the background biological data, population data, model structure and assumptions, the analysis methods applied to the extinction risk assessment, and the conclusions resulting from that assessment. The review panel was composed of four appointed reviewers from the Center for Independent Experts (CIE), one selected as the chair by the CIE. The panel convened at the NMML in Seattle, Washington, from November 13-16, 2007 to review the extinction risk assessment for Cook Inlet belugas. Each reviewer was provided with a set of documents for review in the days prior to meeting in Seattle. The three independent CIE reviewers and CIE chair met during the specified meeting dates to discuss and compile the draft peer-review reports. The authors of the primary review documents were available during the review meeting to address questions from the CIE reviewers. NMML received the reviews from the experts and a summary document from the panel chairman on January 10, 2008.

Overall, the CIE review panel agreed that the assessment represented the best available science and that the conclusions were supported by the scientific findings presented in the Status Review. The panel went on to recommend that the following information be included in a subsequent Status Review:

- A thorough explanation of the abundance survey technique and analyses (see Section 5.1.1).
- Using video records to provide information on population structure (currently in development).
- A discussion of the published vital rates and the key parameters used in the model (see Section 5.1.1).
- More information on the progressive reduction of the area in Cook Inlet used by belugas (see Section 2.2.1).
- A separate analysis of the survey data since 1999 (see Sections 2.3.2 and 5.1.2).
- A review of beluga populations once depleted and now recovering (see Section 3.12).
- The influence of variability in the data series on the estimation of turnover rates in the models (see Section 5.1.2).
- A clear statement of the assumption that environmental conditions will remain unchanged (model variations with on average at least one change in environmental conditions during the years 1994-2007 are included, see Section 5.1.1).

1.2.11. Status Review—2008

This revised Status Review addresses scientific issues raised during the public comment period (that closed on August 3, 2007) and updates the November 2006 Status Review to account for scientific data and other information that has become available in the interim including abundance estimates from 2006 and 2007. The CIE review panel comments on the November 2006 Status Review and updated and auxiliary analysis will be addressed in the final revisions prior to publication of the Status Review in April 2008.

1.3. Key Questions in ESA Evaluations

1.3.1. The ‘Species’ Question

For the purpose of the ESA, Congress has defined a species as “any subspecies of fish or wildlife or plants, and any distinct population segment of any species of vertebrate fish or wildlife which interbreeds when mature.” As amended in 1978, the ESA allows listing of “distinct population segments” of vertebrates, as well as named species and subspecies. Guidance on what constitutes a DPS is provided by the joint NMFS-USFWS interagency policy on vertebrate populations (61 FR 4722, February 7, 1996). To be considered “distinct,” a population, or group of populations, must be “discrete” from other populations and “significant” to the taxon (species or subspecies) to which it belongs. During the 1999 Status Review, it was concluded that Cook Inlet belugas are discrete from other Alaska beluga populations. In particular, all available data, including morphology, core and summer ranges, as well as genetics, indicated that the Cook Inlet belugas are an independent population that is distinct from other populations (65 FR 121, June 22, 2000). In addition, the loss of the population would result in a significant gap in the range of the taxon. Therefore, Cook Inlet belugas were considered significant with respect to the Alaska taxon and were designated a DPS on June 22, 2000 (65 FR 121). Additional information is presented in Hobbs et al. (2006).

1.3.2. The ‘Extinction Risk’ Question

The ESA defines the term endangered species as “any species which is in danger of extinction throughout all or a significant portion of its range.” The term threatened species is defined as “any species which is likely to become an endangered species within the foreseeable future throughout all or a significant portion of its range.” The ESA states that a variety of information should be used in evaluating the level of risk faced by a species or a DPS. Important considerations include Section 4(a)(1) of the Act which establishes whether a species is endangered or threatened based on one or more of the following five factors:

A) The present or threatened destruction, modification, or curtailment of its habitat or range;

B) Overutilization for commercial, recreational, scientific, or educational purposes;

C) Disease or predation;

D) The inadequacy of existing regulatory mechanisms; or

E) Other natural or man-made factors affecting its continued existence.

According to the ESA, the determination of whether a species is threatened or endangered should be made on the basis of the best scientific information available on its current status, after taking into consideration conservation measures that are proposed or are in place. This document is a compilation of biological data and a description of past, present, and likely future threats to the Cook Inlet belugas. It does not represent a decision by NMFS on whether this taxon should be proposed for listing as threatened or endangered under the ESA. That decision will be made by NMFS after reviewing this document, other relevant biological and threat data not included herein, and all relevant laws, regulation and policies. The results of the decision will be announced in the *Federal Register*.

2. UPDATES ON THE BIOLOGY, BEHAVIOR, ECOLOGY, AND POPULATION DYNAMICS OF COOK INLET BELUGAS

This section includes new or revised information that has become available since publication of the 2006 Status Review. Additional information on each topic is provided in Section 2 of Hobbs et al. (2006).

2.1. Beluga Biology and Behavior

2.1.1. Identifying Characteristics

Age and growth

The ages of belugas are estimated by counting growth layers in sections of their teeth. The initial hypothesis, that two growth layer groups (GLGs) per year were deposited in beluga teeth, was made by Sergeant (1959) and this hypothesis has been supported by many successive studies (Brodie 1969, 1982; Sergeant 1973; Goren et al. 1987; Brodie et al. 1990; Heide-Jorgensen et al. 1994). The deposition of two layers per year makes belugas unique among most mammals. Evaluations of previous work and analysis of two captive belugas (Hohn and Lockyer 1999), and radiocarbon signatures in beluga teeth (Stewart et al. 2006), however, indicate that GLGs form annually, not semiannually (though there have been some exceptions noted (see Lockyer et al. (2007)). This affects a number of the beluga population parameters presented within the 2006 review by effectively doubling, for example, the age at sexual maturity, age at first birth, life expectancy, and lifetime reproductive capacity. To remove ambiguity, these parameters have been updated to show the number of GLGs instead of ages (see Section 2.3.3).

Teeth from harvested and stranded Cook Inlet belugas, collected from 1992 to 2001, were used to establish GLG/length curves for female and male Cook Inlet belugas (Vos 2003). A total of 372 teeth from 58 whales were cut and analyzed. Growth curves were developed for females ($R^2 = 0.95$) and males ($R^2 = 0.93$). Sexual dimorphism was exhibited with males being longer than females at equal GLG counts.

Additional information about identifying characteristics is provided in Hobbs et al. (2006).

2.1.2. Distribution of Beluga Populations

Belugas are distributed widely in Arctic and subarctic waters and are generally associated with areas seasonally covered by sea ice (Hazard 1988). Beluga populations differ from one another in their use of specific summer habitats and physical characteristics such as body size, contaminant loads, and vocal repertoire leading to the identification of a number of independent stocks (O'Corry-Crowe 2002). The International Whaling Commission (IWC) currently recognizes 29 beluga stocks, many of which are depleted or their status is unknown (IWC 2000). Five populations of belugas occur in Alaska waters (Fig. 2.1.2-1): Cook Inlet, Bristol Bay, eastern Bering Sea, eastern Chukchi Sea, and the Beaufort Sea (Angliss and Lodge 2004; O'Corry-Crowe et al. 1997, 2002). Summer populations are found as far southeast as Yakutat Bay (northern portion of Southeast Alaska, 60°N 140°W) and northeast into the Canadian Beaufort Sea (east of 70°N 140°W).

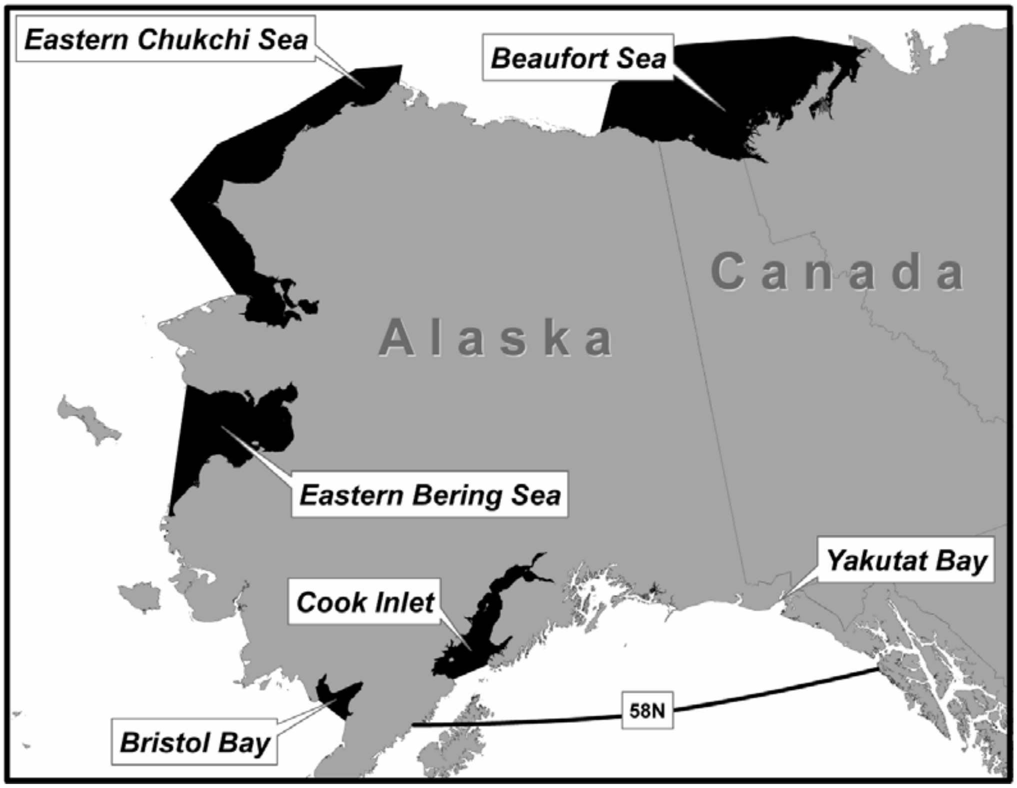


Figure 2.1.2-1. Summer locations of belugas found in Alaska waters.

Brown Gladden et al. (1999) divided the North American belugas into two evolutionarily significant units (based on mitochondrial and nuclear DNA), distinguishing Alaskan belugas from Canadian populations in Hudson Strait, Baffin Bay and the St. Lawrence River. Further distinctions between Alaska populations have focused on patterns of female beluga dispersal over time based on analyses of mitochondrial DNA (O'Corry-Crowe et al. 1997, 2002).

O’Corry-Crowe et al. (2007, 2008) examined genetic variation in several nuclear DNA markers (microsatellites) in belugas across Alaskan and northwest Canadian waters to characterize male beluga dispersal and breeding patterns. The report also includes further investigations of mtDNA variation. As with the earlier and current analyses of mtDNA, Cook Inlet (Fig. 2.1.2-2) was also the most distinct for microsatellite variation than all other Alaska populations. Based on these findings, O’Corry-Crowe et al. (2007, 2008) concluded that the Cook Inlet population may experience periodic immigration but it is demographically and reproductively isolated from other populations of belugas to the west and north. The phylogeographic structuring of mtDNA and microsatellite variation indicate that low male and female dispersal rates into the Cook Inlet population were long-established patterns.

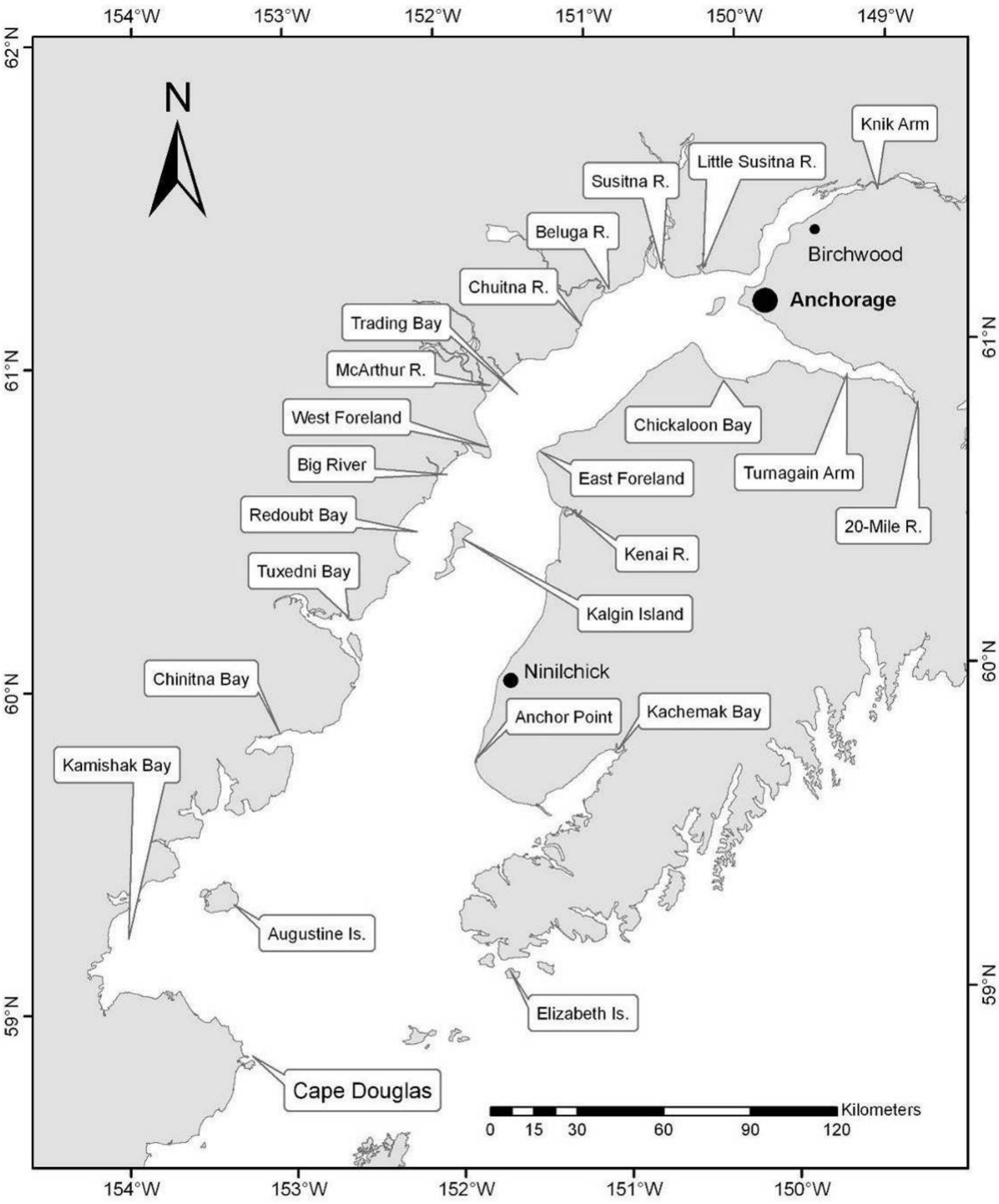


Figure 2.1.2-2. Cook Inlet and place names mentioned in the text.

As noted in the 2006 Status Review, Yakutat Bay is the only location in the Gulf of Alaska, outside of Cook Inlet where there is a known, persistent population of belugas (O'Corry-Crowe et al. 2006). Belugas were first reported during scientific surveys in Yakutat Bay in 1976 (see review in Laidre et al. 2000, O'Corry-Crowe et al. 2006). However, tribal elders recall regularly seeing belugas in the area since the 1930s, and sightings have occurred in all months except

December and January (O’Corry-Crowe et al. 2006). Preliminary genetic analysis of biopsy samples obtained from at least five individuals showed limited variation among Yakutat whales suggesting either high inbreeding or a closely related family unit (O’Corry-Crowe et al. 2006; G. O’Corry-Crowe, Harbor Branch Oceanographic Institution, February 13, 2008, pers. comm.). The single mitochondrial DNA haplotype shared by these whales is also found in other Alaska beluga populations, including Cook Inlet, although this haplotype occurs at a much lower frequency in Cook Inlet and other stocks (O’Corry-Crowe et al. 2006). It is not clear from available data whether this group is isolated from Cook Inlet.

2.1.3. Prey Preferences and Feeding Behavior

A more thorough analysis of the contents of stomachs collected from belugas that stranded or were harvested in Cook Inlet (Table 2.1.3-1) replaces the analysis presented in Hobbs et al. (2006). This analysis is ongoing (as noted in Table 2.1.3-1) and provides much needed evidence on prey availability and prey preferences of Cook Inlet belugas. Cook Inlet belugas feed on a wide variety of prey species (Table 2.1.3-2), focusing on specific species when they are seasonally abundant (Table 2.1.3-3). In the spring, eulachon (*Thaleichthys pacificus*) and gadids were preferred prey (Table 2.1.3-3). In April 1998, the stomach of a beluga harvested near the Susitna River was filled exclusively with eulachon (Table 2.1.3-2). Gadids, such as saffron cod (*Eleginus gracilis*) are indigenous to shallow coastal waters and are found near and in rivers within the zone of tidal influence (Morrow 1980, Cohen et al. 1990). Adult cod exhibit seasonal movements: inshore during winter for purposes of spawning and offshore during summer for feeding (Cohen et al. 1990). Pacific cod (*Gadus macrocephalus*) instead move to progressively deeper water as they age, spawning in deeper, offshore waters in winter and migrating to shallower water in the spring to feed (Cohen et al. 1990). Both species of cod are opportunistic epibenthic feeders (Cohen et al. 1990). Cod consume polychaetes, shrimp, amphipods, mysids, as well as other fish (e.g., walleye pollock (*Theragra chalcogramma*) and flatfish) (see Seaman et al. 1982, Clausen 1981, Cohen et al. 1990) suggesting that many of the invertebrates and possibly some of the fish species found in the stomachs of belugas may be the result of secondary ingestion (Tables 2.1.3-3 and 2.1.3-4).

Table 2.1.3-1. Characteristics of 24 Cook Inlet belugas analyzed for stomach contents collected between 1995-2007 (ID* = ID matched to Table 2.1.3-2). U = unknown.

Sample ID	ID*	Sample date	Year	Sex	Length	Color	Reason for death
692-BLKA-021	1[a]	3 May	1995				
DL9801	2[a]	9 Apr	1998				
692-BLKA-051/50	3[a]	13 May	1998	M			
9904-DL-BM	4[a]	30 Aug	1999				
9901-DL-BM	5[a]	1 Sep	1999				
9903-DL-BM	6[a]	1 Sep	1999				
17 Sep 00	7[a]	17 Sep	2000				
21 Jul 01	8[a]	21 Jul	2001	U	U	U	U
12 Oct 01	9[a]	12 Oct	2001	F	5' 5”	Gray	Stranding
DL-CI-02-01	10	U	2002	U	14' 3"	U	Harvest
DL-CI-02-02	11	28 Sep	2002	F	12' 2"	White	Stranding
28-29 August 2003	12	28-29 Aug	2003	M	13' 8"	White	Stranding
12 Sep 03	13	12 Sep	2003	F	11' 5"	White	Stranding
10 15 03 Motorcross	14	15 Oct	2003	M	15' 2"	White	Stranding
5 Nov 03	15	5 Nov	2003	U	U	U	U
DL-CI-01-03, BLKA-079	16	4 Aug	2003	F	12'	White	Harvest
DL 2003-017, 692-BLKA-078	17	1 Apr	2003	F	12'	White/ gray	Stranding
692-BLKA-080	18[b]	24 Jul	2005	M	14'	U	Harvest
692-BLKA-081	19[b]	11 Oct	2006	F	12'	White	Stranding
DL062907	20	29 Jun	2007	M	8' 4"	Gray	Stranding
3 Oct 2007	21	3 Oct	2007	U	12' 4"	U	Stranding
7 Oct 2007	22	7 Oct	2007	M	13' 9"	White	Stranding
16 May 2007	–[c]	16 May	2007				
1 Oct 2007	–[c]	1 Oct	2007	U	12' 2"	White	Stranding

a – Incomplete data so it was not included in Table 2.1.3-4: results from stomach samples examined by Pacific ID, Canada (ID 1-9), were not broken down into categories of weight, number, length, and percents—instead only a description of species was provided.
b – Awaiting invertebrate identification; invertebrates not identified in Table 2.1.3-2 or 2.1.3-3 or included in Table 2.1.3-4.
c – Awaiting stomach content identification; individuals not included in Table 2.1.3-2, Table 2.1.3-3 or Table 2.1.3-4.

Table 2.1.3-2. Prey species identified from 22 Cook Inlet beluga stomachs collected between 1995-2007. Gray shading indicates no prey items found in stomach.

Taxon	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22
Invertebrates																		X	X			
Annelid																						
Polychaete (jaws and eggs)																	X					
Crustacea																						
Decapoda																						
Shrimp *Crangon franciscorum*																	X					
Crab *Chionoecetes bairdi*																	X					
Fishes																						
Catostomidae																						
Longnose sucker *Catostomus catostomus*																X						
Cottidae																						
Pacific staghorn sculpin *Leptocottus armatus*														X								
Gadidae																						
Saffron cod *Eleginus gracilis*													X	X			X					
Cod *Gadidae* sp.													X						X			
Pacific cod *Gadus macrocephalus*			X														X					
Walleye pollock *Theragra chalcogramma*																	X		X			
Osmeridae																						
Eulachon *Thaleichthys pacificus*		X															X					
Pleuronectidae																						
Yellowfin sole *Limanda aspera*												X		X								
Starry flounder *Platichthys stellatus*														X								
Salmonidae																						
Chum salmon *Oncorhynchus keta*												X				X			X			
Coho salmon *Oncorhynchus kisutch*										X		X	X					X				
Salmon species *Oncorhynchus* sp.	X			X	X	X		X					X			X	X	X				
Unidentified fish			X						X										X			
Other																						
Gravel													X		X					X	X	
Wood, vegetation																	X					
Sea lice *Caligus* sp.												X						X	X			
Parasitic worms (e.g., nematodes)													X		X	X	X	X	X	X		X

Table 2.1.3-3. Prey species identified by month from 21 Cook Inlet beluga stomachs collected between 1995-2007. Sample date was not known for whale ID 10 (see Table 2.1.3-1). X = number of whale(s) found with prey item in their stomach.

Taxon	April (n = 2)	May (n = 2)	June (n = 1)	July (n = 2)	August (n = 3)	September (n = 5)	October (n = 5)	November (n = 1)
Invertebrates				X			X	
Annelid								
Polychaete (jaws and eggs)	X							
Crustacea								
Decapoda								
Shrimp *Crangon franciscorum*	X							
Crab *Chionoecetes bairdi*	X							
Fishes								
Catostomidae								
Longnose sucker *Catostomus catostomus*					X			
Cottidae								
Pacific staghorn sculpin *Leptocottus armatus*							X	
Gadidae								
Saffron cod *Eleginus gracilis*	X					X	X	
Cod *Gadidae* sp.						X	X	
Pacific cod *Gadus macrocephalus*	X	X						
Walleye pollock *Theragra chalcogramma*	X						X	
Osmeridae								
Eulachon *Thaleichthys pacificus*	XX							
Pleuronectidae								
Yellowfin sole *Limanda aspera*					X		X	
Starry flounder *Platichthys stellatus*							X	
Salmonidae								
Chum salmon *Oncorhynchus keta*					XX		X	
Coho salmon *Oncorhynchus kisutch*				X	X	X		
Salmon species *Oncorhynchus* sp.	X	X		XX	XX	XXX		
Unidentified fish		X					XX	
Other								
Gravel			X			X	X	X
Wood, vegetation	X							
Sea lice *Caligus* sp.				X	X		X	
Parasitic worms (e.g., nematodes)	X		X	X	X	X	XX	X

Table 2.1.3-4. Percent number and frequency of prey species in stomach contents of belugas collected in Cook Inlet from 2002-2007. Stomachs contents of whales processed by Pacific ID, Canada, (Whale IDs 1-9) could not be included because prey weights, lengths, percents, and stomach volume were not obtained during identification of prey species.

TAXON	Percent number	Percent frequency
INVERTEBRATES (n = 11)	**–**	**9**
Annelid		
Polychaete jaws and eggs	–	9
Crustacea		
Decapoda		
Shrimp *Crangon franciscorum*	–	9
Crab *Chionoecetes bairdi*	–	9
FISHES (n = 13)	**–**	**62**
Catostomidae	**1**	**8**
Longnose sucker *Catostomus catostomus*	1	8
Cottidae	**1**	**8**
Pacific staghorn sculpin *Leptocottus armatus*	1	8
Gadidae	**49**	**31**
Saffron cod *Eleginus gracilis*	31	23
Cod *Gadidae* sp.	4	15
Pacific cod *Gadus macrocephalus*	1	8
Walleye pollock *Theragra chalcogramma*	13	15
Osmeridae	**5**	**8**
Eulachon *Thaleichthys pacificus*	5	8
Pleuronectidae	**4**	**15**
Yellowfin sole *Limanda aspera*	3	15
Starry flounder *Platichthys stellatus*	1	13
Salmonidae	**39**	**54**
Chum salmon *Oncorhynchus keta*	9	23
Coho salmon *Oncorhynchus kisutch*	21	31
Salmon species *Oncorhynchus* sp.	8	31
Unidentified fish	1	8
OTHER (n = 13)		
Gravel	–	31
Parasitic worms (e.g., nematodes)	–	46

Although not evident in the stomach contents reported in the tables presented in this section, Natives describe Cook Inlet belugas feeding on anadromous steelhead trout (*Oncorhynchus mykiss*), freshwater fish such as whitefish (*Coregonus oidschian*), northern pike (*Esox lucius linnaeus*), and grayling (*Thymallus arcticus*) (Huntington 2000), and other marine fish such as tomcod (*Microgadus proximus*) during the spring (Fay et al. 1984). These species are abundant in the Susitna River system (www.adfg.state.ak.us/pubs/notebook/notehome.php, accessed October 2006). By late spring, belugas begin to shift from lipid-poor prey to lipid-rich species (Abookire and Piatt 2005, Litzow et al. 2006) as anadromous fish runs of Pacific salmon (*Oncorhynchus* spp.) enter the inlet (Table 2.1.3-3).

From late spring and throughout the summer months, the majority of beluga stomachs contained Pacific salmon coincident with the timing of fish runs in the area. Anadromous smolt and adult fish concentrate at river mouths and adjacent intertidal mudflats to osmoregulate during emigration and immigration, respectively (ADF&G 2004). Five Pacific salmon species: Chinook (*O. tshawytscha*), pink (*O. gorbuscha*), coho (*O. kisutch*), sockeye (*O. nerka*), and chum (*O. keta*) spawn in rivers throughout Cook Inlet (Moulton 1997, Moore et al. 2000). Calkins (1989) recovered 13 salmon tags from the stomach of an adult beluga found dead in Turnagain Arm. Beluga hunters in Cook Inlet reported one whale having 19 adult Chinook salmon in its stomach (Huntington 2000). Salmon, overall, had the greatest percent frequency of occurrence of the prey species found in the stomachs of Cook Inlet belugas (Table 2.1.3-4). Prey selection likely depends on the size of the whale. Belugas are sexually dimorphic with males being significantly larger than females of the same age (Burns and Seaman 1986, Vos 2003); in accordance, males have been found to consume larger fish than females (Seaman et al. 1982). Similarly, young whales consume significantly smaller prey items than adults (Seaman et al. 1982).

In the fall, as anadromous fish runs begin to decline, belugas again return to consume the fish species found in nearshore bays and estuaries. This included cod species observed in the spring diet as well as other bottom-dwellers: Pacific staghorn sculpin (*Leptocottus armatus*) and flatfishes such as starry flounder (*Platichthys stellatus*) and yellowfin sole (*Limanda aspera*) (Table 2.1.3-3). Pacific staghorn sculpin are commonly found near shore in bays and estuaries on sandy substrate (Eschmeyer et al. 1983). Flatfish are typically found in very shallow water and estuaries during the warm summer months and move into deeper water in the winter as coastal water temperatures cool (though some may occur in deep water year-round) (Morrow 1980). As late as October, belugas tagged with satellite transmitters continued to use Knik and Turnagain Arm and Chickaloon Bay but some belugas also ranged into the lower Inlet south to Chinitna Bay, Tuxedni Bay, and Trading Bay (MacArthur River) in the fall (Hobbs et al. 2005).

Stomach samples from Cook Inlet belugas are not available for winter months, December through March (Table 2.1.3-3). Dive data from belugas tagged with satellite transmitters suggest that during the winter whales are feeding in deeper waters (Hobbs et al. 2005), possibly on such prey species as flatfish, cod, sculpin, and pollock. In November, belugas moved between Knik, Turnagain Arm, and Chickaloon Bay, similar to patterns observed in September (Hobbs et al. 2005). By December, belugas were distributed throughout the upper to mid-Inlet. From January into March, belugas moved as far south as Kalgin Island and slightly beyond in central offshore waters. Belugas also made occasional excursions into Knik and Turnagain Arm in February and March in spite of ice cover greater than 90% (Hobbs et al. 2005).

According to local Native knowledge, the importance of the anadromous fish runs during the summer feeding period is evident in the blubber layer of these whales. In spring, the whales were described as thin with blubber only 2-3 inches (5-8 cm) thick compared to the fall when the blubber may be up to 1 ft (30 cm) thick (Huntington 2000). Mature females have additional energy requirements. Distinct mating periods, calving dates, and calving areas for the Cook Inlet beluga population are not well documented; however, calves are present during the summer months (Huntington 2000, Hobbs et al. 2005). Assuming a gestation period of 14 months and the known presence of pregnant females in late March, April, and June (Mahoney and Shelden 2000, Vos and Shelden 2005) suggests breeding may be occurring in late spring into early summer. Calves depend on their mother’s milk as their sole source of nutrition and lactation lasts up to 23 months (Braham 1984) though young whales begin to consume prey as early as 12 months of age (Burns and Seaman 1986). Therefore, the summer feeding period is critical to pregnant and lactating belugas.

2.2. Ecology of Cook Inlet Belugas

2.2.1. Temporal Changes in Distribution

As recommended by the CIE review panel, this section now provides more information on the progressive reduction of the area in Cook Inlet used by belugas. Declines in abundance may be reflected in changes in distribution either by a population shift away from impacted areas (via emigration or by dying off) or by a contraction of population range to within a preferred habitat (Fig. 2.2.1-1). The latter, a population proportionally reduced throughout the range but seen least in peripheral habitats, and therefore documented by biologists as a decreased range (Brown 1995), appears to be occurring in Cook Inlet.

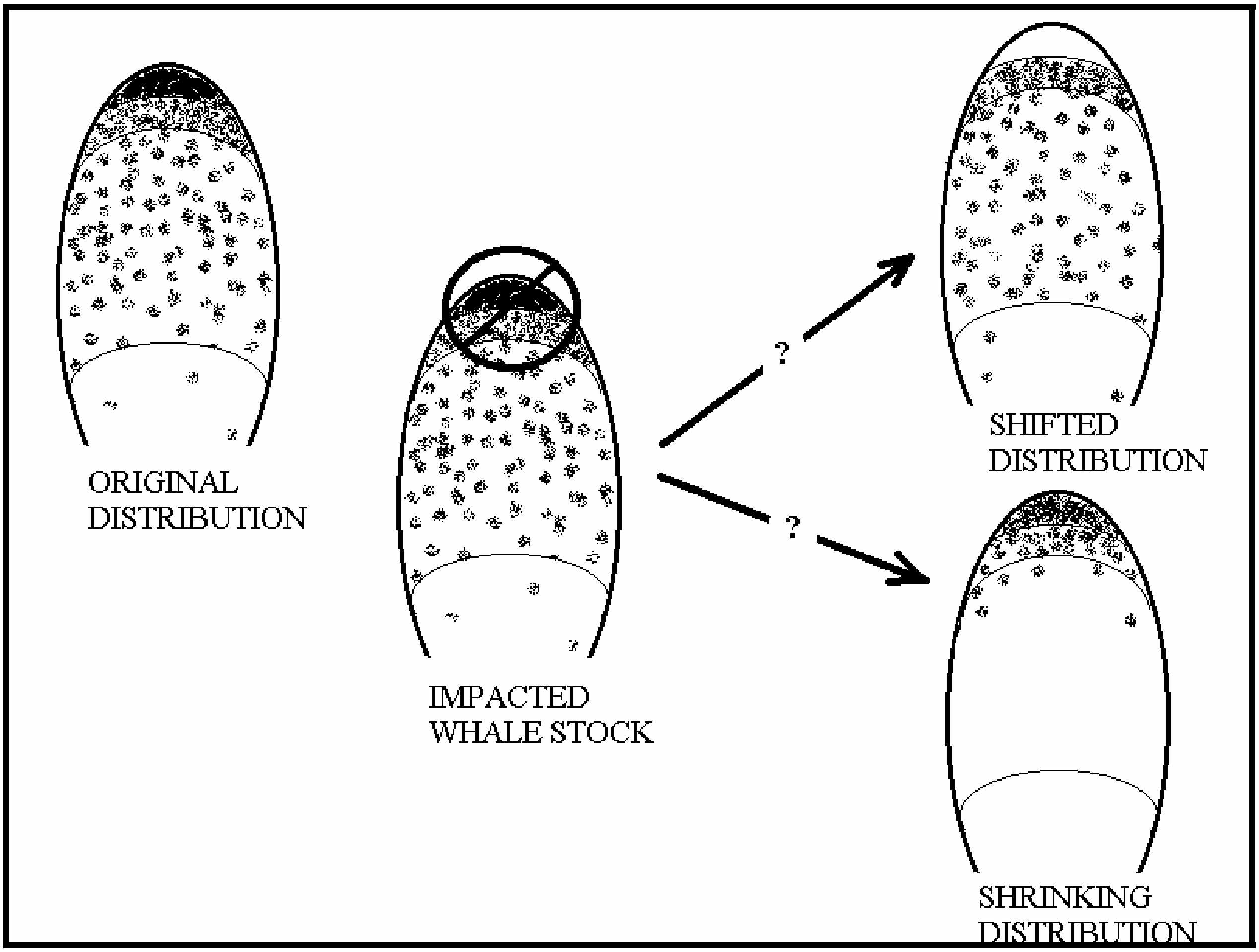


Figure 2.2.1-1. Changes in distribution of a whale population as it declines in abundance.

The extent of the summer distribution (June and July) of belugas in Cook Inlet has changed considerably since the late 1970s. Belugas were distributed over a relatively large area in the 1970s with 10% of the groups observed during aerial surveys occurring south of the Kenai River and Kalgin Island (60º 30' N), and half of the sightings occurring south of the McArthur River and Moose Point (60º 53' N) (Fig. 2.2.1-2a). From 1993 to 2007, most (96-100%) of the beluga sightings occurred in upper Cook Inlet and have been concentrated in shallow areas near river mouths north and east of the Beluga River (61º 12' N) and Point Possession (61º 01' N), rarely occurring in the central or southern portions of the inlet (Fig. 2.2.1-2b, c). The proximity to Anchorage has increased significantly since the late 1970s. The median distance from Point Woronzof (the western tip of Anchorage) for individual whales was 26 nautical miles (nmi) (48 km) with 90% of the whales within 70 nmi (129 km) in the late 1970s (Fig. 2.2.1-2a). These radii decreased to 18 nmi (33 km) ($P = 0.03$) and 23 nmi (43 km) ($P = 0.01$), respectively, for the period 1993-1997 (Fig. 2.2.1-2b) and decreased further to 13 nmi (24 km) ($P < 0.01$) and 20 nmi (38 km) ($P < 0.01$), respectively, in the last 10 years, 1998-2007 (Fig 2.2.1-2c).

Belugas have remained in the area of highest impact from hunting (on the north end of Cook Inlet, near Anchorage), and have disappeared from peripheral habitats (in the southern end of the inlet). It is unknown if the current contracted distribution is a result of changing habitat (Moore et al. 2000), predator avoidance (Shelden et al. 2003), or a shift of a reduced population into preferred habitat areas (Rugh et al. 2001, Goetz et al. 2007), regardless, the result is a greater proximity to Anchorage and a smaller range.

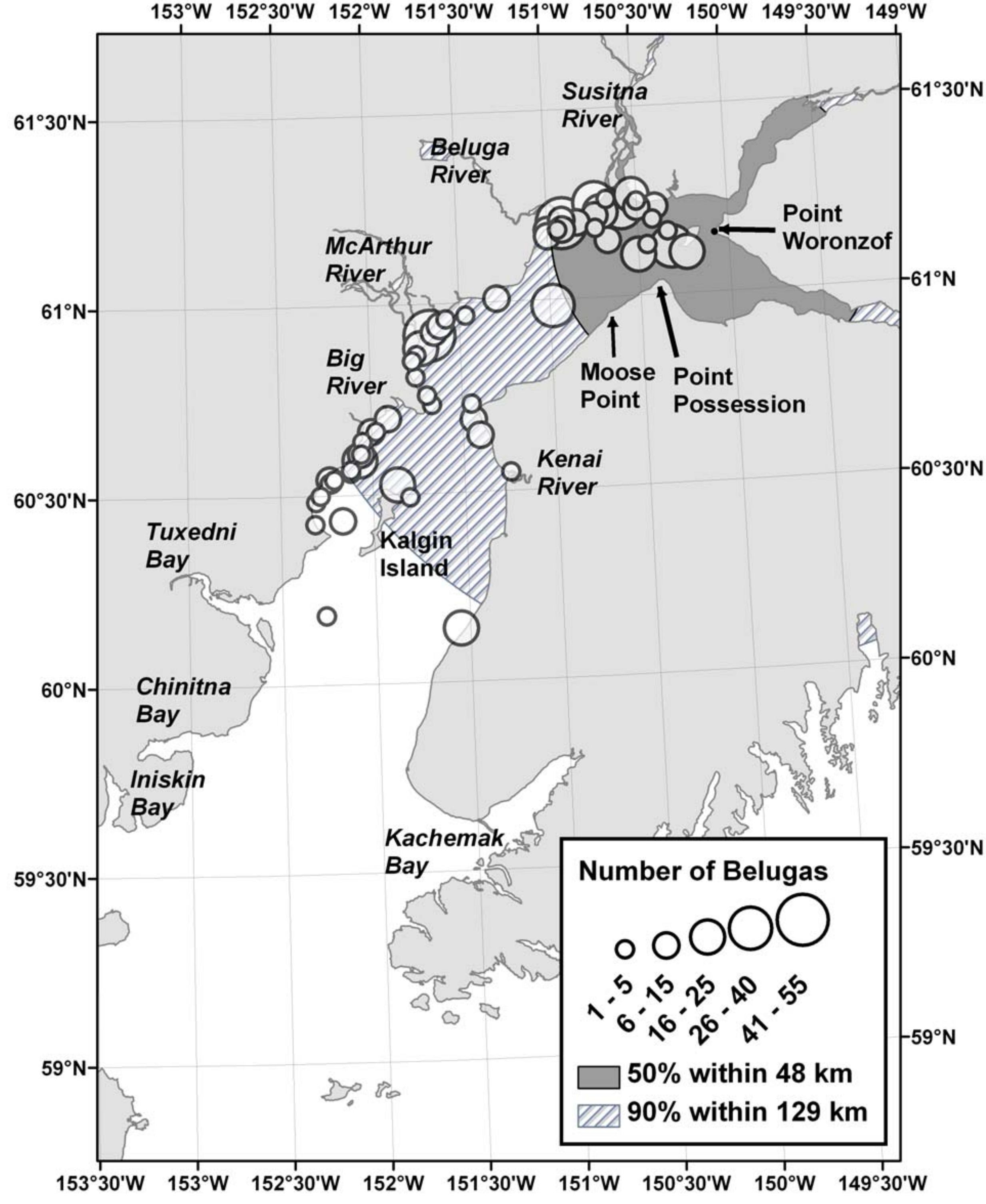


Figure 2.2.1-2a. Areas occupied by belugas in Cook Inlet in June and July in 1978-1979.

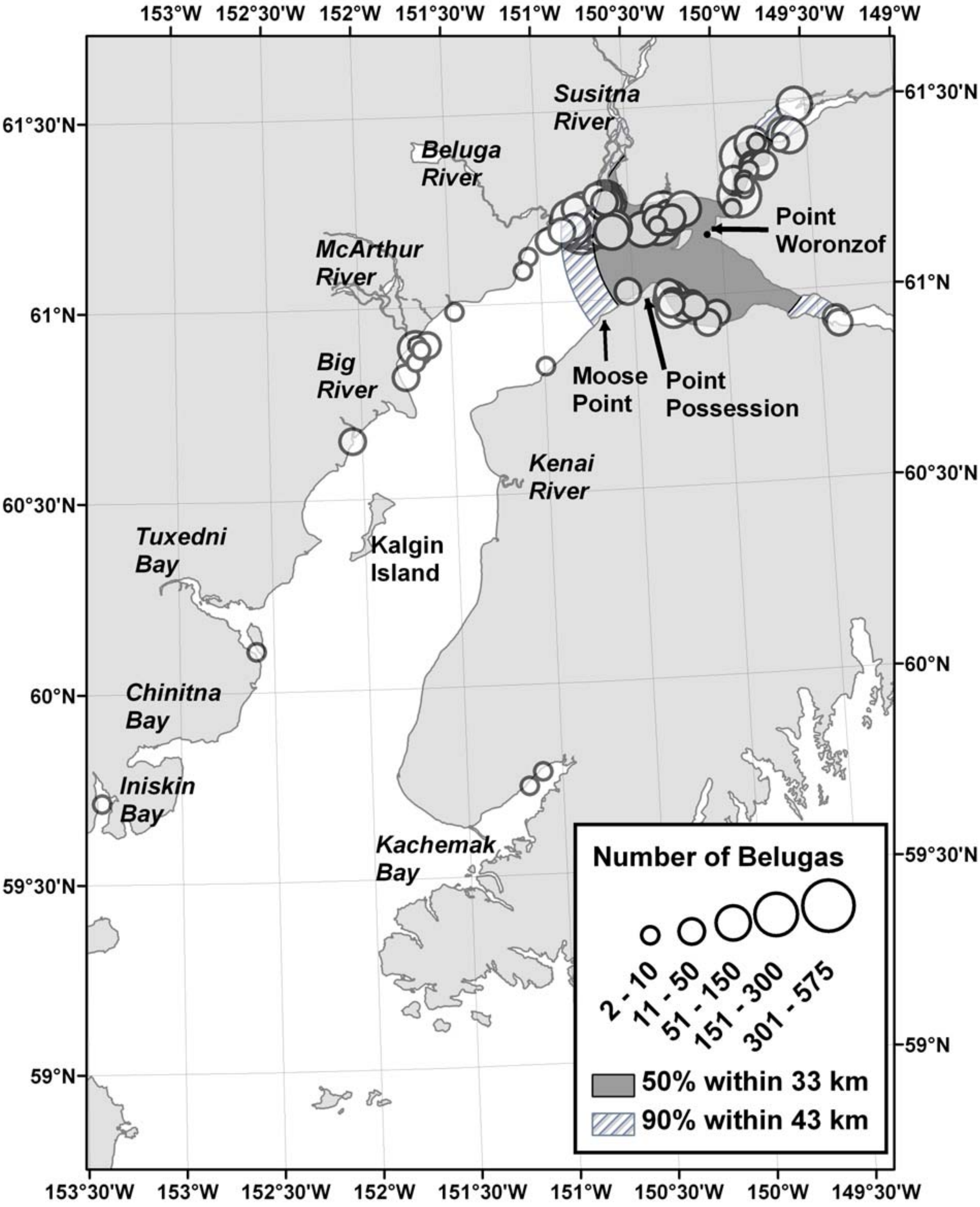


Figure 2.2.1-2b. Areas occupied by belugas in Cook Inlet in June and July in 1993-1997.

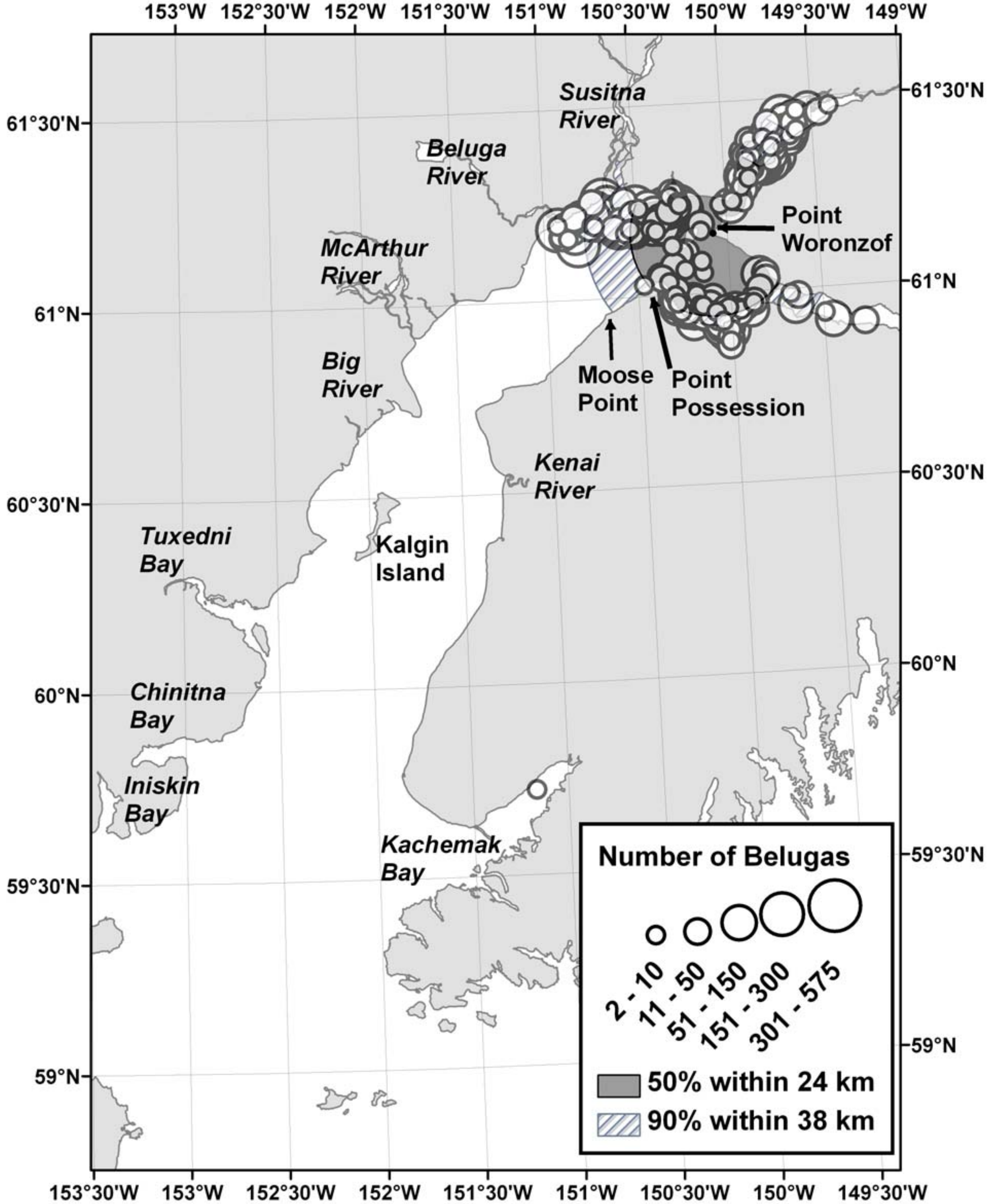


Figure 2.2.1-2c. Areas occupied by belugas in Cook Inlet in June in 1998-2007.

2.2.2. Habitat Use and Requirements

Information about habitat use is provided in Hobbs et al. (2006).

2.3. Population Dynamics

2.3.1. Population Size

The National Marine Fisheries Service (NMFS) has adopted 1,300 as the value for the carrying capacity (*K*) to be used for management purposes (65 FR 34590, May 31, 2000). NMFS began comprehensive, systematic aerial surveys of the beluga population in Cook Inlet in 1993. These surveys documented a decline in abundance of nearly 50% between 1994 and 1998 (Fig. 2.3.1-1) from an estimate of 653 (CV = 0.43) whales to 347 (CV = 0.29) whales (Hobbs et al. 2000a). Estimates since 1998 have ranged from 435 (CV = 0.23) to 278 (CV = 0.18) whales (Fig. 2.3.1-1).

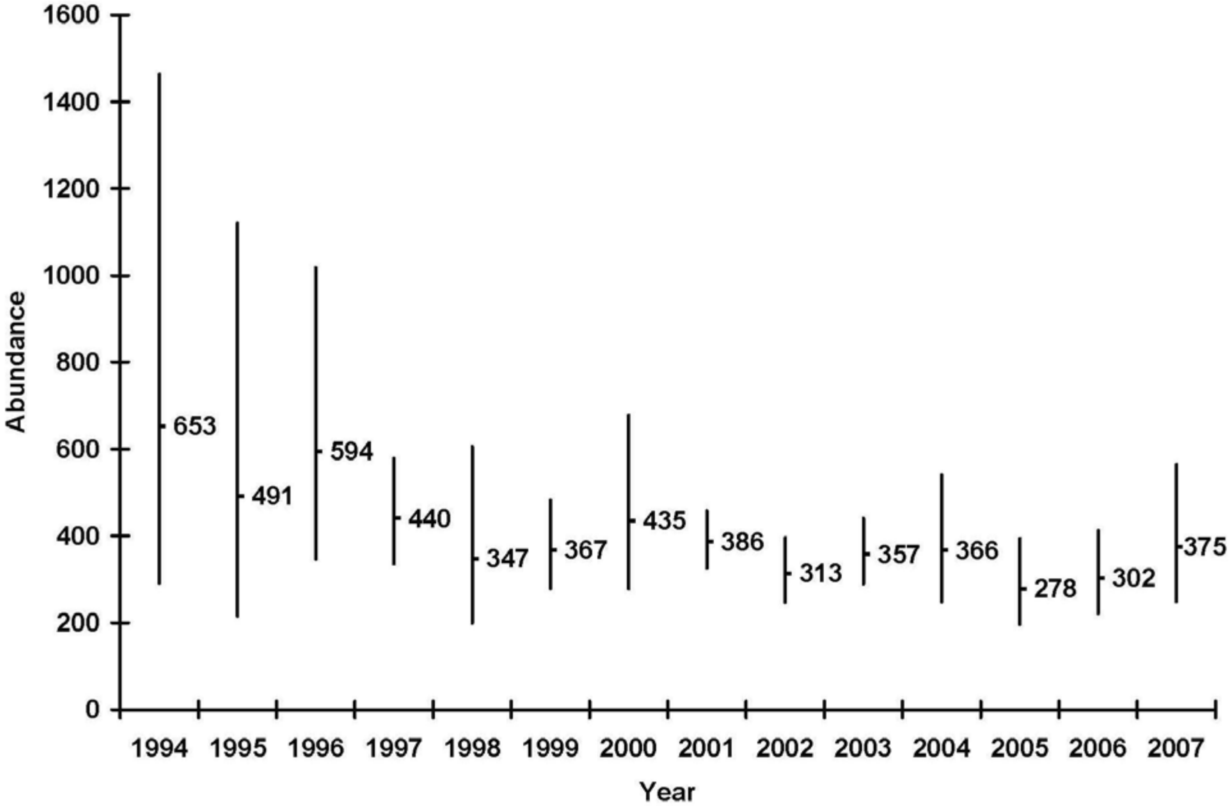


Figure 2.3.1-1. Estimated abundance of Cook Inlet belugas from NMFS annual aerial surveys, 1994-2007, showing average abundance and 95% confidence interval for each year.

2.3.2. Population Trends

With the very limited hunt between 1999 and 2007, NMFS anticipated that the population would begin to recover at a rate of 2% to 6% per year. When only the 1999-2007 time series of abundance estimates is considered, the rate of decline is estimated at -2.75% per year. While this is not significantly different from a growth rate of 0% per year, it is significantly different from a growth rate of +2% per year and, therefore, the population is not recovering at the minimum rate expected. For the 1994-2007 time series, the rate of decline is -3.96% per year and is significantly different from a trend of 0% per year, indicating the population is not growing.

2.3.3. Life History Parameters

Information about life history parameters can be found in Hobbs et al. (2006). As mentioned in Section 2.1.1, the table provided here (Table 2.3.3-1) and parameters used in the extinction risk models now show number of GLGs acquired in beluga teeth annually instead of ages.

Table 2.3.3-1. Review of female beluga life history parameters found in the published literature**.**

Parameters	Data		Source(s)
Age at sexual maturity	8-15 growth layer groups (GLGs) 0% at 8-9 GLGs 33% at 10-11 GLGs 94% at 12-13 GLGs 9.1 ± 2.8 GLGs		1,2,3,4,5,6, 6[a] 7
Age at color change (gray to white)	12 GLGs 22 GLGs		1 2
Age at 1st conception	54% at 8-9 GLGs 41% at 10-11 GLGs 94% at 12-13 GLGs		6[b]
Age at senescence	42-43 GLGs		1
Pregnancy and birth rates	with small fetuses: 0.055 at 0-11 GLGs 0.414 at 12-21 GLGs 0.363 at 22-45 GLGs 0.267 at 46-57 GLGs 0.190 at 58-77 GLGs	with full-term fetuses or neonates: 0.000 at 0-11 GLGs 0.326 at 12-21 GLGs 0.333 at 22-45 GLGs 0.278 at 46-51 GLGs 0.182 at 52-57 GLGs 0.125 at 58-77 GLGs	6
Lifespan	>60 GLGs (oldest female estimated at 70+ GLGs) 64-65 GLGs 60-61 GLGs 50-51 GLGs		6 8 1 2

Parameters	Data	Source(s)
Adult annual survival	0.96-0.97 0.955 (based on pilot whale data) 0.935 0.91-0.92 0.906 (includes both natural and human-caused mortality) 0.84-0.905 (based on body length and lifespan)	9 10 11 12 6 13
Immature annual survival	.0905 (for neonates in the first half year of life)	2
Reproductive rate	0.010-012 0.11 (based on annual calf production rates) 0.13 (based on annual calf production rates) 0.09 (based on annual calf production rates) 0.09-0.12 (based on annual calf production rates) 0.09-0.14 (based on calf counts) 0.12 (based on calf counts) 0.08-0.14 (based on calf counts) 0.06-0.10 (based on calf counts) 0.08-0.10 (based on calf counts) 0.08 (unknown)	14[c] 6 2 1 5 5 15, 2 16 17 10 18
Calving interval	< 3 years 2 years and 3 years	6[d] 2[e]

1. Brodie 1971; 2. Sergeant 1973; 3. Ognetov 1981; 4. Seaman and Burns 1981; 5. Braham 1984; 6. Burns and Seaman 1986; 7. Robeck et al. 2005; 8. Khuzin 1961 (cited in Ohsumi 1979); 9. Béland et al. 1992; 10. Brodie et al. 1981; 11. Lesage and Kingsley 1998; 12. Allen and Smith 1978; 13. Ohsumi 1979; 14. Perrin 1982; 15. Ray et al. 1984; 16. Davis and Evans 1982; 17. Davis and Finley 1979; 18. Breton-Provencher 1981.

[a] Alaska sample (52 whales). Sampling occurred in June, a time when most Alaskan belugas are born. It is possible non-pregnant 8-9 GLGs belugas would have conceived before their 10-11 GLGs birth date.

[b] Alaska sample (22 whales).

[c] Based on a literature review and adopted by the International Whaling Commission.

[d] For some female beluga whales. This was a tentative conclusion based on high conception rates noted in some females between the ages of 12-13 GLGs and 44-45 GLGs.

[e] Two-year intervals were for 25% of mature female belugas in eastern Canada (7 of 29 sampled); presumed after noting pregnancies occurred during lactation. Three year intervals were for 75% of mature females in eastern Canada. Sergeant (1973) concludes “overlap of pregnancy and previous lactation is infrequent so that calving occurs about once in three years.”

3. POTENTIAL RISK FACTORS FOR COOK INLET BELUGAS

The following section provides an update to the previous Status Review (Hobbs et al. 2006) and discussion about potential factors which are believed to have some impact on the Cook Inlet beluga population and mitigation measures that are currently in place. At reduced numbers and with contraction of their range, this population is far more vulnerable to losses due to stranding, predation, or disease. This population relies heavily on several fish species that are available only seasonally and are also of considerable commercial interest. Disturbances that cause belugas to temporarily or permanently abandon summer feeding areas could reduce their ability to survive through the winter months. These risk factors are also described within the MMPA Conservation Plan (NMFS 2005).

3.1. Stranding Events

In 2007, 13 beluga mortalities were reported in Cook Inlet, none of which were associated with a mass stranding (NMFS, unpublished data). Information on stranding events is provided in Hobbs et al. (2006).

3.2. Predation

On June 14, 2007, an adult beluga was chased and killed by a pod of killer whales near Anchor Point in lower Cook Inlet (NMFS, unpublished data). Information on predation is provided in Hobbs et al. (2006).

3.3. Parasitism and Disease

Introduction and background

The section on parasitism and disease has been expanded beyond that presented in Hobbs et al. (2006) to include additional information on Cook Inlet belugas and review infections in marine mammals and potential sources of parasites and pathogens that exist within the Cook Inlet environ. Infectious diseases and pathogens have been identified as among the top risks endangering species, possibly resulting in extinction (Harvell et al. 2002, Deem et al. 2001, Daszak et al. 2000, Anderson and May 1992, Scott 1988), and have extirpated certain local populations (Daszak et al. 1999, McCallum and Dobson 1995), however, the available data supporting these contentions is mostly anecdotal (Smith et al. 2006). Epidemiological theory suggests that species are driven to extinction by pathogens only under specific conditions: 1) the pre-epidemic host population size is small (i.e., the species is endangered); 2) the pathogen/parasite relies on non-density dependent transmission between hosts; or 3) when the pathogen reservoir is the abiotic environment (de Castro and Bolker 2005). However, while disease may not cause the complete extinction of the species, it can produce enough mortality to threaten the species or trigger the disappearance of local stocks or populations increasing the risk posed by other mechanisms (de Castro and Bolker 2005). Small population size is the most widely cited driver of disease-induced extinction of the three previously mentioned conditions. Lack of genetic variability is another feature of the risk associated with small population size. The lack of genetic variability in a species, or stock, may reduce its immune response, leaving it more susceptible to disease (O'Brien and Evermann 1988).

Although other factors such as habitat loss and overexploitation have been listed as single causes driving a species to extinction, infectious disease has not been listed alone (IUCN 2004). This suggests that disease alone may be unlikely to drive a species to extinction, but may be much more likely when in combination with other contributing risk factors such as pollution, habitat loss, and human disturbance (Smith et al. 2006). Nearly every wild animal has some parasites, and the role of parasites in causing disease and mortality is often difficult to interpret. Potential causes of stranding and mortality in marine mammals include environmental conditions, biotoxins, nutritional factors, human interaction, predators, congenital defects, neoplasia and possibly environmental contaminants, parasites, other disease agents.

Information on parasites, disease agents and pathology in belugas is available in the literature, but little has been published on parasites, disease agents and pathology of the Cook Inlet stock, therefore, little information is available about the role these may play in the decline and long-term viability of Cook Inlet belugas. This has been exacerbated by small sample sizes and the poor quality of most of the Cook Inlet beluga carcasses examined up to this point (K. Burek, Alaska Veterinary Pathology Services (AVPS), January 2008, pers. comm.). Between 1998 and 2007, varying degrees of necropsies and sampling have been completed on 36% (13 of 36) of Cook Inlet beluga carcasses. In many instances, carcasses were in advanced autolysis, so minimal diagnostics could be performed; however, some data on parasites and possible diseases were collected. When logistically feasible, an attempt is made to respond to a stranded animal, perform a necropsy and complete as much tissue sampling as possible, given the state of decomposition and geographic location of the animal. Some tissue samples are examined histopathologically, while others are submitted for bacteriology and virology testing. In the few instances of Cook Inlet whales killed during subsistence harvests, an examination was done by a biologist and tissues were sampled. Belugas from other Alaska populations hunted by Native Alaskans have also been sampled to various degrees.

In a paper evaluating the threats of infectious disease on a population of endangered resident killer whales in the Pacific Northwest, Gaydos et al. (2004) identified several high priority pathogens that warrant further study. These pathogens were identified through analysis of infectious diseases reported for other killer whales, both free-ranging and captive, as well as sympatric toothed whales. Gaydos et al. (2004) advocated the development of standardized necropsy protocols which would include analysis for these and other appropriate agents. Their study also noted that in long-lived species, infectious diseases that affect fecundity (fertility) or reproductive success and juvenile survival, could have population effects.

Adapting from the methods in Gaydos et al. (2004), an attempt was made to identify not only infectious pathogens (bacteria, fungi, protozoa, and viruses), but also parasites in free-ranging and captive belugas, as well as those in free-ranging and captive sympatric species found within and around Cook Inlet. Cook Inlet belugas frequently encounter harbor seals (*Phoca vitulina*) (Montgomery et al. 2005) and may regularly encounter harbor porpoises (*Phocoena phocoena*) (Rugh et al. 2005a), northern sea otters (*Enhydra lutris kenyoni*) (Wynne 1997) and killer whales (Shelden et al. 2003). Several species of large whales that include the lower portions of the Inlet in their range and seasonal distribution are the fin (*Balaenoptera physalus*), sei (*Balaenoptera borealis*), humpback (*Megaptera novaeangliae*), gray (*Eschrichtius robustus*), and sperm (*Physeter macrocephalus*) whales. Occasional or rare occurrences in the Inlet include Dall's porpoise (*Phocoenoides dalli*), Stejneger's beaked whale (*Mesoplodon stejnegeri*) (Shults et al. 1982), minke whale (*Balaenoptera acutorostrata*), Pacific white-sided dolphin (*Lagenorhynchus obliquidens*), and Pacific walrus (*Odobenus rosmarus divergens*) (Wynne 1997). If sympatric species within the Cook Inlet beluga range are susceptible to an infectious pathogen, then the beluga could also be susceptible, bearing in mind, however, the locality of the pathogen and the probability of a Cook Inlet beluga being exposed when assessing risk and the type of pathogen in question. It is also important to take into account potential sources of pathogens and parasites from terrestrial mammals such as brown bear (*Ursus arctos*) (Van Daele 2007) moose (*Alces alces*), American black bear (*Ursus americanus*), north American beaver (*Castor canadensis*), river otter (*Lontra canadensis*), domestic cats (*Felis silvestris catus*) and small rodents, with ranges and seasonal distributions along the Inlet estuary.

While there are literally scores of diseases and parasites that have been documented and described in marine mammals, in this section only those of most importance with respect to reproductive effects and long-term viability of the Cook Inlet population are discussed in detail (see Section 3.12).

Infectious pathogens

Diseases of most concern in conservation of a rare species are those with a broader host range, or if the species is an accidental host for pathogens that are introduced, or pathogens that are altered by climate change, pollution, or habitat destruction (Lafferty and Gerber 2002). A more extensive review of various bacteria isolated from a variety of marine mammals is available (Higgins 2000).

Bacteria

Bacterial agents are a part of normal flora in many species of marine mammals, and presence of these organisms should be interpreted with caution to determine whether they are commensal organisms, pathogens or secondary invaders. According to some reports, bacterial infection, particularly of the respiratory tract, is one of the most common diseases encountered in marine mammals including small cetaceans. Bacterial pneumonia, either alone or in conjunction with parasitic infection, is a common cause of beach stranding and death (Howard et al. 1983).

Numerous bacterial agents were recovered from belugas caught for immediate release in the Churchill River, Manitoba, Canada, and from several that were transported to aquaria and maintained in captivity (Buck et al. 1989). From the St. Lawrence Estuary population, cases of septicemia with *Nocardia* spp. secondary to mastitis and *Vibrio cholerae* have been described (Martineau et al. 1988). Nocardiosis has also been reported in several odontocetes and in a young, captive-born beluga (MacNeil et al. 1978). A case of *Erysipelothrix* as a cause of systemic disease and dermatitis has been reported in a captive beluga (Calle et al. 1993), as well as *Vibrio parahaemolyticus* and *Edwardsiella* as causes of sepsis (Higgins 2000). The presence of *Mycobacterium* in belugas is not very well documented, but has been associated with a dermatitis and panniculitis in a captive animal with aortic rupture (Bowenkamp et al. 2001).

Several organisms have been reported in free-ranging and captive sympatric species; however, the probability of exposure may be low due to the geographic location of the reported case or the fact it was a captive animal. *Edwardsiella tarda* was reported to cause mortality in a free-ranging southern resident killer whale, but has not been reported in Alaska killer whales (Ford et al. 2000). The organism was also isolated, along with *Escherichia coli* and *Clostridium perfringens*, from transmural intestinal abscesses in a stranded juvenile gray whale off the northern California coast (Dailey et al. 2000).

Brucella is thought to have potential for primary disease including abortion, other reproductive lesions, and brain lesions (Jepson et al. 1997). *Brucella* antibodies have been detected in Canadian Arctic belugas but the risk to beluga whales is currently unknown (Nielsen et al. 2001b). Reproductive lesions, mainly in the ovaries, have been shown in Canadian belugas, which may have the potential to lower fecundity and affect long-term population viability (IWC 2007). Virulence of *Brucella* is low in individuals but may potentially reduce fecundity, thus affecting long-term viability of Cook Inlet belugas as a population (Gaydos et al. 2004). Due to the quality of carcasses of Cook Inlet belugas, very little bacterial culture work has been done.

Viruses

Viruses are widespread in marine mammals and have become recognized as important causes of individual and mass mortalities, and include the morbilliviruses, influenza and possible herpesviruses (Munn 2006). Viruses can also compromise an animal's immune system and render it susceptible to secondary invaders such as bacterial agents and parasitic protozoa. Other viruses with varying effects on health have been identified in other marine mammal species; however, viral infections have not been well-studied in belugas.

In a review of morbillivirus, poxvirus and papillomavirus infections in cetaceans by Van Bressem et al. (1999), the authors discussed the effects of these viruses on host population dynamics. Infection by poxviruses was not believed to affect the general health of cetaceans (i.e., low mortality rate); however, it was hypothesized that mortality would be higher in neonates and calves, thus eventually affecting long-term population dynamics (Van Bressem et al. 1999). Poxviruses generally produce mild skin lesions and might indicate other illness or stress (Nollens et al. 2005).

Papillomaviruses cause genital and cutaneous warts in a variety of cetacean species and have been identified in gastric papillomas in belugas (De Guise et al. 1994). A survey for selected viral infections in beluga whales in Canada yielded animals seropositive (46% by serum neutralization) to bovine herpesvirus-1, 0% to dolphin mortillivirus (DMV) and porpoise mortillivirus (PMV). Another survey for selected viral agents of serum and whole blood was performed on 54 harvested belugas from the Northwest Territories (Canada) and yielded a prevalence of 2% seropositive to phocine herpesvirus (PHV) and 7% to dolphin rhabdovirus (DRV) (Philippa et al. 2004). The authors suggested the latter infection (DRV), or infection with an antigenically closely-related virus, may be enzootic in belugas or the result of interspecies transmission. Although the rhabdoviruses, of which the rabies virus is a member, are highly virulent, the threat to the Cook Inlet beluga population is considered low, unless the rabies virus becomes enzootic in terrestrial species utilizing the rivers and nearshore waters of Cook Inlet (Philippa et al. 2004).

Low numbers and percentages of belugas seropositive to influenza A have been detected in Arctic Canada with no evidence of associated disease (Nielsen 2001a). Infection with the influenza virus results in respiratory illness, commonly resulting in secondary bacterial infections. These isolates are closely related to avian influenza in birds, are highly virulent, and transmitted most efficiently during the cetacean's respiration in the vicinity of seabirds (Munn 2006). Ohishi et al. (2004) and Ohishi et al. (2002) found serological evidence that influenza A and B strains, closely related to those in humans, occur in seals. The seals may then act as reservoirs of infection and provide opportunities for new strains to emerge via recombination and potentially infect belugas in the Inlet (Osterhaus et al. 2000). Four influenza A viruses have been documented in harbor seals in outbreaks that occurred along the New England coast (Geraci et al. 1982, Hinshaw et al. 1984, Callan et al. 1995), but may still pose a threat to Cook Inlet belugas due to the highly virulent nature of the viruses.

Herpesviruses have been linked to skin lesions, esophageal lesions, encephalitis, and neoplasia. Herpesviruses have also been reported to cause encephalitis in harbor porpoises (Kennedy et al. 1992) and may cause large-scale mortality in belugas. These viruses have been detected in belugas and several sympatric species, and may persist in an infected host with periodic or continuous shedding. Cases of a necrotizing/ulcerative dermatitis, and possibly esophageal ulcers due to a herpesvirus, have been reported in belugas in several populations outside of Alaska (Martineau et al. 1988, Barr et al. 1989, Mikaelian et al. 1999). In Cook Inlet, there have been a few necropsy cases, and a few live animal reports, in which belugas had skin lesions suggestive of a viral etiology such as a herpesvirus. In one necropsy case of a juvenile female, there was a systemic vasculitis and herpesvirus was detected by electron microscopy (EM) in necrotic adrenal gland tissues suggesting this animal died of a systemic herpesviral infection. Several cases of skin lesions in hunter-killed animals in other stocks have been seen, however, researchers have not been able to confirm the viral etiology at this point. Ongoing investigations include viral polymerase chain reaction (PCR) at the University of Florida and more EM work. Other differentials for skin lesions include poxvirus, papillomavirus, caliciviruses, drug reactions, and a variety of bacterial agents including *Erysipelothrix*, *Vibrio* spp., *Dermatophilus* spp.

Morbilliviruses may affect a population long-term through enzootic infections or recurrent epizootics, more so through the latter. Morbilliviruses tend to sustain themselves in large host populations as infection with the virus characteristically results in either rapid death or recovery and lifelong immunity. Closely related variants of cetacean morbillivirus (CMV) are responsible for the disease in porpoises, dolphins, and whales, while the phocine distemper virus is a distinct species (de Swart et al. 1995). In a larger study in the St. Lawrence Estuary, NWT, Nunavut, 445 belugas were negative for the marine mammal morbilliviruses indicating the animals either are not susceptible to these viruses or have not been exposed (Nielsen et al. 2000). Morbillivirus epizootics can occur as a result of cross-infection of different species or animals from different stocks and is encouraged by the widespread migratory habits of many marine mammals (Munn 2006). Several sympatric species that are known to have ranges that overlap that of the Cook Inlet belugas have been shown to be infected with morbillivirus in other parts of the world and include harbor porpoises (Van Bressem et al. 2001) and harbor seals (Kennedy-Stoskopf 2001).

Marine morbilliviruses have not been detected in the Pacific Ocean, nevertheless, given the small size of the Cook Inlet beluga population and its immunological naïveté to morbillivirus, the likelihood of an epizootic involving this organism is high and could potentially impact the viability of this stock. Canine distemper also has been reported in outbreaks in different parts of Alaska.

A captive Pacific white-sided dolphin was reported as having chronic hepatitis caused by a hepatitis B-like virus (Bossart et al. 1990).

Fungi

Mycotic infections in marine mammals represent a relatively small, but significant fraction of infectious diseases in marine mammals, and have been isolated from at least 27 species, including the beluga and sympatric species (Reidarson et al. 1999). Pulmonary aspergillosis is the most common mycotic infection in marine mammals and has been isolated in a captive beluga, harbor seal and killer whale (Reidarson et al. 2001). Various mycotic organisms have been isolated from both immunocompromised and immunocompetent animals. Infection with zygomycotic fungal organisms have been reported in a captive killer whale and Pacific white-sided dolphin (Robeck and Dalton 2002), and while virulent, pose a low epizootic potential and threat to Cook Inlet belugas. Mycotic diseases, such as *Aspergillus fumigatus*, have been reported in captive belugas (Young et al. 1999). The probability of free-ranging belugas being exposed to fungal organisms is low compared to captive animals, given the former are less frequently in contact with terrestrial fungal sources and antibiotic usage, two well-known risk factors in captive belugas (Reidarson et al. 2001). Given this information, however, mycotic pathogens should still be considered a mild to moderate health threat to Cook Inlet belugas, especially in animals that may already be immunocompromised from contaminants, other disease, and stressors.

Protozoa

Toxoplasmosis has been reported sporadically in the St. Lawrence Estuary animals (Mikaelian et al. 2000). Encysted protozoal organisms within muscle tissues of Cook Inlet belugas are commonly found. The parasite is consistent with *Sarcocystis* spp., which in other marine mammals in this tissue phase, is thought to be incidental and non-pathogenic. This has been reported in belugas in the St. Lawrence Estuary (De Guise et al. 1993).

Giardia spp. and *Cryptosporidium* spp. have not been isolated from belugas (Olson et al. 1997, Hughes-Hanks et al. 2005), but they have been detected in harbor seals (Measures and Olson 1999). These protozoa have also been detected in marine waters and can survive in this environment, which may implicate human waste through discharge of municipal or boat wastewater and terrestrial wildlife as sources of contamination (Fayer et al. 2004). Giardia has been documented in voles (*Clethrionmys* spp.) whose distribution includes the estuaries along Cook Inlet (ADF&G 2005). Evaluation of the presence of *Giardia* and *Cryptosporidium* and their role in beluga health should be further investigated.

Parasites

Some parasites have been implicated as causes of strandings. For example, aberrant migrations of the trematode *Nasitrema* through the brain have been linked to strandings (O’Shea et al. 1991, Degollada et al. 2002). Some lung worms can be associated with secondary bacterial infections and severe pneumonia (Geraci and Lounsbury 2005).

In a study on helminths in 10 hunter-killed belugas in the Mackenzie Delta, Canada, in 1984, the following parasites were documented: *Contracaecum* spp. (abundant – stomach and proximal small intestine), *Anisakis simplex* (rare in stomach), *Pharurus pallasii* (ears), *Hadwenius seymouri* (intestine) and *Leucastella arctica* (rectum). There was no evidence of negative effects on the animals (Wazura et al. 1986), so most likely represent typical fauna under normal conditions.

In a review paper by Measures (2001), lung worms (nematodes) described in belugas included: *Pharurus pallasii*, *Stenurus artomarinus, Halocercus monoceris* and possibly *Stenurus minor*. *Pharurus pallasii* are reported to be very common in some populations (85% to 88%) in Canada. “Lung worms” often not only parasitize the lungs, but also the sinuses, ears, auditory tubes, cardiovascular system, liver, and potentially the cranial vault. *Stenurus*, a nematode of the nasal sinuses and respiratory tract is commonly found in the auditory or eustachian tubes, middle and inner ears and cranial sinuses. These locations are all areas where disruption of function could contribute to strandings; however, they have been noted with little apparent effect on body condition and no firm evidence that they cause strandings (see Dailey 2001). Hunter-killed belugas in Pt. Hope and Pt. Lay, Alaska, have also been found to have similar lung worm lesions. Gross evidence of pulmonary nematode infection was observed in 56% (14 of 25) of beluga lungs examined in the two villages, with Pt. Hope animals far more often (85%), and more severely infected, than those in Pt. Lay (38%) (Woshner 2000). Examination of the sinuses, ear canals, tympanic bullae and cranial vault are, of course, necessary to diagnose these conditions. It is also unclear whether lung worms alone, or only when combined with secondary bacterial infections, were involved in strandings due to pneumonia, as pneumonia is a relatively common finding in stranded cetaceans. In hunter-killed belugas from the Churchill River basin in Manitoba, 89% (8 of 9) were parasitized with *P. pallasii* in the lung, accessory sinuses, ear canal and cerebral spinal fluid (Kenyon and Kenyon 1977), and in another study on St. Lawrence belugas, the prevalence in adults and juveniles was 88% and 72%, respectively (Houde et al. 2003). Infection is thought to occur when young beluga feed on infected prey items. The latter study also demonstrated that body condition indices did not correlate well with parasite intensities, suggesting that the presence of this parasite does not affect the animal’s general health or its ability to forage. In the St. Lawrence estuary, pneumonia was common and usually of parasitic origin (Martineau et al. 1994).

The arctic form of *Trichinella spiralis* (a parasitic nematode) is known to infect many northerly species of marine mammals including polar bear (*Ursus maritimus*), walrus, and to a lesser extent ringed seal (*Phoca hispida*) and belugas (Rausch 1970, Forbes 2000), and could potentially infect killer whales and gray whales (Mazzone 1987). The literature on "Arctic trichinosis" is dominated by reports of periodic outbreaks among Native people (Margolis et al. 1979); however, the effect of the organism on the host marine mammal is not known (Geraci and St. Aubin 1987). A single report in cetaceans was confirmed in an Arctic beluga (Brandly and Rausch 1950).

Large nematodes of the genus *Crassicauda* infect the cranial sinuses and may cause bone damage, while others injure renal blood vessels and mammary tissue to an extent that may impact population health. *Crassicauda* spp. have been recovered from a Stejneger’s beaked whale from the southeastern Bering sea (Shults et al. 1982). Similar parasites have been mentioned rarely in belugas from Pt. Lay (O'Hara and Woshner 2006). Similar lesions were not mentioned in reports from St. Lawrence belugas (Martineau et al. 1988, De Guise et al. 1995a) nor from Mackenzie River belugas, but are reported in eastern Canadian marine mammals as occurring in beluga and bowhead whales (*Balaena mysticetus*) (Vlasman and Campbell 2003).

With *Crassicauda boopsis*, a similar parasite in the large baleen whales, the proposed life cycle is that larva are ingested, and then have a somatic migration most likely up along the mesenteric arteries. The larvae enter the blood stream through the mesenteric artery lumen, go to the kidney and develop to adults. The tail of the adult extends into the renal calyxes, with release of eggs and larva into the urine. The body of the adult is associated with a marked inflammatory response, and the anterior end extends into the renal veins. The tissue response to the adult can result in obstruction of vessels draining the kidney, and can result in thrombi with thromboembolism to other organs, notably the lungs. It has been suggested that in large whales, *Crassicauda* may act as an important regulator of the population due to mortality in juvenile whales (Lambertson 1986, 1992). Although extensive damage and replacement to tissues have been associated with this parasite in some of the Cook Inlet belugas, it is unclear at this time whether this can result in functional damage to the kidney (Burek 1999a), or whether it is affecting the status of the population.

In approximately 80% of Cook Inlet belugas examined, the nematode *Crassicauda giliakiana* has been seen in the kidneys. It is hypothesized that when mesenteric calcifications are described in Cook Inlet belugas, these are most likely areas of damage to vessels and associated phlebitis/arteritis due to the parasite migration. There may also be aberrant adult development in these plaques since eggs are present in some of the plaques. Severe secondary effects of thromboembolism to other organs have not been observed to date in Cook Inlet whales, so it is most likely that under usual circumstances and levels of infestation, these animals live with this parasite with no clinical effect. It is possible though that with heavy infestation, there could be replacement of enough of the kidney (2/3 to 3/4 of the kidney tissue) to affect function or obstruction of urine outflow. This severity of infestation has not been observed in the small of number of carcasses examined to this date.

Anisakis simplex is a gastric nematode which often causes ulcers and more rarely gastric perforation. It is recorded commonly from the stomach of belugas in eastern Canada and Russia (Kleinenberg et al. 1964, Klinkhart 1966, DFO 1995). These infestations have not been considered to be extensive enough to have caused clinical signs, although *Anisakis* worms associated with stomach ulcers in St. Lawrence belugas were attributed as the cause of death in two animals (DFO 1995). In most cases in which the stomach was examined, there were either nematodes evident grossly, or an eosinophilic gastritis suggestive of parasitism. Stomach parasites (most likely *Contracaecum* or *Anisakis*) are often present in Cook Inlet belugas though have not been identified to genus or species.

Liver flukes have rarely been reported in belugas (Treshchev 1968) which may indicate the minimal role this parasite plays in the health of Cook Inlet belugas. The *Hadwenius* sp. trematodes have been described in the pancreas and pancreatic ducts in other beluga populations (Measures et al. 1995).

Lung worms appear to be common in Cook Inlet belugas, although this is primarily based upon histologic findings at this point. Where in 67% (6 of 9) of the animals in which the lung was examined histologically, inflammation suggestive of parasitic etiology was present, and one of these cases had intralesional parasites. More intensive gross examinations will most likely reveal the extent of lung worm infestation, and adult parasites are needed to identify the parasite to genus and species.

Trichinella has not been recorded within the Cook Inlet population of belugas; however, the risk and impact on Cook Inlet animals is probably low since the acquisition of *Trichinella* is usually through consumption of infected carcasses or amphipods or fish that have fed on infected carcasses (Forbes 2000).

Aberrant migrations of *Nasitrema* (Lewis and Berry 1988) and *Contracaecum osculatum* (Martin et al. 1970) have been reported in stranded and captive Pacific white-sided dolphins and could potentially affect Cook Inlet belugas on an individual basis that result in stranding. One Cook Inlet beluga demonstrated a grossly evident lesion in the liver which, histologically was due to a liver trematode. This trematode was not identified to species, but was most likely a *Campulid* type trematode.

Terrestrial mammals in the Cook Inlet estuary

The risk of diseases and parasites in belugas acquired from terrestrial wildlife found within the Cook Inlet estuary is possible but has not been studied. A review of pertinent wildlife diseases and parasites is given in Samuels et al. (2001) and Williams and Barker (2001), but a few diseases and parasites of particular significance are mentioned here. While many wildlife diseases are species-specific such as papillomaviruses (Sundberg et al. 2001), pathogens such as caliciviruses (Lenghaus et al. 2001) and *Salmonella* spp. (Minnette 1986, Mörner 2001) have the potential to infect belugas through exposure to bodily fluids and tissues from terrestrial species released into waters flowing into Cook Inlet and from fecal contamination of estuary waters by terrestrial animals such as domestic cats (Miller et al. 2002). Similarly, parasitic diseases are often species-specific in their host and life cycle preferences (Samuels et al. 2001); however, organisms such as *Giardia* spp., *Cryptosporidium* spp., and *Toxoplasma gondii* may originate in terrestrial species and empty into bodies of water, potentially infecting coastal marine mammal species (Hanni et al. 2003, Conrad et al. 2005, Gaydos et al. 2007).

Cook Inlet belugas may be exposed to a variety of pathogens that originate in fecal wastes from either humans or animals. They may be exposed not only indirectly from consumption of prey items contaminated with fecal microorganisms, but also directly to waterborne pathogens within their marine environment. Examples of organisms of concern are fecal coliform indicator bacteria such as *E. coli* and *Enterococcus* spp. which are used to assess the contribution of various host species to fecal pollution in water (Anderson et al. 2006). Alternatively, using other local marine mammal species as sentinels of ecosystem health, such as harbor seals, may be possible since they are relatively easier to handle and sample for epidemiologic studies (Thompson et al. 2002, Stoddard et al. 2005). Monitoring indicator pathogens (e.g., fecal, respiratory, serology) and parasites in harbor seals within Cook Inlet could provide better insight into the extent to which Cook Inlet belugas are exposed to pathogens and parasites from terrestrial wildlife and anthropogenic sources (see Section 3.8).

3.4. Ice Entrapment

Information on ice entrapment is provided in Hobbs et al. (2006).

3.5. Small Population Effects

Information on small population effects is provided in Hobbs et al. (2006). Also see revised extinction risk models in Section 5.

3.6. Fishery Interactions

Information on fishery interactions is provided in Hobbs et al. (2006).

3.7. Anthropogenic Sound

Information on anthropogenic sound is provided in Hobbs et al. (2006).

3.8. Pollution

The section on pollution has been expanded from the Hobbs et al. (2006) assessment to include more information on potential sources of pollutants in Cook Inlet and to review the detrimental health effects (both hypothesized and documented) that occur when these pollutants are found in high concentration in marine mammals.

Oil spills

Petroleum production, refining, and shipping in Cook Inlet present a possibility for oil and other hazardous substances to be spilled, and to affect the Cook Inlet beluga whale stock. The Outer Continental Shelf Environmental Assessment Program estimated that 3,339 m^3 (21,000 barrels) of oil were spilled in the inlet between 1965 and 1975, while 1,590 m^3 (10,000 barrels) were spilled from 1976 to 1979 (MMS 1996). In July 1987, the Tanker/Vessel (T/V) *Glacier Bay* struck an uncharted rock near Nikiski, Alaska, discharging an estimated 214.6 to 604.2 m^3 (1,350 to 3,800 barrels) of crude oil into Cook Inlet (USCG 1988). Belugas are found in the area where this spill occurred. In February 2005, T/V *Seabulk Pride* was torn from its moorings by heavy ice and tides in mid-Cook Inlet. Approximately 302.8 liters (80 gallons) of product spilled before the tanker was safely retrieved.

Contaminants

Contaminants are a concern for beluga whale health and subsistence use (Becker et al. 2000). The principal sources of pollution in the marine environment are: 1) direct discharges from industrial activities (petroleum, seafood processing, and ship ballast); 2) discharges from municipal wastewater treatment systems; 3) runoff from urban, mining, and agricultural areas; and 4) accidental spills or discharges of petroleum and other products (Moore et al. 2000). The Environmental Protection Agency regulates the discharges from these offshore platforms, which include drilling muds, drill cuttings, and production waters (the water phase of liquids pumped from oil wells). Drilling fluids (muds and cuttings) discharged into Cook Inlet average 89,000 barrels annually (244 barrels daily), containing several pollutants (MMS 1996). At the peak of infrastructure development, there were 15 offshore production facilities, three onshore treatment facilities, and approximately 368 km (230 miles) of undersea pipelines in upper Cook Inlet (MMS 1996).

Produced waters

In this section, the characteristics of the produced waters, as well as other discharges described, except drilling muds and cuttings, are based on information obtained during the Cook Inlet Discharge Monitoring Study conducted between April 10, 1988, and April 10, 1989 (EBASCO Environmental 1990a, b). These waters are part of the oil/gas/water mixture produced from oil wells, and contain a variety of dissolved substances. In oil drilling activities, chemicals are added to the fluids used in processes including: water flooding; well work-over, completion, and treatment; and the oil/water separation process. Before discharging into Cook Inlet, produced waters pass through separators to remove oil. The treatment process removes suspended oil particles from the wastewater, but the effluent contains dissolved hydrocarbons or those held in colloidal suspension (Neff and Douglas 1994).

Municipal waste and runoff

Cook Inlet is the major population center in Alaska, with a 2006 estimated population (U.S. Census Bureau) for the Anchorage Borough exceeding 280,000, the Matanuska-Susitna Borough at 77,174 and the Kenai Peninsula Borough at 51,350. Ten communities currently discharge treated municipal wastes into Cook Inlet. Wastewater entering these plants may contain a variety of organic and inorganic pollutants including: metals, nutrients, sediments, drugs, bacteria, and viruses. Wastewater from the Municipality of Anchorage, Nanwalek, Port Graham, Seldovia, and Tyonek receives only primary treatment, while wastewater from Homer, Kenai, and Palmer receives secondary treatment (NOAA 2003). Eagle River and Girdwood have modern tertiary treatment plants (Moore et al. 2000).

The Anchorage Wastewater Treatment Facility was built in 1972 and serves the entire Anchorage area. This facility has been upgraded twice: in 1982 to a 105,992 m^3 (28 million gallons) per day facility and in 1989 to a 219,554 m^3 (58 million gallons) per day facility. Plant influent is primarily of domestic origin, although an industrial component is included. The existing facility provides primary treatment for a design average flow of 219,554 m^3 (58 million gallons) per day and a maximum hourly flow of 582,953 m^3 (154 million gallons) per day. An average daily discharge of 136,275 m^3 (36 million gallons) per day was projected for 2005, with the exiting outfall discharged to Knik Arm. The outfall extends 245 m (804 ft) from shore and terminates as a trifurcated diffuser in water with a mean lower low water depth of 4.5 m (15 ft). The discharge depth of the diffuser during the typical 24 hour tidal cycle studies range from 3.5 to 12.3 m (11.5 ft to 40.5 ft). Existing treatment units provide screening, grit removal, sedimentation, skimming, and chlorination. Sludge from the primary clarifiers is thickened and dewatered. The dewatered sludge and skimmings are incinerated and the ash disposed in a sanitary landfill. Within the permit period, sludge volume is expected to increase above incinerator capacity. The excess sludge will be dewatered and disposed at the city’s landfill. Chlorinated primary effluent is discharged through a 305 cm (120-inch) diameter chlorine contact tunnel and then through a 213 cm (84-inch) diameter outfall to Cook Inlet.

The Municipality of Anchorage operates under a National Pollutant Discharge Elimination System (NPDES) storm water permit to discharge storm water to U.S. receiving waters. The Stormwater Phase I Rule (55 FR 47990; November 16, 1990) requires all operators of medium and large municipal separate storm sewer systems (MS4) to obtain a NPDES permit and develop a stormwater management program designed to prevent harmful pollutants from being washed by stormwater runoff into the MS4 (or from being dumped directly into the MS4), then discharged from the MS4 into local water bodies.

The Municipality of Anchorage's (MOA) NPDES stormwater permit (AKS05255) is a 5-year term permit to discharge stormwater to U.S. receiving waters issued jointly to the MOA and the Alaska Department of Transportation and Public Facilities by the U.S. EPA Region 10. An annual report to EPA is required by the permit (MOA 2006). The stormwater NPDES program addresses many aspects of stormwater management. The 2005 report (MOA 2006) addresses coordination and education, land use policy, new development management, construction site runoff management, flood plain management, street maintenance, and best management practices for pollutant sources and controls, illicit discharge management, industrial discharge management, pesticides management, pathogens management, watershed mapping, hydrology, water quality, ecology and bioassessment, and watershed characterization.

Contaminant effects

The detrimental effects of persistent organic pollutants (POPs) in marine mammals include, but are not limited to, reproductive disorders (Helle et al. 1976, Béland et al. 1993, Martineau et al. 1994), immune system depression (De Guise et al. 1995b, de Swart 1995, Ross 1995), and subsequent greater risk of infection (Jepson et al. 1999, 2005; Hall et al. 2006); however, the effects on the health of the animal are often difficult to discern. For instance, no experimental studies on the reproductive effects of contaminants in cetaceans have been performed, and indirect evidence for the association is lacking (O'Hara and O'Shea 2001). Nevertheless, organochlorines are suspected to be broadly affecting the health and reproduction of cetaceans globally through disruption of endocrine receptors (Colborn and Smolen 1996). Toxic elements such as cadmium and mercury are known to accumulate in marine mammal tissues and are most likely a reflection of diet in cetaceans (Becker et al. 2001); however, accumulation of some elements such as zinc have been associated with poor health in harbor porpoises (Das et al. 2004, Pierce et al. 2007).

The evidence for reproductive disorders due to contaminants in belugas is strongly hypothesized, but the evidence is not yet convincing. Although no cause-and-effect relationship has been established between contaminants and reproductive disorders in belugas, researchers have suspected that elevated organochlorines have impacted reproduction in St. Lawrence whales (Béland et al. 1993, Martineau et al. 1994). In the study by Béland et al. (1993) that investigated the potential health and reproductive effects of contaminants in St. Lawrence belugas, the authors observed the population size to be stable, resulting from low calf production and/or decreased survival to adulthood. The whales also had a high prevalence (40%) of tumors, of which 53% occurred in the digestive system, 45% in the mammary glands of adult females, and 11% in other glandular tissues. No such lesions were found in Arctic belugas necropsied as a comparison group.

Attempts at linking disease epizootics and elevated contaminant levels in marine mammals have proven challenging given the relatively small number of studies that have investigated the potential impact of contaminants on the health of marine mammals, however, indirect evidence for their negative effects has been documented (O’Hara and O’Shea 2001). In the United Kingdom, several studies have investigated the potential impact of contaminants on marine mammal immunity and health status (Jepson et al. 1999, 2005; Hall et al. 2006; Pierce et al. 2007), while more direct studies of contaminants and immunosuppression have been performed in harbor seals (de Swart et al. 1996, Ross et al. 1996). Levels of contaminants in blubber of belugas can be compared to the threshold levels established for other species of marine mammals to determine the level of risk posed by exposure to these compounds. However, caution is warranted in these evaluations as the threshold effects levels were determined for non-beluga species, and belugas may be more or less susceptible to the toxicological effects of these compounds.

St. Lawrence Estuary whales have a surprisingly high incidence of a variety of neoplastic conditions, estimated to be about 163 cases/100,000 belugas (Martineau et al. 1994, 1999, 2002;

Theriault et al. 2002) with 24% in mature animals, possibly related to the high levels of persistent organic pollutants (Martineau et al. 1994). Examples of neoplasia in belugas include gastric papilloma (due to papillomavirus), hemangioma of the urinary bladder, possible splenic tumor, granulosa cell tumor, intrapulmonary lipoma, transitional cell carcinoma, salivary gland adenocarcinoma, mammary gland adenocarcinoma, hepatocellular carcinoma, intestinal adenocarcinomas and poorly differentiated malignant tumors (Martineau et al. 1988, 1994; De Guise et al. 1994). Other lesions in the St. Lawrence animals include cystic degeneration of the adrenal glands, again possibly related to POPs (De Guise et al. 1995a). Compared to St. Lawrence belugas, relatively low numbers of neoplasms are reported in marine mammals, which may be due to unobserved marine mammal mortalities, early mortality before reaching old age, and delay in collecting samples, rendering diagnosis of neoplasia difficult (Newman and Smith 2006).

The Cook Inlet beluga population has much lower concentrations of PCBs and chlorinated pesticides than that reported for other Arctic beluga populations, and may represent a less significant health risk than for other populations; however, in combination with other stressors such as disease, parasites, low prey availability, noise and other anthropogenic factors, contaminants may yet compromise Cook Inlet whale health (Becker et al. 2001). Trace elements and heavy metals have been investigated in several populations of belugas, including the Cook Inlet population (Becker et al. 2001).

Contaminant levels in Cook Inlet belugas are low relative to other beluga populations, however, the risk of an epizootic combined with other stressors could impact the long-term viability of the Cook Inlet population. The potential health effect of anthropogenic pollutants from industrial activities, municipal wastewater, mining and agricultural runoff , and spills have the potential to compromise, individually and in combination with other stressors, the reproductive success and long-term viability of the Cook Inlet beluga population (Moore et al. 2000). Correlations between levels of contaminants and actual health effects should be closely studied in order to determine the significance of these levels.

Although several anthropogenic factors that may potentially injure or have detrimental effects on the health of belugas are discussed elsewhere in this Status Review, a few warrant further discussion. Since Cook Inlet belugas inhabit near-shore marine ecosystems, they are vulnerable to human development. Coastal human development may give rise to protozoal and bacterial organisms, harmful algal blooms and antibiotic resistance (Johnson et al. 1998, Scholin et al. 2000, Miller et al. 2002, Wong 2002, Blackburn 2003).

Water quality is a concern for wildlife, especially since cetaceans may be vulnerable to antibiotic resistant strains of bacteria (Stoddard et al. 2005), terrestrial pathogens (Minnette 1986, Higgins 2000), and biochemical markers (Kannan et al. 2005). When considering the development of water quality standards for coastal cetaceans, the difficulty in obtaining good scientific data to support water quality guidelines is due to the logistics of collecting data from wild populations to develop cetacean-dose response relationships (Thompson 2007). As mentioned in Section 3.3, monitoring indicator pathogens (e.g., fecal, respiratory, serology) and parasites in harbor seals within Cook Inlet could provide better insight into the extent to which Cook Inlet belugas are exposed to pathogens and parasites from terrestrial wildlife and anthropogenic sources. Eventually, these data could aid with development of water quality standards for belugas within Cook Inlet.

3.9. Ship Strikes

Information on ship strikes is provided in Hobbs et al. (2006).

3.10. Subsistence Hunting

Since publication of the 2006 Status Review (Hobbs et al. 2006), NMFS entered into only one co-management agreement to hunt one beluga in 2006, but the hunt was not successful. No co-management agreement and, therefore, no hunt occurred in 2007.

3.11. Research

Information on research is provided in Hobbs et al. (2006).

3.12. Summary of Potential Risk Factors

The potential risk factors and their possible effects on Cook Inlet belugas at the individual and population level are summarized in Table 3.12-1. Factors were grouped into the categories of infectious diseases and pathogens, environmental stresses, toxicants, predators, and anthropogenic stresses.

Table 3.12-1. Potential risk factors for Cook Inlet belugas and possible effects at the individual and population level. Factors are grouped into the categories of infectious diseases and pathogens, environmental stresses, toxicants, predators, and anthropogenic stresses.

Threat	Individual effect	Population effect	References/Notes
INFECTIOUS DISEASES AND PATHOGENS			
Viruses			
Herpesvirus	Skin lesions Encephalitis	Potential for large-scale mortality	Van Bressem et al. 1999 Kennedy et al. 1992
Morbillivirus	Bronchial pneumonia; alveolitis	Epizootic; high mortality	Van Bressem et al. 2001
Bacteria			
Bartonella	Endocarditis	Unknown; potential for mortality	Has been documented in Alaska sea otters (high morbidity and mortality) – currently under investigation
Brucella	Potential for abortion; brain lesions	Negative impact on reproduction (decreased fecundity/reproductive success); decreased population viability	Jepson et al. 1997, Miller et al. 2002

Threat	Individual effect	Population effect	References/Notes
Antibiotic resistant / non-adapted pathogenic bacteria	Mortality?	Unknown, not studied to date.	Johnson et al. 1998, Wong 2002, Blackburn 2003; untreated sewage released into Cook Inlet from Anchorage and surrounding area.
Parasites			
Protozoa (Giardia Crypto-sporidium Toxoplasma, Sarcocystis)	Severe diarrhea and dehydration (Giardia and Crypto); encephalitis (Toxo and Sarco)	Morbidity	Olson et al. 1997, Hughes-Hanks et al. 2005
Crassicauda giliakiana	Obstruction of renal vessels; potential renal function compromise (?)	High morbidity; potential for high mortality (?)	Burek 1999a; potential for high mortality (see Lambertson 1986, 1992)
ENVIRONMENTAL STRESSES			
Decreased prey availability and/or quality	Increased susceptibility to disease; increased morbidity and mortality	High morbidity; increased susceptibility to disease; decreased population viability	Several animals noted with thin blubber layer at necropsy in late summer (K. Burek, AVPS, unpubl. data)
Increased ice cover, fast ice formation	Entrapment; injury; death	Mass entrapment, mortality	No evidence in Cook Inlet, reported in Arctic waters (Heide-Jørgensen et al. 2002, Armstrong 1985)
TOXICANTS			
Biotoxins	mortality; seizures		Scholin et al. 2000, Silvagni et al. 2005; climate change issues; has not been investigated in Cook Inlet belugas and surrounding area
Organochlorines	Endocrine, immune function, reproductive, and bone quality disruption		Ross 1995, Becker et al. 2001, Hall et al. 2006; appear to be less of a problem in Cook Inlet belugas

Threat	Individual effect	Population effect	References/Notes
Heavy metals	Unknown		Becker et al. 2000, Pierce et al. 2007; appear to be low in Cook Inlet belugas
PAHs	Mortality?	Low reproductive success?	Becker et al. 2001; drilling muds
PREDATORS			
Killer whales, humans (subsistence hunting)	Mortality; stranding	Usually in balance in a normal situation. Killer whales possible prey switching in Kodiak to Aleutians.	Mahoney and Shelden 2000, Shelden et al. 2003; killer whale issues for sea otters, Steller sea lions, harbor and northern fur seals
ANTHROPOGENIC STRESSES			
Blast effects/ construction, drilling/seismic	Injury, stranding, abandoning habitat?		Funk et al. 2005, FHWA and KABATA 2006, Blackwell and Greene 2002
Oil spills	Mortality, injury?		MMS 1996, Loughlin 1994
Ship strikes	Potential injuries/mortality as ports developed and vessel traffic increases		Burek 1999b; NMFS, unpublished data
Fishery Interactions, entanglements	Mortality, injury		Murray and Fay 1979, Burns and Seaman 1986

In order to begin to determine factors that may affect the recovery of Cook Inlet belugas, a population comparison study will be undertaken focusing on belugas in Bristol Bay. The Bristol Bay population is located during the summer months about 1,500 km away by sea and separated from Cook Inlet by the Alaska Peninsula that extends 3 degrees of latitude south of the southern limit of the Bristol Bay beluga population. This population, in comparison to Cook Inlet, is increasing at about 4.5% annually and numbers about 2,000 animals (L. Lowry, University of Alaska Fairbanks, pers. comm.). Two field seasons are planned for 2008 in May and September and additional years will be added as funding allows. Points of comparison will include: movements and dive behavior, habitat use, relative dependence on summer salmon feeding for total annual calories, annual caloric requirements, and types and prevalence of disease and parasites.

4. DETERMINATION OF DPS

4.1. ESA Discreteness and Significance

Joint NOAA/USFWS policy defines a population to be a DPS if it is both discrete and significant relative to the taxon to which it belongs (61 FR 4722, February 7, 1996). Under the policy, a population may be considered discrete if it satisfies one of the following conditions:

- It is markedly separated from other populations of the same taxon as a consequence of physical, physiological, ecological, or behavioral factors. Quantitative measures of genetic or morphological discontinuity may provide evidence of this separation.
- It is delimited by international governmental boundaries within which differences in control of exploitation, management of habitat, conservation status, or regulatory mechanisms exist that are significant in light of Section 4(a)(1)(D) of the ESA.

Data relevant to the distinctiveness question include the physical, ecological, behavioral, and genetic data that are presented in Section 2 and summarized below. If a population segment is considered discrete, NMFS must then consider whether the discrete segment is “significant” to the taxon to which it belongs. A discrete population segment needs to satisfy only one of the following criteria to be considered significant:

- persistence of the discrete segment in an ecological setting unusual or unique for the taxon,
- evidence that loss of the discrete segment would result in a significant gap in the range of the taxon,
- evidence that the discrete segment represents the only surviving natural occurrence of a taxon that may be more abundant elsewhere as an introduced population outside its historical range, or,

- evidence that the discrete segment differs markedly from other populations of the species in its genetic characteristics.

The policy also allows for consideration of other factors if they are appropriate to the biology or ecology of the species. Data relevant to the significance question include the morphological, ecological, behavioral, and genetic data presented in Section 2 and summarized below.

4.2. Evaluation of ESA Discreteness

The evaluation has not changed since Hobbs et al. (2006). It is not clear from available data whether the group of belugas found in Yakutat Bay is isolated from Cook Inlet.

4.3. Evaluation of ESA Significance

The evaluation has not changed since Hobbs et al. (2006).

5. ASSESSMENT OF EXTINCTION RISK

5.1. Population Viability Analysis

A detailed population viability analysis (PVA) model was developed for the Cook Inlet beluga population in Hobbs et al. (2006) to assess the extinction risks faced by this small population. The model included immature and mature phases of both sexes (i.e., age- and sex-structured) and focused on behavior of a declining population at sizes less than 500 belugas. Small population effects were taken into account by examining survival and fecundity under a range of scenarios that considered demographic stochasticity, harvest, density dependence, Allee effects (Allee et al. 1949), constant mortality effects (e.g., predations), and unusual mortality events (e.g., catastrophes). In response to reviews, environmental variability was added to the range of scenarios, a likelihood model using only 1999 to 2007 abundance estimates, and a likelihood model assuming over half of the small gray animals under 10 years of age are missed during the aerial surveys. All models have been updated to include abundance estimates through 2007.

5.1.1. Methods

To foresee the growth or decline of this beluga population in the future, a PVA model was developed using life history and population parameters estimated for this and other beluga populations (see Table 2.3.3-1 in Section 2.3.3). In addition to the selection of parameters, mechanisms affecting small populations (as described above) and time lags inherent in long-lived populations (which can result in a delayed response to changes in mortality probabilities) were also considered. One example of a time lag is the period from birth to reaching sexual maturity, which may result in a delayed response of the population growth as the number of adult belugas in the Cook Inlet population rebounds from the effect of the unregulated harvest. Litzky (2001) modeled the fraction of mature animals in the population and found that after cessation of the harvest it took 5-7 years for the adult to juvenile ratio to recover. To account for the time lag from birth to sexual maturity and the preference of hunters for adult animals, an age-structured model was used with adult age classes lumped together. Females and males were also modeled separately to incorporate sex-structure into the model and allow for unequal harvest of males and females.

Demographic stochasticity, the random variations in the number of individuals that happen to die or reproduce in a given year (Begon et al. 1996:927), was included in the model projection from one year to the next. To this extent, survival from year to year and births each year were modeled using a binomial draw. To model the harvest and the stochastic effects of injuring or killing a whale but not retrieving it (struck and lost), we used a negative binomial draw with the reported landings as successes and the probability of success as the probability of landing a struck whale. The resulting stochastic age- and sex-structured model was used to model the current population and project the possible outcomes.

The model was fit to the available abundance estimates for the years 1994 to 2007 (Table 5.1.1-1) using Bayesian statistical methods. The abundance of the Cook Inlet beluga population and subsistence harvest removals from this population were estimated for each year between 1994 and 2007 (Table 5.1.1-1, Fig. 2.3.1-1). Limited information is available to determine the behavior of this population during periods prior to 1994, including the original or pristine size of this population and its sustainable harvest level. As mentioned in Section 2.3.1., NMFS has adopted 1,300 as the value for carrying capacity (*K*) to be used for management purposes (65 FR 34590, May 31, 2000). Finally, although *K* is included as a parameter, the purpose of this exercise was to model the behavior of the population at sizes below 500 animals. By relaxing the constraint of no population growth or decline at *K*, a greater variety of possible trajectories were available to the model within the range of interest.

Table 5.1.1-1. Time series used in the Bayesian analysis. Median aerial counts are the median of all observer counts from complete surveys of upper Cook Inlet. Estimated abundance was calculated from observer and video data. Harvest landings and struck and lost data were from Mahoney and Shelden (2000) and NMFS Alaska Region Office, unpublished data. Where conflicting sources occur, all are listed with the numbers used in the model in bold. Note that killed but lost are included with the struck and lost.

Year	Median Aerial Count	Estimated Abundance	Abundance CV	Harvest Landings (struck and lost)
1994	281	653	0.430	**19(2)**
1995	324	491	0.440	60(14), **52(22)**, 42(26)
1996	307	594	0.280	**49(49-98)**
1997	264	440	0.140	35(30-40), **35(35)**
1998	193	347	0.290	**21(21)**
1999	217	367	0.140	**0(0)**
2000	184	435	0.230	**0(0)**
2001	211	386	0.087	**1(0)**
2002	192	313	0.120	**1(0)**
2003	174	357	0.110	**0(0)**
2004	187	366	0.200	**0(0)**
2005	187	278	0.180	**2(0)**
2006	150	302	0.160	**0(0)**
2007	224	375	0.210	**0(0)**

It is important to note that the abundance data used in the likelihood function for fitting the model resulted from aerial surveys conducted each June from 1994 to 2007 that used essentially the same methods through the entire time series (Hobbs et al. 2000a, b). During a 2 week period in early June, 3 to 7 surveys of the upper inlet and one survey of the lower inlet were conducted.

During each survey the entire coastline to approximately 1 km off shore and all river mouths are surveyed. Transects across the inlet are flown as well (Rugh et al. 2000, 2005a, b). When a group of whales is encountered it is circled in a racetrack pattern 4 to 16 times to allow multiple counts by researchers and the collection of video data.

Two video cameras are used, one to collect a view of the entire group for counting and a second to collect a zoomed in view of a portion of the group to estimate the fraction of missed animals. The video data are the primary source of group size estimates. Useable video sequences are reviewed frame by frame and all individuals are counted. The zoomed video is also reviewed frame by frame and individuals in each zoomed frame are accounted for in each frame of the counting video. Those not found in the counting video are included in the fraction missed. The video counts are also corrected to account for animals that were under water during the video sequence using dive data from radio tags (Lerczak et al. 2000). For groups with no usable video, a correction for the researcher counts is developed by comparing researcher counts of groups to video group size estimates (Hobbs et al. 2000a). Group size estimates for each survey of the upper inlet are summed to get several independent estimates of abundance. Another correction includes data from paired, independent observers to estimate the fraction of whales missed when an observer does not see a group.

While several methods are employed to fully account for beluga missed within the survey area, it is not possible to correct for a large group that may have moved out of the survey area. As a final check, the flight paths of survey days with unusually low estimates are reviewed to determine if a group seen on other survey days could have been missed. If this is the case, then these survey days are discarded. The remaining survey days are averaged to complete the abundance estimate. While the survey methodology has remained the same, the video cameras have not, and over the period from 1995 to 2007 several upgrades have occurred. There is concern that the fraction of small, gray animals missed may have declined through the time series (this issue was raised during the CIE review), therefore, this is now tested in the model analysis.

Life history parameters of particular interest for modeling purposes were: survival probability, birth interval, age at first birth, gestation period, and lactation period. With the exception of survival probability, life history sample sizes from Cook Inlet were not sufficient to estimate the other model parameters. These data were instead obtained from the available literature on several other beluga populations (see Table 2.3.3-1 in Section 2.3.3). Upper and lower bounds for the model parameters are described below.

Survival data for Cook Inlet belugas consist of annual summaries of beach-cast and floating carcasses reported to the NMFS Alaska Regional Office and consequently represents a minimum estimate of mortality for this population. From 1999 to 2005, years in which a limited harvest occurred (Table 5.1.1-1), an average of 12 mortalities were reported each year (Vos and Shelden 2005) during a time when the population size averaged around 350 animals. This provided an estimated annual survival probability of 0.97/year which was used as the upper bound for the model. From the literature, survival probabilities have been estimated as low as 0.84/year but most were above 0.90/year (Table 2.3.3-1). For modeling purposes, values as low as 0.80/year were considered; however, values below 0.85/year were not consistent with other parameters in the model so 0.85/year was the effective lower bound for the annual survival probability.

The birth interval for the average mature female in most beluga populations was thought to be 3 to 4 years although it may be as short as 2 years for younger adults (Table 2.3.3-1). The inverse of the birth interval is the annual probability of giving birth (between 0.25 and 0.33) for each adult female. To keep the model simple, an average value was used for all adult females including senescent females. A reduced birth probability in the Cook Inlet population resulting from external effects such as pollution or poor fish runs suggests a lower value than 0.25 is possible so the interval 0.05 to 0.33 was used in the model.

Female belugas reach sexual maturity between the ages of 8 and 15 years (Table 2.3.3-1). The gestation period lasts about 13 months, so age at first birth was set to start at 10 years in the model. In Hobbs et al. (2006) this had been set to 5 years which was indicated by life history studies (Table 2 in Hobbs et al. (2006)) of several beluga populations. However, the ages in these studies were determined by counting GLGs in tooth sections. Previously, the accepted practice interpreted 2 GLGs as indicating 1 year of age so 8-9 GLGs were interpreted as 4 years old (see Section 2.1.1). Recent research has shown that each GLG should be interpreted as one year resulting in a doubling of the age at first birth when the life history data are reinterpreted in light of this new information. The age of first birth is of importance for the model and is referred to hereafter as the age of maturity or age at first birth. The lactation period typically lasts longer than one year so calf survival was modeled as dependent on the survival of the mother during the first year after birth. Survival probabilities and age at maturity also have been estimated for males. However, these estimates were not sufficiently different from those for females to require additional parameters in the model.

At about the time a beluga reaches maturity, its skin changes from gray to white (Burns and Seaman 1986). Hunters have stated that they focus their hunting effort on white adult animals so vulnerability to harvest was set in the model to coincide with the age at first birth. While not all animals are mature before they turn completely white this was considered a reasonable approximation to simplify the model.

The population was projected as:

$$
\begin{array}{ll}
f_{0,t} = B\left(f_{mat,t}, {}^{b_t}\!/_{2} \right) & m_{0,t} = B\left(f_{mat,t}, {}^{b_t}\!/_{2} \right) \\[2ex]
f_{1,t+1} = B\left(f_{0,t}, s_t^{\,2} \dfrac{f_{mat,t} - H_{f,t}}{f_{mat,t}} \right) & m_{1,t+1} = B\left(m_{0,t}, s_t^{\,2} \dfrac{f_{mat,t} - H_{f,t}}{f_{mat,t}} \right) \qquad\qquad (1) \\[2ex]
f_{a+1,t+1} = B(f_{a,t}, s_t) & m_{a+1,t+1} = B(m_{a,t}, s_t) \\
\qquad \text{for } a = 1 \text{ to } (a_{mat}\text{-}2) & \qquad \text{for } a = 1 \text{ to } (a_{mat}\text{-}2) \\[1ex]
f_{mat,t+1} = B(f_{mat,t} - H_{f,t} + f_{a_{mat}-1,t}, s_t) & m_{mat,t+1} = B(m_{mat,t} - H_{m,t} + m_{a_{mat}-1,t}, s_t),
\end{array}
$$

where,

$f_{a,t}, m_{a,t}$ is the number of females and males, respectively, of age *a* at the beginning of year *t*;

$f_{mat,t}, m_{mat,t}$ is the number of mature females and mature males, respectively, at the beginning of year t;

$B(x, p)$ is a binomial random variable with x trials and p probability of success;

s_t is the probability of an individual in year t surviving to year $t+1$;

b_t is the probability of a mature female giving birth to a live offspring in year t;

a_{mat} is the age of maturity or the age at which a female could first give birth; and

$H_{f,t}$, $H_{m,t}$ is harvest mortality (both landings and struck and lost) of females and males, respectively, in year t.

Harvest mortality was modeled as the sum of the landed whales plus estimates for those struck and lost. During the years 1995-1998 (Table 5.1.1-1), landings were fairly well documented and struck and lost was estimated as between one-half and two whales lost for each whale landed. Or, in other words, for each beluga killed during harvest activities there was a probability between one-third and two-thirds that it would be landed. For the model, this uncertainty in the level of struck and lost for the years 1979 to 1998 was accounted for by drawing from a negative binomial distribution with the landings as the number of successes and the probability of success for each realization of the model drawn from a uniform distribution between one-third and two-thirds (U[1/3, 2/3]). For the years 1999 and later, where harvests have been regulated, the number of struck and lost was set to zero. It was assumed that constant landings occurred from 1979 through 1993 and were similar to the number of landings reported in 1994, so landings for these years were drawn from a uniform distribution between 10 and 30 belugas (U[10, 30]). For the years 1994 to 2007, actual landings (Table 5.1.1-1) were used in the model. For the purposes of the model, no harvest occurred after 2007. Data on the sex of whales killed in the hunt are sparse. From 1992 to 1998, 19 male and 15 female belugas were documented during the harvests (Mahoney and Shelden 2000) corresponding with approximately 55% probability that an animal landed in the harvest was male. Variability in this probability was accounted for by drawing a value for each model realization from a triangular distribution between 0.40 and 0.70 with the peak at 0.55 (TR[0.40,0.55,0.70]). This approximated the beta distribution of relative probabilities for this parameter, without the tails.

The harvest mortality model is

$$H_t = CIBL_t + NB\,(CIBL_t, \mathbf{Pr}(Landing\ Success))$$
$$H_{m,t} = B(H_t, \mathbf{Pr}(Harvest\ Male)) \qquad (2)$$
$$H_{f,t} = H_t - H_{m,t}\ ,$$

where,

H_t is total harvest mortality (both landings and struck and lost) in year t;

$CIBL_t$ is the recorded harvest landings for 1994 to 2007 and a constant harvest landing per year for 1979 through 1993 in year t;

$NB(x, p)$ is a negative binomial random variable of failures (struck and lost) for x successes (landings) and p probability of success for the years 1979 through 1998, after 1998 this is always zero;

Pr(*Landing Success*) is the probability of landing an animal killed in the hunt, drawn from U[1/3,2/3] for years 1979 to 1998, for 1999 and after this is always 1, so all animals struck are landed and, therefore, struck and lost is zero; and

Pr(*Harvest Male*) is the probability that an animal taken in the harvest is a male, drawn from TR[0.40,0.55,0.70].

To allow for density dependence in the annual growth multiplier (ϕ) (discussed in greater detail below), both survival (s) and fecundity (b) in Equation 1 were made density dependent with the following equation:

$$s_t = \left[s_0 - (s_0 - s_k)\left(\frac{N_t}{K}\right)^z \right](s_e) \qquad (3)$$

$$b_t = \left[b_0 - (b_0 - b_k)\left(\frac{N_t}{K}\right)^z \right](b_e)\ ,$$

where,

s_0, s_k, b_0, b_k are the values for *s* and *b* when the size of the population is close to 0 and at *K*, respectively;

s_e, b_e are multipliers for *s* and *b* that reduce survival or fecundity independent of density;

$N_t = \sum_{all\ ages} f_{a,t} + m_{a,t}$ is the size of the population at time *t*;

K = the carrying capacity (1,300); and

z = a shape parameter (2.39)

The annual survival probability and annual fecundity probability consisted of three components: a compensatory density-dependent survival or fecundity (Equation 3: within the square brackets); a density-independent component (s_e, b_e); and a modifier such as Allee effects, unusual mortality events, variable environment, and constant mortality effects which will be discussed later. The density-dependent component used the discrete logistic formulation to decrease the probability of survival and probability of giving birth as the population increases. Parameters were chosen so that the annual growth multiplier (ϕ) of 1.02 to 1.06 (i.e., annual per capita increase between 2% and 6%; cf. Wade and Angliss 1997) fell between these values when the population was small and declined to 1.00 (zero growth) when the population reached carrying capacity. The density-independent components (s_e, b_e) can be set to 1 to model a healthy population with annual growth between 2% and 6% or they can be set to values less than 1 to model processes that decrease survival or fecundity for each individual such as contaminants or ship strikes.

Choosing efficient and still uninformative prior distributions for the parameters of the annual fecundity probability and annual survival probability requires a bit of tuning to the model in question and the parameter constraints. If we treat fecundity (*b*) and survival (*s*) as constant parameters and consider the deterministic projection of the expected values of the abundance with harvest at zero, we have a recursion model in expected births by year. The characteristic equation for this recursion model can be written as:

$$1=\frac{bs^{a_{mat}+1}\phi^{-a_{mat}}}{2(1-s\phi^{-1})} \quad , \qquad (4)$$

which then yields an equation for *b* if *s* and ϕ are known:

$$b=\frac{2(1-s\phi^{-1})}{s^{a_{mat}+1}\phi^{-a_{mat}}} \quad , \qquad (5)$$

where,
ϕ is the annual growth multiplier for an expected stable age distribution.

The density-dependent components (Equation 3: square brackets) represent the basic model for a healthy cetacean population with an annual growth multiplier of 1.02 to 1.06. To create a uniform prior distribution for the annual growth multiplier, ϕ_0 was drawn at random from U[1.02, 1.06]. At *K*, ϕ_K is, of course, 1.00, indicating the population is no longer growing upon reaching carrying capacity. The upper bounds for s_0 and b_0 were 0.97 and 0.33, respectively, and both s_K and b_K were nonnegative. Values for s_0 were then drawn from U[ϕ_0-0.10, 0.97] and s_K from U[s_0-0.9(ϕ_0-1), s_0] which allow anywhere from none to all of the density dependence to effect survival while avoiding a significant range of useless parameter space. Equation 5 was then solved for b_0 and b_K. If b_0 and b_K fell in the intervals [0.05, 0.33] and [0.0, b_0], respectively, then the parameter set was retained; otherwise it was discarded and new values for s_0 and s_K were drawn. This approach allowed the density dependence to entirely affect survival or fecundity or any ratio of the two while maintaining a uniform prior for ϕ_0.

The annual growth multiplier for a healthy cetacean population described in the previous paragraph requires that s_e and b_e are set to 1. To allow a full range of annual growth multipliers, to model populations that may be in decline, we included cases where s_e and b_e

were less than 1. To create a uniform prior distribution for the annual growth multiplier, ϕ_0 was drawn at random from U[0.94, 1.06], where the annual per capita change ranged from -6% to +6%. In these cases, ϕ_K is not necessarily 1.00 and instead was chosen from U[ϕ_0-0.06, minimum(ϕ_0-0.02, 1.00)] so that the annual per capita change would be 0% or < 0% when the population was at *K*. Density-independent components, s_e and b_e, were multiplied through the density-dependent portion of Equation 3 (square brackets) to form composite parameters: $s_0 s_e$, $s_K s_e$, $b_0 b_e$ and $b_K b_e$. As in the Healthy Population model (where s_0 and b_0 were 0.97 and 0.33, respectively, and both s_K and b_K were non-negative), the upper bounds for the composite parameters $s_0 s_e$ and $b_0 b_e$ were also set to 0.97 and 0.33, respectively, and both $s_K s_e$ and $b_K b_e$ were non-negative. Values for $s_0 s_e$ were then drawn from U[0.85, 0.97] and $s_K s_e$ from U[$s_0 s_e$-0.90(ϕ_0-ϕ_K), $s_0 s_e$] which allowed all of the density dependence to affect survival. Equation 5 was then solved for $b_0 b_e$ and $b_K b_e$. If $b_0 b_e$ and $b_K b_e$ fell in the intervals [0.05, 0.33] and [0.0, $b_0 b_e$], respectively, then $s_0 s_e$ and $s_K s_e$ were retained, otherwise they were discarded and a new set was drawn. Note that parameters must be drawn as composites in this model, which we will refer to as the Baseline model, to maintain a uniform and uninformative prior distribution. If the parameters are drawn individually and then multiplied together the resulting prior distribution would be peaked and highly informative.

Modifiers to survival and fecundity were intended to model specific processes. These processes included a constant mortality effect and a stochastic or unusual mortality event to modify survival, and an Allee effect to modify fecundity. Environmental variability was included in both survival and fecundity as a correlated normal random deviate. These were included in the model by rewriting Equation 3 as:

$$s_t = \left[s_0 - (s_0 - s_k) \left[\left(\frac{N_t}{K} \right)^z + \varepsilon_t \right] \right] s_e \left(\frac{N_t}{N_t + C} \right) \{ 1 - M_e B(1, P_{Me}) \}, \tag{6}$$

$$b_t = \left[b_0 - (b_0 - b_k) \left[\left(\frac{N_t}{K} \right)^z + \varepsilon_t \right] \right] b_e \left(\frac{f_{mat,t}}{f_{mat,t} + A} \right) \left(\frac{m_{mat,t}}{m_{mat,t} + A} \right),$$

$$\varepsilon_t = \rho \varepsilon_{t-1} + \sigma \sqrt{1 - \rho^2} z_t$$

where,

C is the parameter of the constant mortality effect and represents expected annual mortalities;

M_e is the individual probability of mortality during an unusual mortality event;

P_{Me} is the probability of an unusual mortality event occurring in a given year;

A is the Allee effect parameter,

ε_t is a stationary, correlated, random environmental deviation with mean = 0, variance = σ^2 and correlation = ρ (Morris and Doak 2002:139).

z_t is a normal random deviate with mean = 0 and variance = 1.

Note that these processes were formulated so that if any of these parameters were zero then the corresponding effect does not modify survival or fecundity. The constant mortality effect was intended to model mortality resulting from annual killer whale predation in which the killer whales were thought to take a number of belugas proportional to their own needs regardless of the size of the beluga population. It could also model illegal harvest if that harvest remained constant from year to year regardless of the population size. The values for *C* represent the average mortalities per year due to killer whale predation or some other constant mortality. Shelden et al. (2003) estimate an average of one observed predation mortality per year. This was considered a minimum since unobserved predation events may also be occurring. Values considered for the parameter (*C*) were 0, 1, 2, and 5, with zero mortalities per year occurring when the constant mortality effect was absent and five mortalities per year included as an extreme example.

The unusual mortality event (P_{Me} within the curly brackets of Equation 6) models random events such as mass stranding mortality. In this formulation, it included a mortality fraction and a binomial draw which determined whether or not an event occurred that year. The expected mortality from this source was the product of the mortality fraction and the probability of occurrence so that for the values used here ($M_e = 0.20$ and $P_{Me} = 0.05$), the expected or average annual unusual mortality event when it was included was 0.01 (i.e., an increase of average annual mortality of 1% of the population).

The Allee effect (Allee et al. 1949) is thought to occur in small populations where small numbers of adult females and adult males results in reduced mating opportunities or reduced variety of mate selection with consequent declining fecundity. Although other mechanisms affecting both fecundity and survival have been included under the definition of the Allee effect (Courchamp et al. 1999, Stephens and Sutherland 1999), for the purposes of this modeling exercise, its effect was applied to fecundity only (A in Equation 6). Note that in the formulation above the birth probability is zero when either sex is not present. The Allee parameter was set to 0.5 or 0.0 depending on presence or absence of the effect, respectively. There is little information on which to base a choice of this parameter instead it was tuned to affect the population when there were fewer than 50 whales in total as a proxy for a variety of small population effects.

To date, no environmental time series and mechanism has been identified as impacting survival or fecundity of the Cook Inlet beluga population, so environmental variation is included as random variation in both the probability of birth and survival in proportion to the effect of density dependence. A new environmental time series is drawn for each run of the model and results from this analysis will indicate the response of the model to autocorrelated variation in fecundity and survival rather than a specific environmental time series. The environmental variation is in the form of a stationary, correlated, normal random deviation with mean = 0, variance = σ^2 and correlation = ρ (Morris and Doak 2002:139). When this feature is included in the model, the value of σ is set at 0.2 of the growth rate range of the density dependence, between 2% and 6%. Consequently 95% of the variation will fall within ± 0.8% to ± 2.4% per capita annual growth for density dependent ranges from 2% to 6%. However, because these are applied to survival and birth probabilities, they remain subject to the biological constraints of survival in the interval zero to one and birth rate in the interval zero to 0.333. A measure of the effect of the correlation is the distribution of runs of positive or negative variation (i.e., good conditions leading to a positive bias in growth or poor conditions leading to a negative bias in growth). The correlation is set at 0.8 which gives a median run of 2.3 years, with 90% of runs at 12 years or less and 99% of runs at 26 years or less. While this choice is arbitrary, it provides at least one change of environment during the time series of abundance estimates in most cases.

The remaining parameter in the age-structured model was the age at first birth (a_{mat}) which was set to start at 10 years. Note that because birth is a discrete event, either a female gives birth to a calf or not, the value of b determined the distribution of ages of first giving birth for the model population. For instance, if b was 0.25 then 25% of females first gave birth at age 10, 25% at age 11, etc., resulting in an age at first birth distributed from ages 10 to 13. By the same reasoning if b was 0.14 then age at first birth was distributed from ages 10 to 16, or if b was 0.10 then age at first birth was distributed from ages 10 to 19.

To set up the initial age structure and a nearly uniform prior for the population abundance in 1994 (N_{1994}), N_{1994} was drawn from a uniform distribution ranging from 450 to 950 belugas (U[450,950]), a constant harvest level $H_{79\text{-}93}$ was chosen from U[10,30] and when environmental variation is included, an environmental time series is drawn. The population starts in 1979 (N_{1979}) and is projected forward to 1994 to set up the age structure. The required value for N_{1979} is found using a bisection method between the extremes of 450 and 2950 which covers the 95% confidence interval around the abundance estimate in 1979 of 1,300 belugas if it is given the same CV as the 1994 abundance estimate. The bisection is conducted by choosing extreme values for the 1979 abundance with the low end of the abundance, $NL_{1979} = 400$ and the high end of the abundance, $NH_{1979} = 3000$ and a trial value, NT_{1979}, half way between 400 and 3,000. A stable age distribution was set up using s_{NT1979} (the survival rate calculated based on NT_{1979}) and setting ϕ to 1.00. Age and sex classes were filled as a multinomial distribution of NT_{1979} by density at age for each sex. The population was then projected from 1979 to 1994. The population size in 1994, NT_{1994}, is then compared to N_{1994} to determine if the value for NT_{1979} is a useable starting point. If NT_{1994}, is within $N_{1994} \pm 25$ it was considered sufficiently close and the simulation was continued from that point—the stochastic nature of the model prevented an exact match. If $NT_{1994} > N_{1994} + 25$, then the value of NL_{1979} is set to $NT_{1994} - 50$, if $NT_{1994} <$

N_{1994} – 25, then the value of NH_{1979} is set to NT_{1994} + 50, a new NT_{1979} is calculated and the process is repeated until a useable value of NT_{1994} is found or 12 trial values of NT_{1979} are discarded and a new value for N_{1994} is drawn. Projecting the model through 15 years prior to 1994 allowed the juvenile ages to be filled with values derived from the population model and the adult segment to be subject to the pre-1994 harvest level while maintaining nearly uniform and independent prior distributions for N_{1994} and ϕ_0. Each population was then projected from 1994 to 2007 and likelihood was calculated as:

$$L_j = \prod_{t=1994}^{2007} T\left(\frac{N_{t,j} - \overline{N}_t}{\overline{N}_t CV(\overline{N}_t)}, DF = 10 \right) \quad , \qquad (7a)$$

where,

L_j is the relative likelihood of the *jth* population projection;

$T(X, DF = 10)$ is the density of Student's-t distribution at *X* with 10 degrees of freedom;

$N_{t,j}$ is the population size of the *jth* projection in year *t*; and

$\overline{N}_t, CV(\overline{N}_t)$ are the estimated abundance (point estimate) and associated coefficient of variation in year *t*.

In the case where small or gray animals are less likely to be counted either by observers or in the video analysis,

$$L_j = \prod_{t=1994}^{2007} T\left(\frac{S_{t,j} - \overline{N}_t}{\overline{N}_t CV(\overline{N}_t)}, DF = 10 \right) \quad , \qquad (7b)$$

where,

$S_{t,j} = \sum_{all\ ages} w_a \left(f_{a,t,j} + m_{a,t,j} \right)$ is the observed size of the population excluding the missed animals at time *t*;

$w_a = \begin{cases} a/10 & \text{for } a = 0 \text{ to } 9, \\ 1 & \text{for } a \geq 10. \end{cases}$ are weights such that animals in older age classes are more likely to be seen and counted, with all adults seen.

The Student's-t distribution was chosen for the likelihood model as the best fit compared to the gamma distribution, log-normal distribution and normal distribution to bootstrap results from annual abundance estimates for this population (R. Hobbs, NMFS, NMML unpublished data). Projections to 2007 with likelihoods less than 10^{-10} × the maximum possible likelihood (i.e., the likelihood if the model N_t was equal to the abundance point estimate in all years) were discarded as having no contribution to the posterior distribution. A Sampling-Importance-Resampling (SIR) algorithm was followed (Rubin 1988) in which the acceptable parameter sets were weighted by their relative likelihoods from projections to 2007, and a resample drawn with replacement to give a posterior distribution of outcomes. Projections to 2307 (300 years into the future) for this posterior parameter set were done to estimate the probability of decline and extinction during that period. Model comparisons between the various models were done using the Bayes factor (Kass and Raftery 1994, Wade 2002), calculated as twice the natural logarithm of the ratio of the average likelihoods of the two resamples:

$$BayesFactor(x,y) = 2\ln\left[\frac{\sum_{j=1}^{SIRtot} L_{j,x} / SIRtot}{\sum_{j=1}^{SIRtot} L_{j,y} / SIRtot}\right], \qquad (8)$$

where,

BayesFactor(x,y) is the Bayes factor comparing model *x* and model *y*;

ln[] is the natural logarithm of the value in [];

$L_{j,x}$ and $L_{j,y}$ are the likelihoods of the *jth* projection of model *x* and model *y*, respectively; and

SIRtot is the number of projections in the SIR subsample.

Where the Bayes factor had absolute value greater than 2 the model with the higher average likelihood was considered to be the more likely of the two, otherwise the models were of equivalent likelihood.

All models were compared to the Baseline model (U[0.94, 1.06]). The probability of the Healthy Population model (U[1.02, 1.06]) was compared using the Bayes factor. The three options for modifying the Baseline model, the constant mortality effect (*C*), the unusual mortality event (P_{Me}) and the Allee effect (*A*) were each considered. The time series of abundance and harvest data covered a sufficient range of population sizes (270-660 belugas) to compare between the Baseline and the Healthy Population models but not among the remaining options. Six models with the modifiers for survival and fecundity were considered, three of the Baseline with the *C* parameter at 1, 2 or 5 mortalities per year, one of the Baseline with the Allee parameter at 0.50, one of the Baseline with an unusual mortality event (P_{Me} = 0.05), and one of the Baseline with the *C* parameter at 1 and an unusual mortality event (P_{Me} = 0.05). Two additional models were included to test the sensitivity of the parameters: a Baseline and Healthy Population model that included an unusual mortality event and a *C* of five mortalities. Ten additional cases were included as variations of the model itself which are provided as tests of the underlying assumptions of the model: The Baseline model and the Baseline with the *C* parameter at 1 and an unusual mortality event (P_{Me} = 0.05) with missed small and gray animals, missed small and gray animals but with the fraction missed declining to zero by 2004, environmental variation, and with survival of immature animals set to 95% of the adult survival, and a final set that compared the Baseline model to the Healthy Population model with only the years 1999-2007 included in the likelihood.

For each model, 100,000 trials were projected to 2007 and the likelihood was calculated. Each population projection was fully defined by the 11 parameters: s_0, s_K, b_0, b_K,(or $s_0 s_e$, $s_K s_e$, $b_0 b_e$, $b_K b_e$), N_{1989}, H_{89-93}, *C*, M_e, P_{Me}, *A*, and a_{mat}, though, the stochastic nature of the projection meant two projections with identical parameters would have different outcomes. A sample of 10,000 of these trials, weighted by the likelihoods, was drawn with replacement for the SIR algorithm resample for further analysis. For all populations the population size in 1994, 2007, 2107, 2207 and 2307 was retained and for declining populations the year that the population dropped below 200, 100, and 10 animals was retained. A population with 1 or 0 individuals or only one sex was considered extinct.

5.1.2. PVA Results

The 20 models allowed a range of possible behaviors for the theoretical populations as they became small while behaving similarly within the range of actual abundance estimates (278-653). Examples of the deterministic annual growth multipliers associated with each model are given in Figure 5.1.2-1. For these examples, Equation 4 was solved iteratively for ϕ using values for s and b calculated at population sizes varying from 1 to 500 (Equation 6). In all of the solid line examples, density-dependent survival parameters were chosen so that at a population size of 350, the annual growth multiplier was 1.01. These "tuned" survival parameters were then used throughout the range. For the dashed line example the survival parameters were tuned such that ϕ = 1.00 at a population size of 350. The strong density-dependence example set $\phi_0 - \phi_K = 0.06$ while the weak density-dependence example set $\phi_0 - \phi_K = 0.02$, all other examples used strong density-dependence. Where growth increased as population declined crossing the value 1.00 (the dashed example), a stable equilibrium point was formed and, without stochastic variation, the population settled at this size (350 belugas). Where growth decreased as population declined crossing the value 1.00, an unstable equilibrium point resulted forming a population size threshold below which, without stochastic variation, the population continued to decline, and above which, without stochastic variation, the population increased. Consequently, if stochastic variation in the form of demographic stochasticity or unusual mortality events pushed the population below the population size threshold, the population would likely continue to decline to extinction. Note that in these examples, the annual growth multiplier fell below 1.00 for the Allee effect at a population size of about 15 belugas. For varying levels of C, the population size thresholds occurred around 60 belugas for 1 mortality per year, 120 for 2 mortalities, and 200 for 5 mortalities. This demonstrates the possibility of thresholds at different population sizes depending on the parameters used in each model.

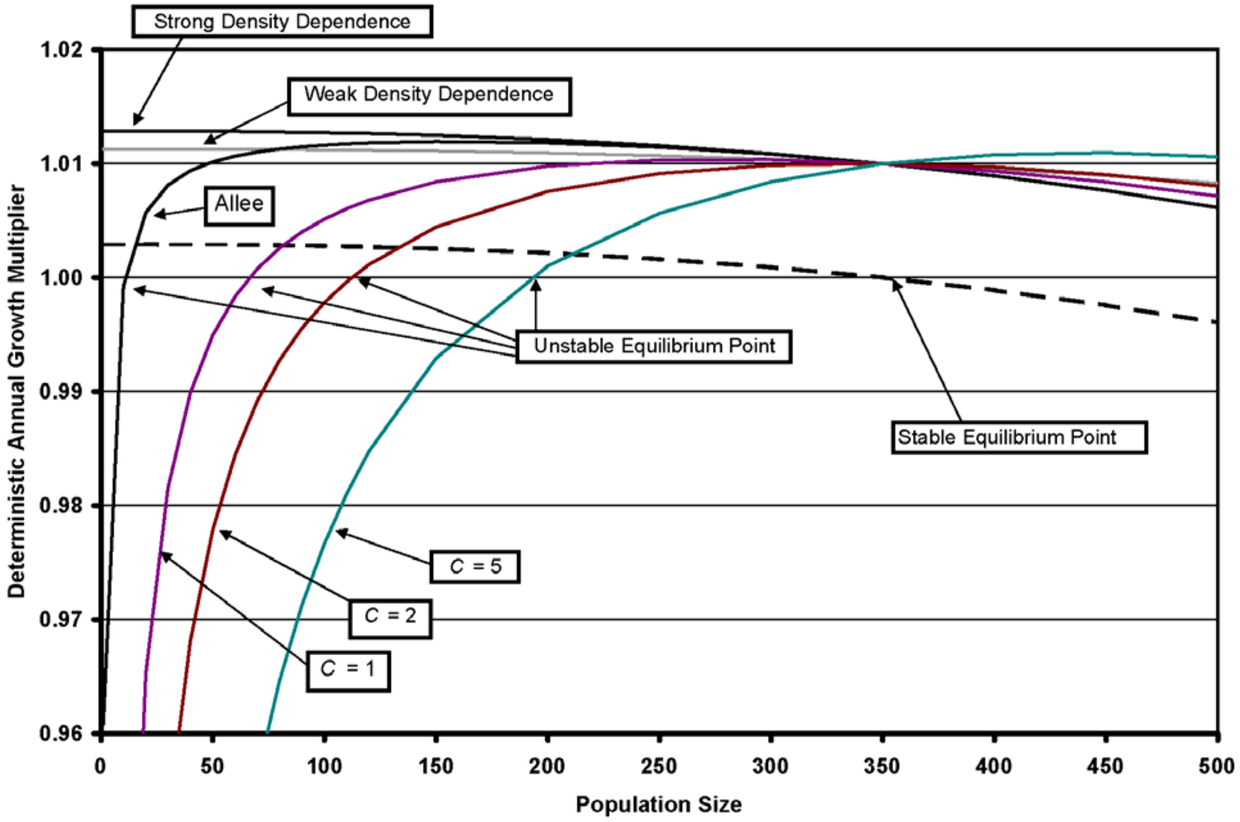


Figure 5.1.2-1. Solutions for the annual growth multiplier (ϕ) to the characteristic equation (Equation 4 in text) by population size for examples of the models. Solid line examples include density-dependent survival parameters for a population of 350 belugas chosen to set $\phi(350) = 1.01$. The dashed line example was tuned to $\phi(350) = 1.00$. Strong density-dependence was set at $\phi_0 - \phi_K = 0.06$; weak density-dependence at $\phi_0 - \phi_K = 0.02$; all other examples used strong density-dependence. *C* is the annual constant mortality effect parameter.

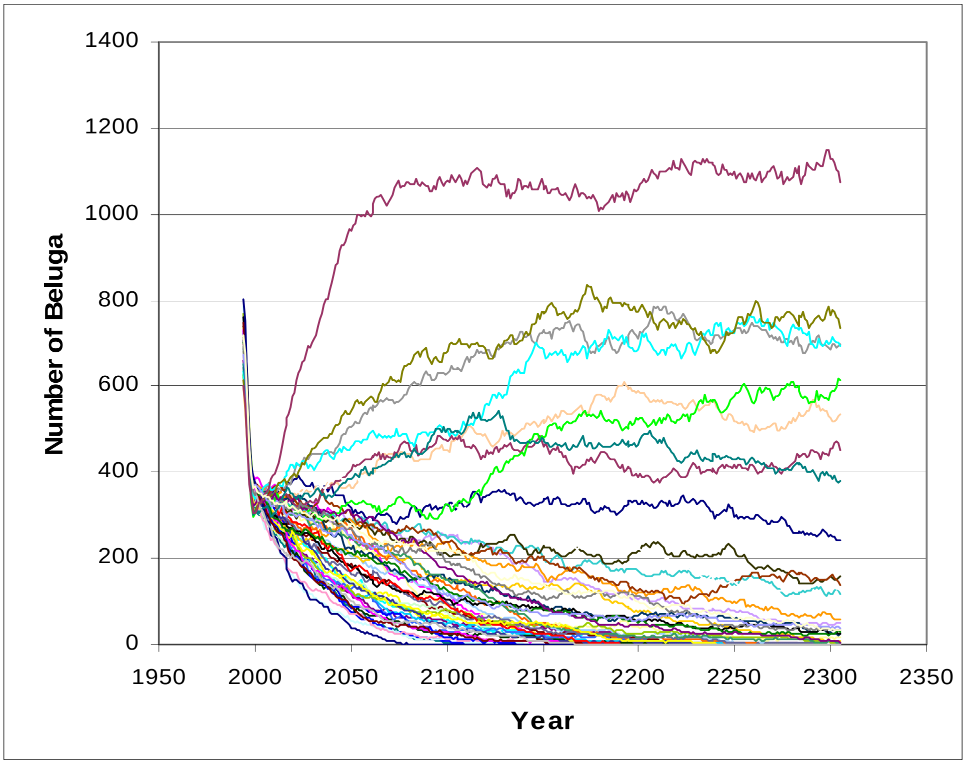
These growth multipliers and resulting behavior of the populations are reflected in the abundance time series for these example populations (Fig. 5.1.2-2a-j). Note that although the projections match the abundance time series closely during the period from 1994 to 2007, after 2007 there was considerable variation in behavior. Although there was no harvest in these models after 2007, the examples with $C > 0$ could be considered examples of the effect of a constant harvest level. Considering the Baseline model (Fig. 5.1.2-2a), which allowed a growth multiplier between 0.94 and 1.06, we had three typical behaviors: a slow decline to extinction, an increase to *K*, and an approach to stable equilibria between extinction and *K*. When only a limited range of values for the growth multiplier (between 1.02 and 1.06) was considered, as in the Healthy

Population (Fig. 5.1.2-2b) only one behavior resulted, an increase until the population leveled off near *K*.

Inclusion of an effect that created a threshold, such as predation (*C*) or Allee (Fig. 5.1.2-2c-f), added an additional behavior of a rapid decline to extinction, with the steepness determined by the height of the threshold effect. This threshold was particularly pronounced when the predation parameter was two or greater (Fig. 5.1.2-2e-f) causing a visible break point below which there was little probability of avoiding extinction. Including an unusual mortality event (Fig. 5.1.2-2g-j) had the effect of raising the population size threshold because populations above but near the threshold were still at risk of falling below after an unusual mortality event, and once below the threshold the population most likely would continue to decline. Because unusual mortality events periodically reduced the population, this prevented these populations from settling near an equilibrium. Extreme values of the *C* parameter alone and mixed with unusual mortality events (Fig. 5.1.2-2f, i, j) provided for sensitivity analysis. In these examples a population size threshold occurred within the range of recent abundance estimates (278-653), and in the Healthy Population model an unusual mortality event combined with $C = 5$ resulted in a significant number of extinctions, populations that would have recovered without these effects (Fig. 5.1.2-2j).

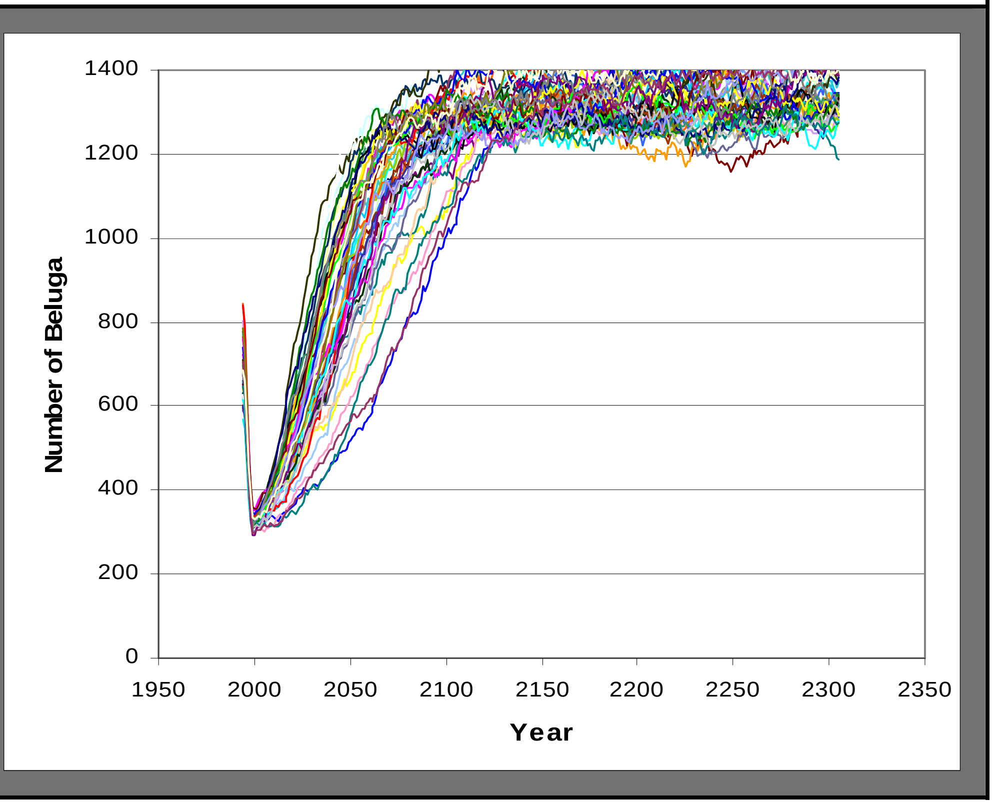
a. Baseline Model

ϕ_0 drawn from U(0.94-1.06)



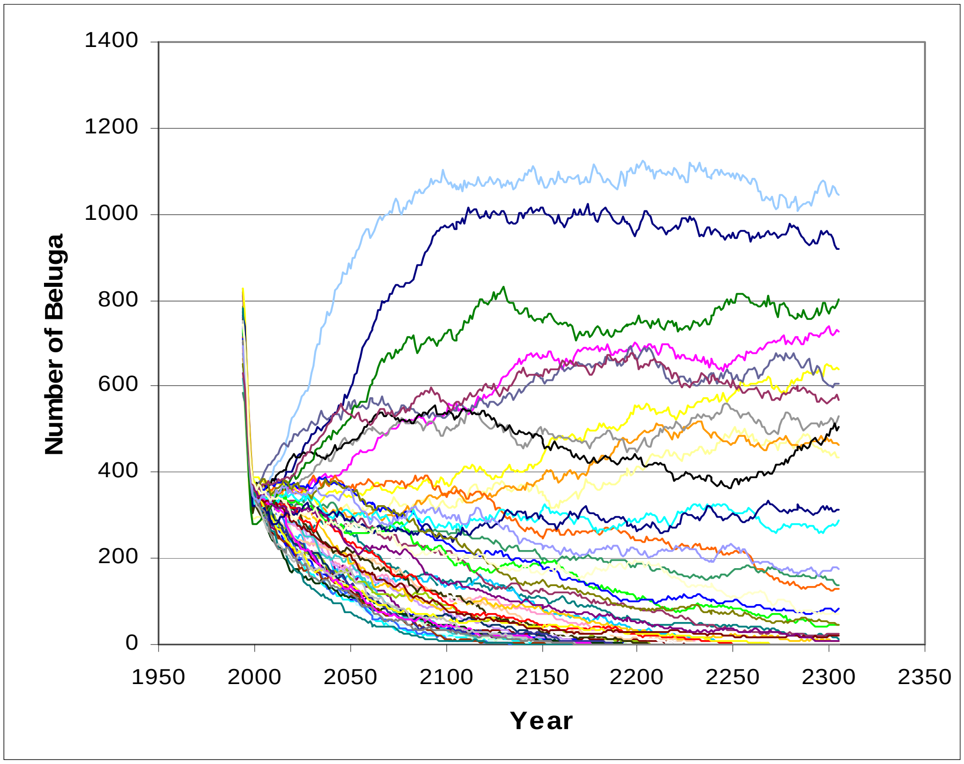
b. Healthy Population Model

ϕ_0 drawn from U(1.02-1.06)



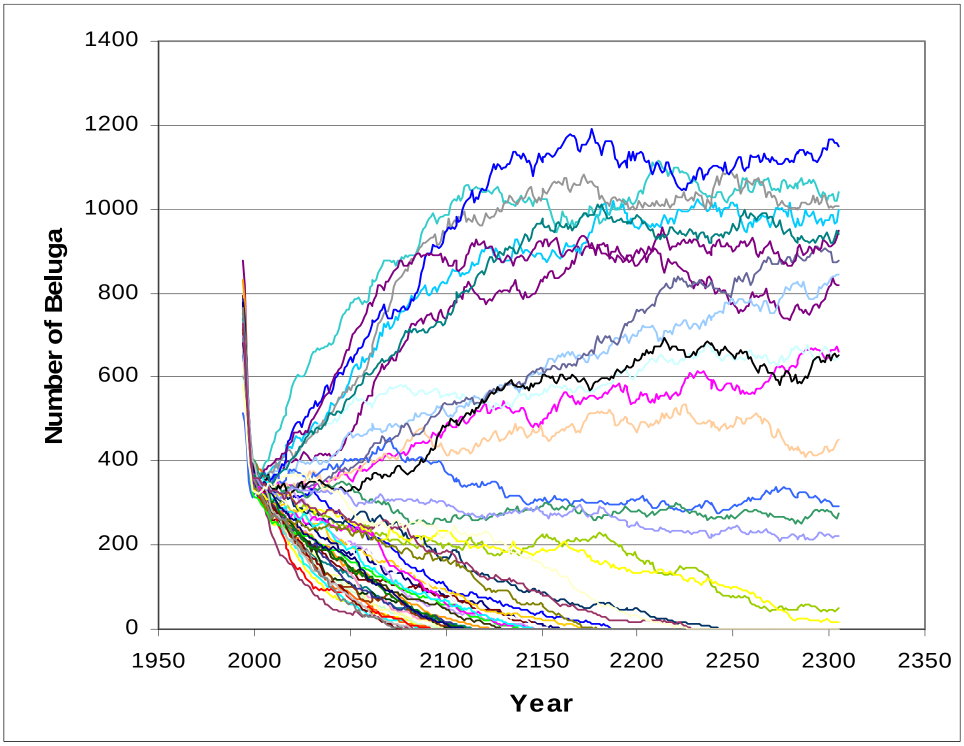
c. Baseline Model

+ Allee effect



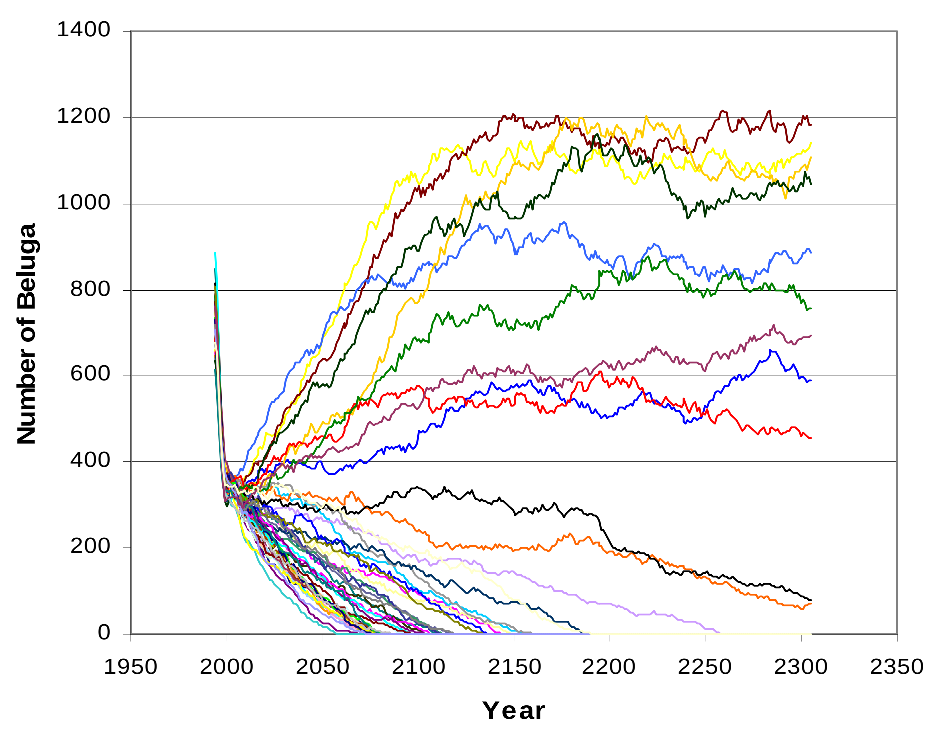
d. Baseline Model

+ *C* = 1



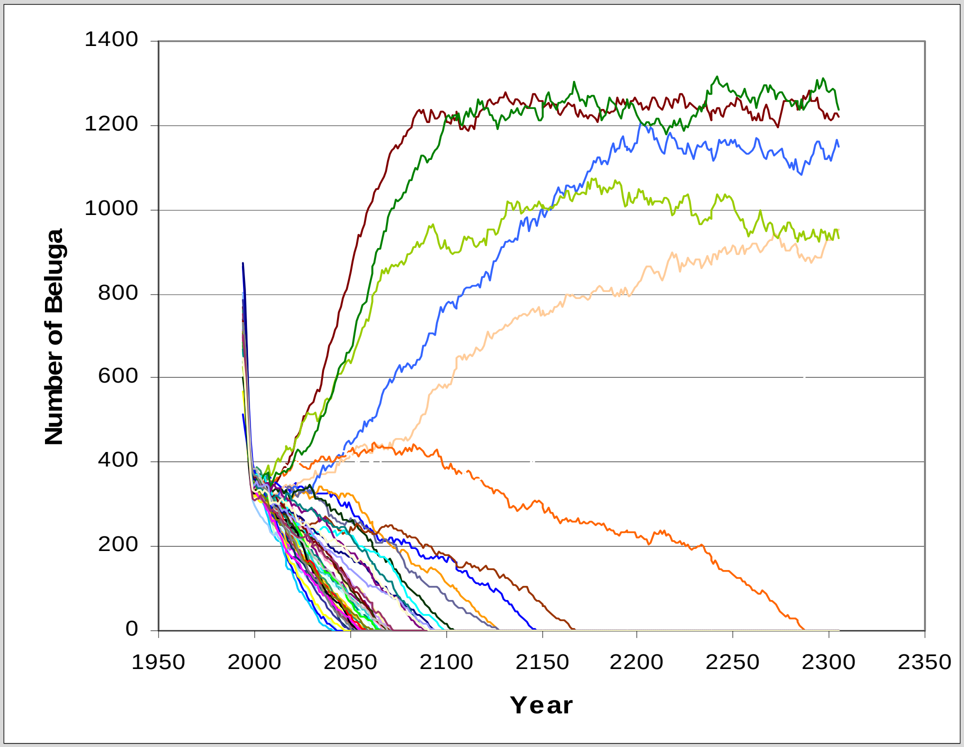
e. Baseline Model

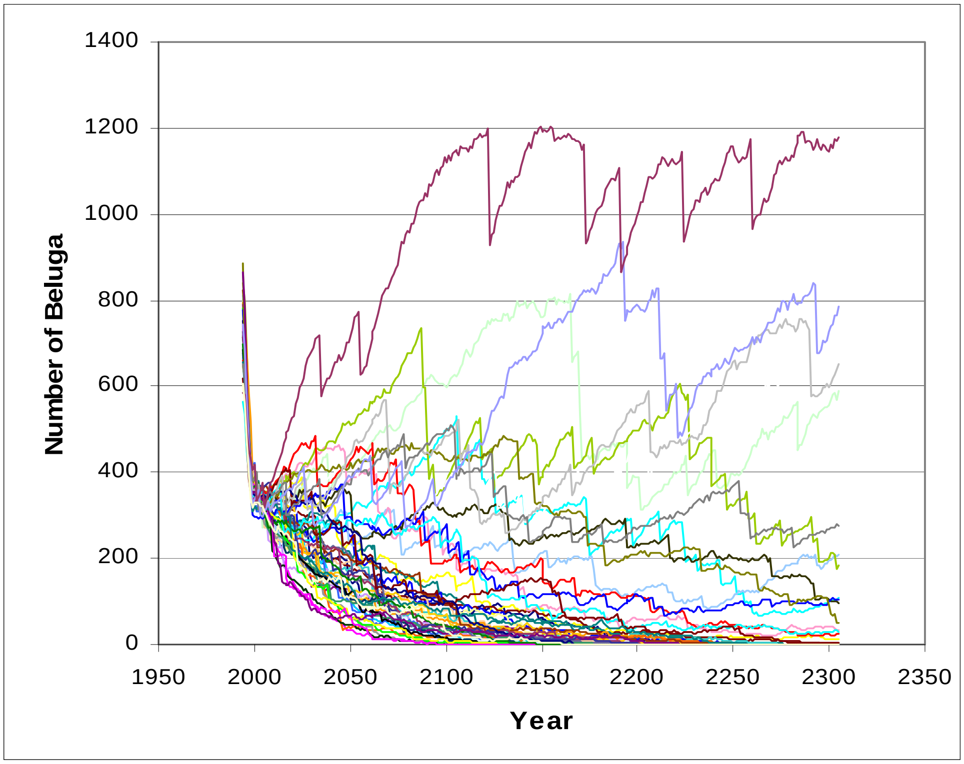
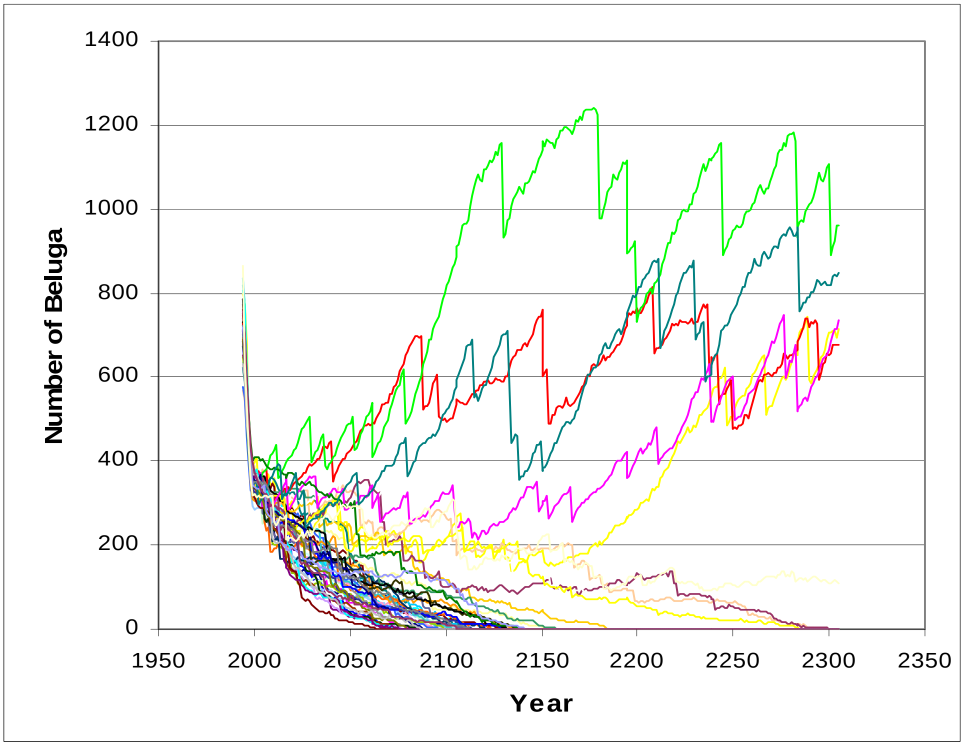
+ *C* = 2



f. Baseline Model

+ *C* = 5

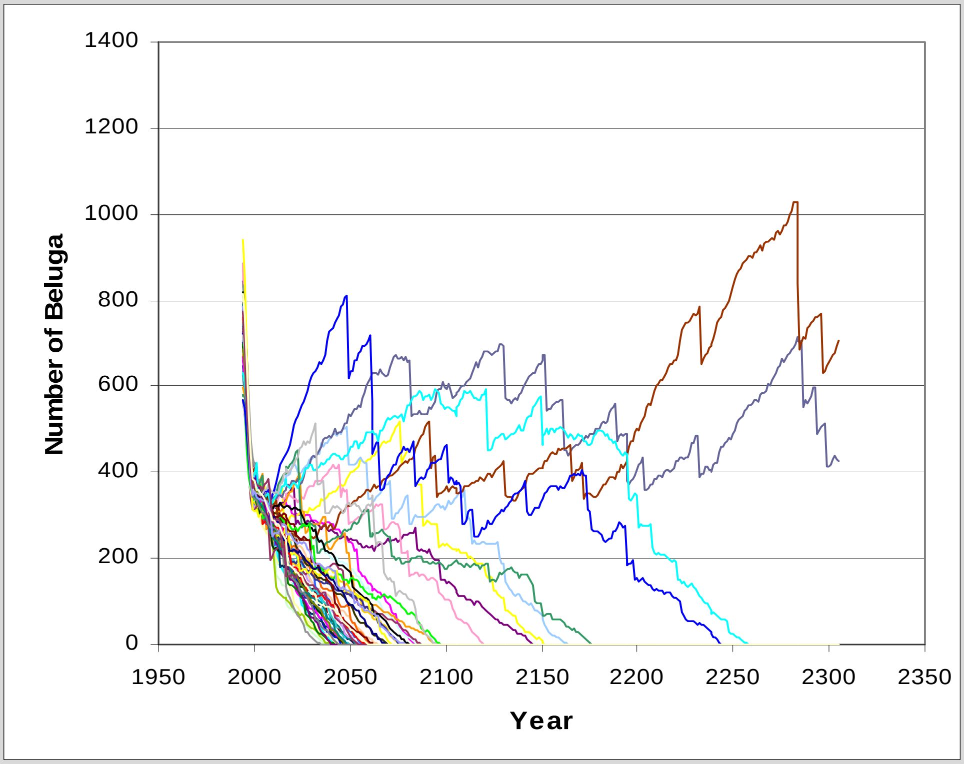


g. Baseline Model **+ P_{Me} = 0.05**	
h. Baseline Model **+ P_{Me} = 0.05** **+ C = 1**	

i. Baseline Model

+ P_{Me} = 0.05

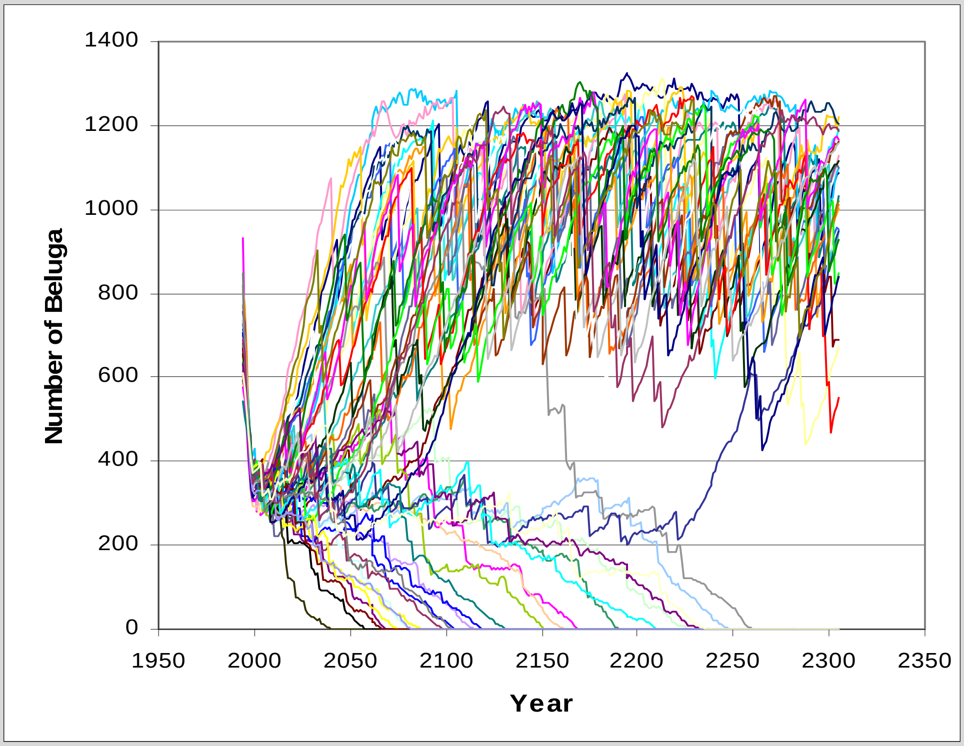
+ C = 5



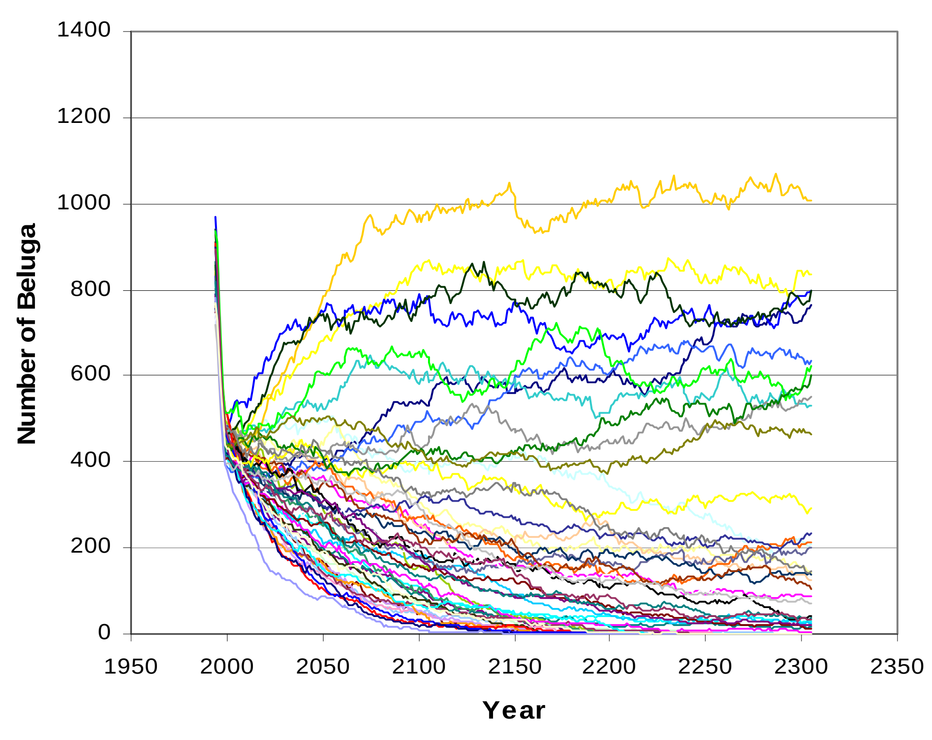
j. Healthy Population Model

+ P_{Me} = 0.05

+ C = 5



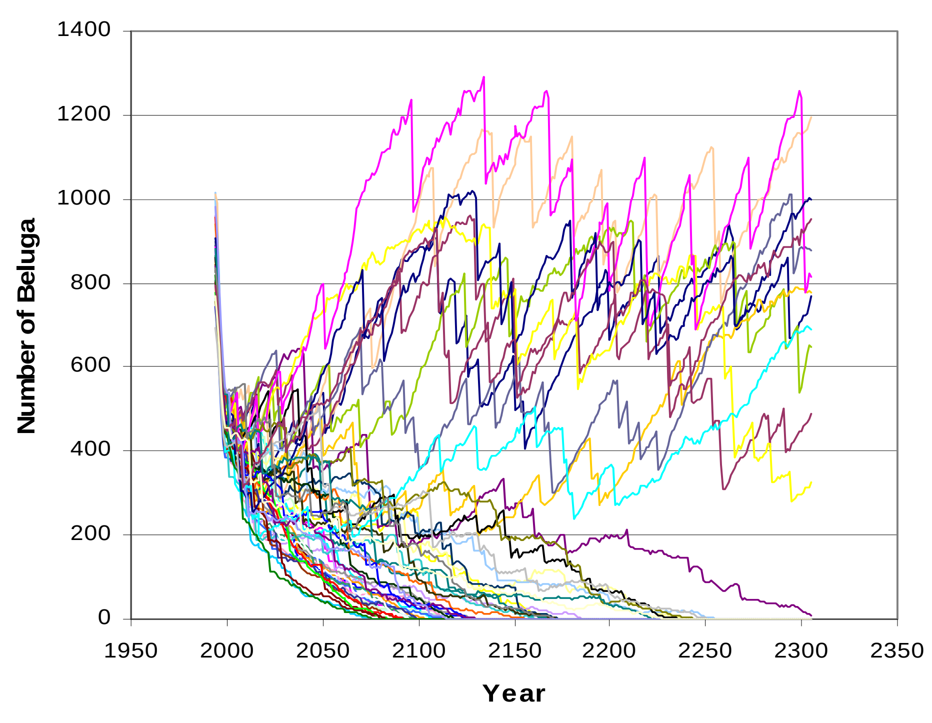
k. Missed small, gray

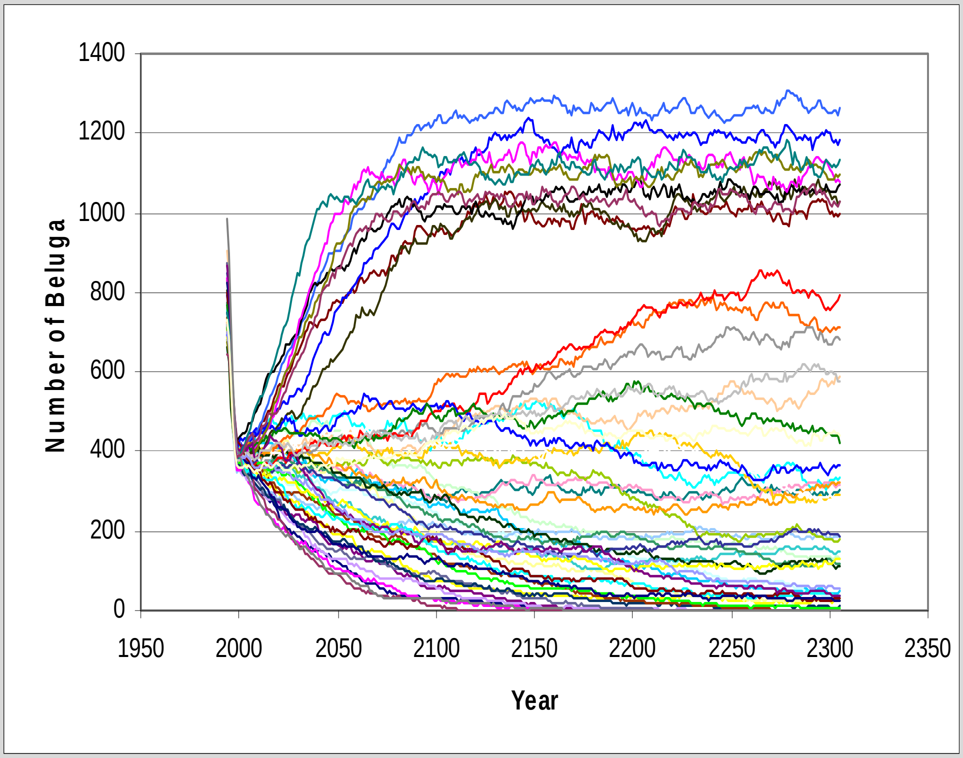
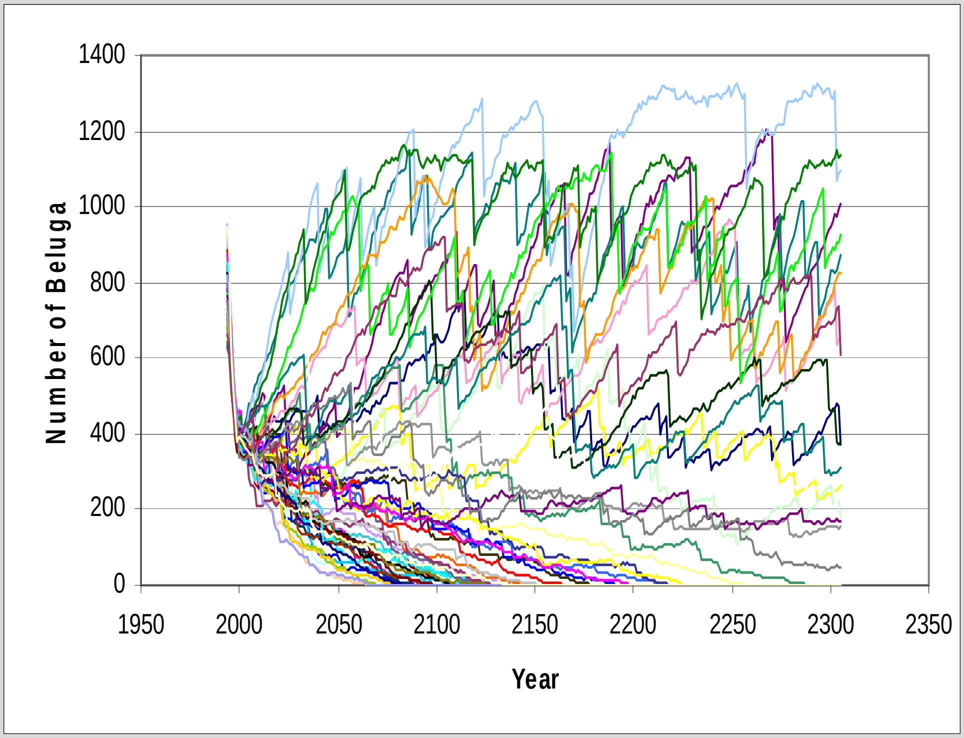


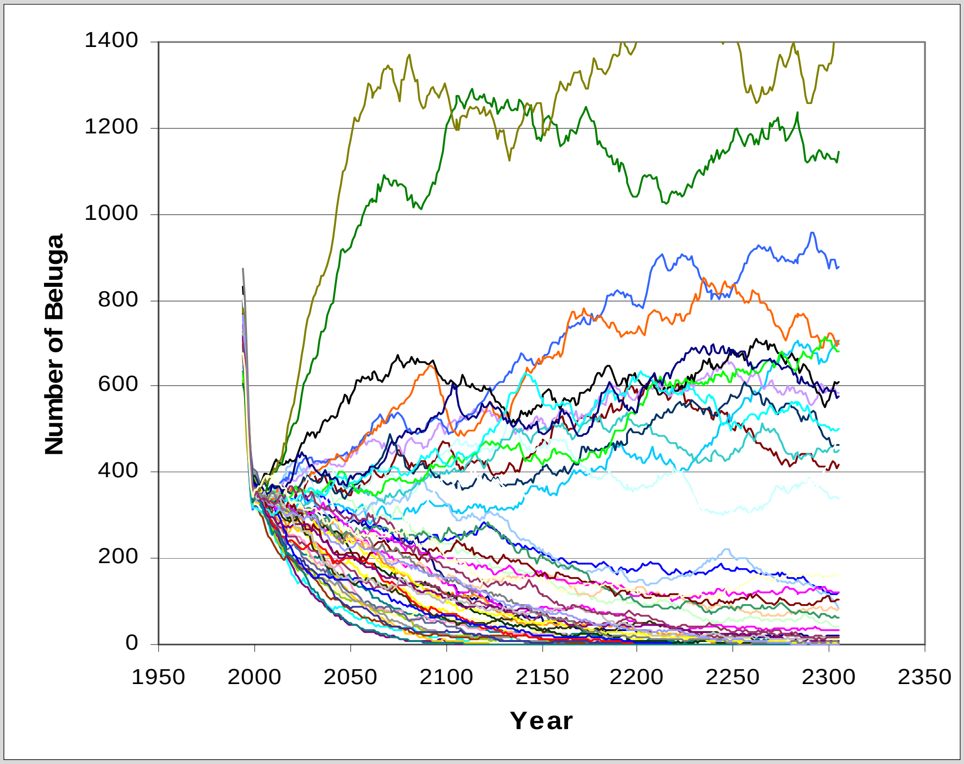
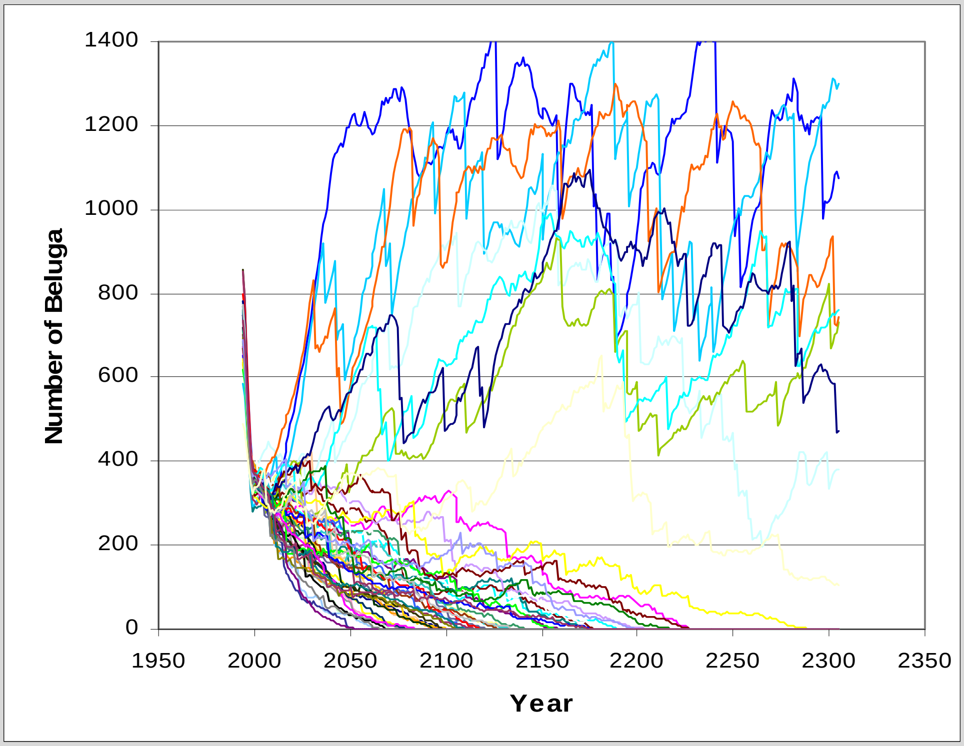
l. Missed small, gray

+ P_{Me} = 0.05

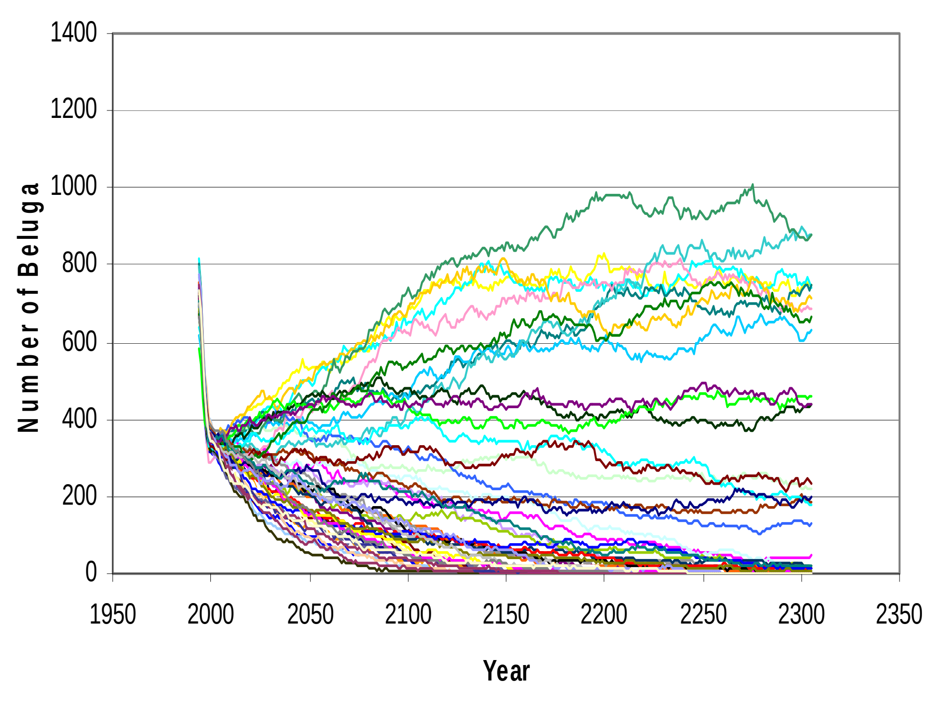
+ C = 1



m. Missed small, gray **Decreasing effect 1994-2003**	
n. Missed small, gray **Decreasing effect 1994-2003** **+ P_{Me} = 0.05** **+ C = 1**	

o. Variable Environ.	
p. Variable Environ. **+ $P_{Me} = 0.05$** **+ $C = 1$**	

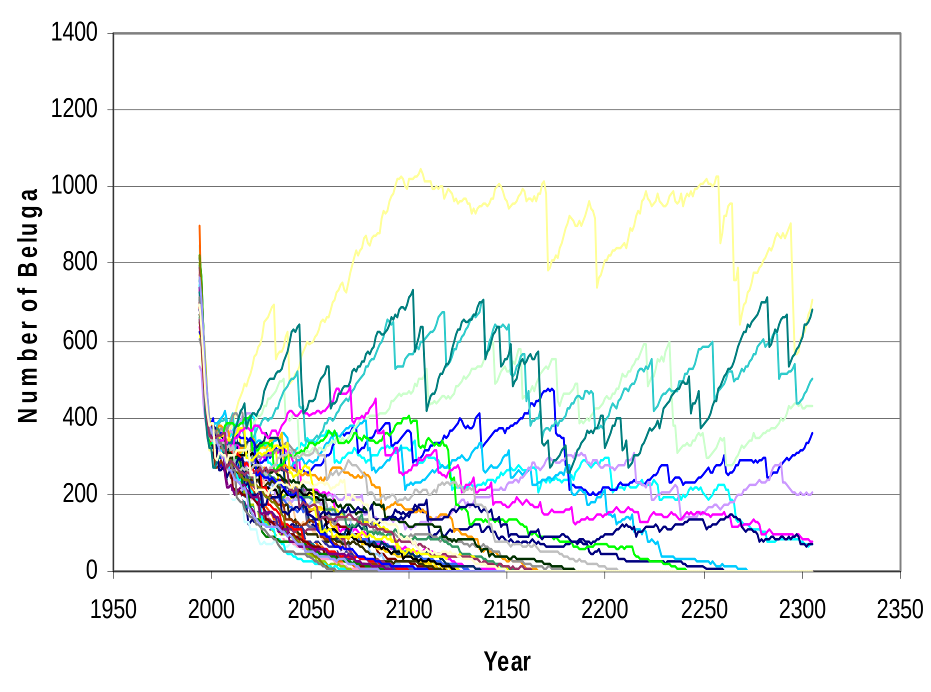
q. Immature survival 95% of adult survival



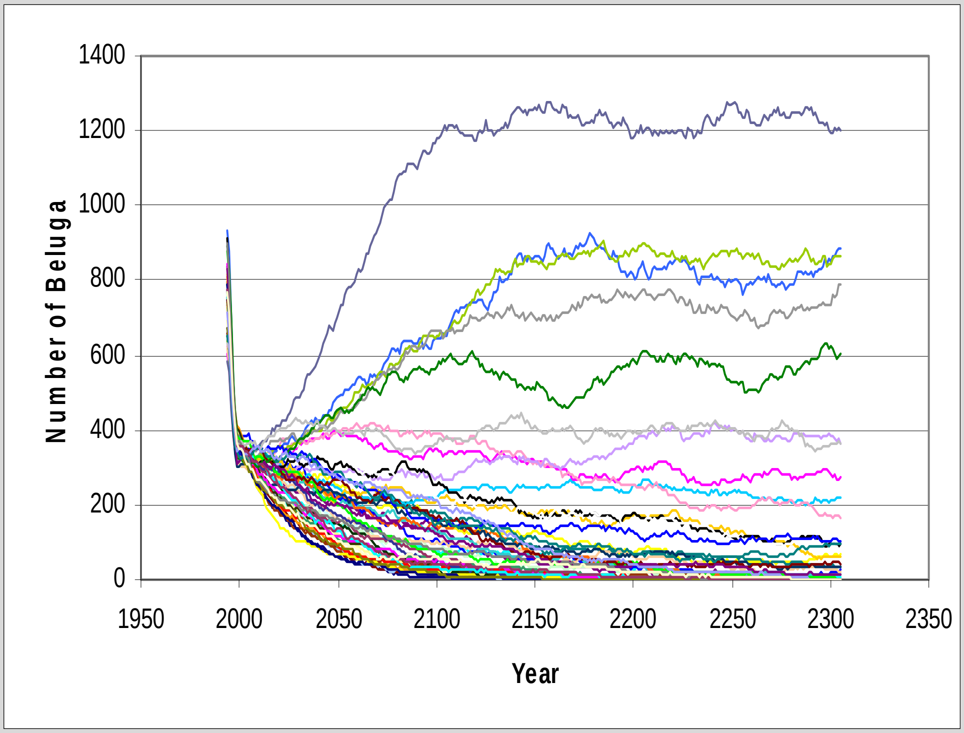
r. Immature survival 95% of adult survival

+ $P_{Me} = 0.05$

+ $C = 1$



s. Baseline w/ 1999-2007 Likelihood



t. Healthy Population w/ 1999-2007 Likelihood

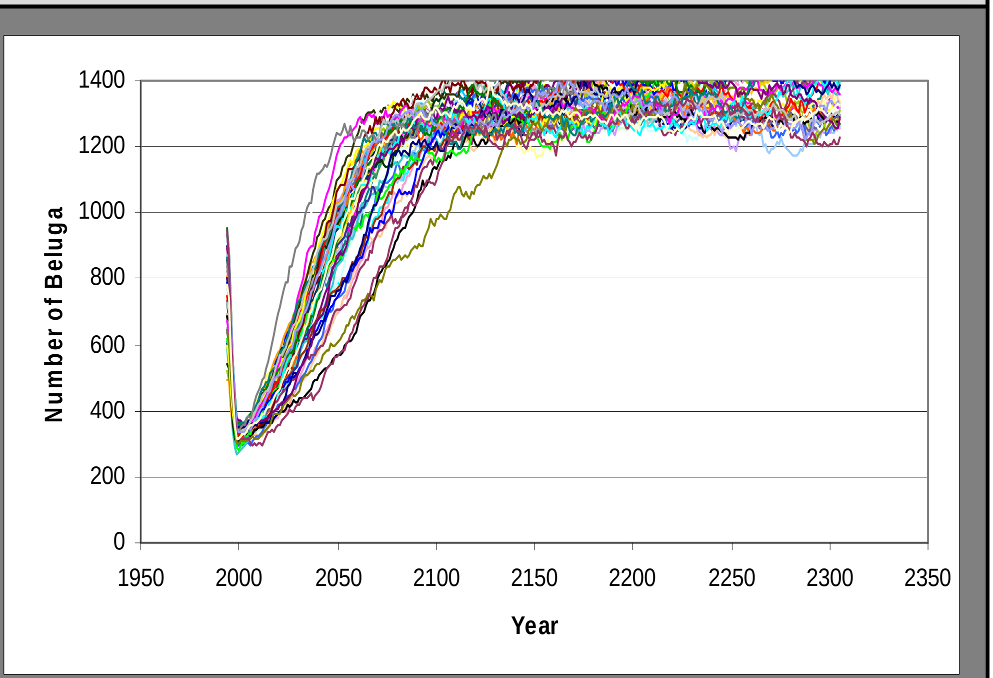


Figure 5.1.2-2. Projections of 50 example cases from the posterior sample of 10,000 trials for each of the 20 models (a-t). The dark shading (b, t) is the Healthy Population and the light shading (f, i - s) used parameters and model variations outside the range supported by the available Cook Inlet beluga data and are meant for sensitivity analysis only.

The SIR algorithm provided a posterior distribution for ϕ_0 (Fig. 5.1.2-3) shown here for the Baseline model in the cumulative (black line and left axis) and the density (bars and right axis) forms. The value of ϕ_0 is the annual growth multiplier for a small population (approaching zero) and can be interpreted as similar to R_{max}+1 in the potential biological removal (PBR) population model (Wade and Angliss 1997). However, it should be noted that unlike the PBR model these are idealized growth multipliers and the average per capita growth will be less than these values due to demographic stochasticity. The median value for ϕ_0 is 0.990 and 90% of the probability falls between 0.970 and 1.017. Also note that 96% of the probability falls below ϕ_0 = 1.02 (i.e., the minimum 2% growth that was anticipated for a healthy population).

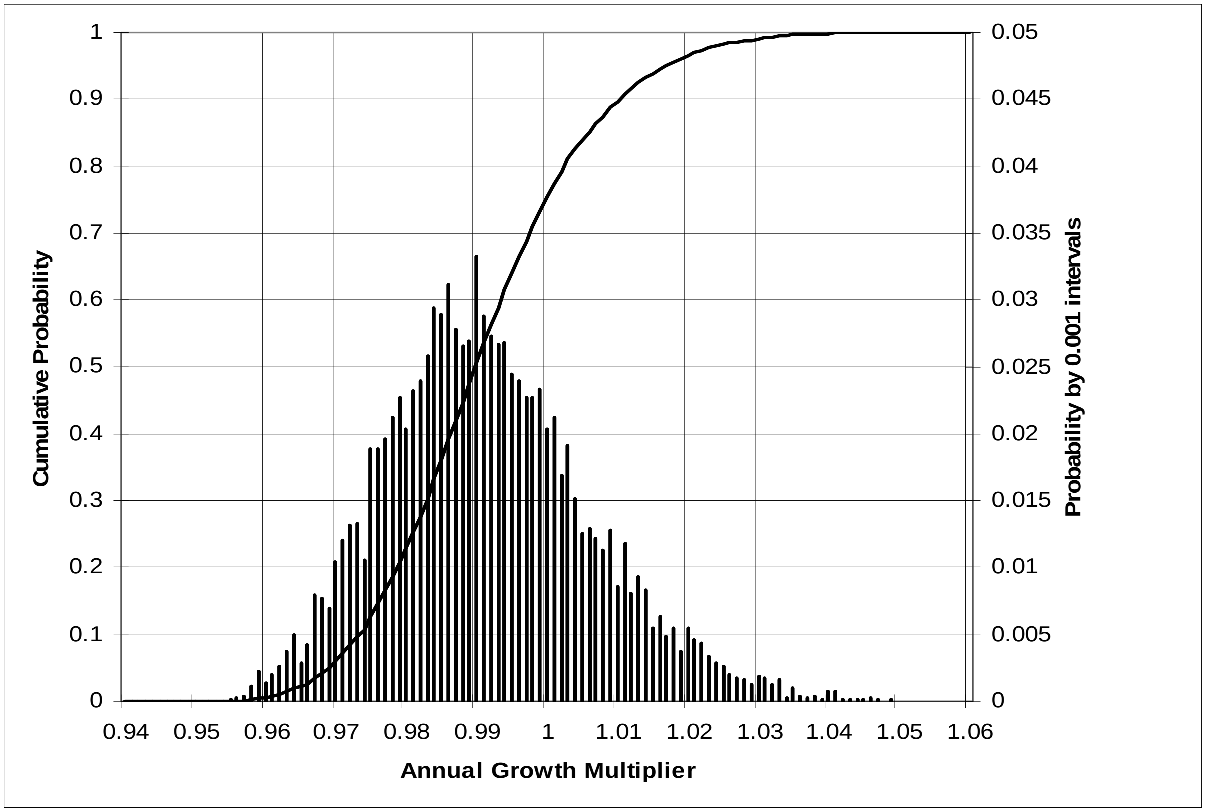


Figure 5.1.2-3. The posterior distribution of ϕ_0 from the Baseline Model which had a prior distribution for ϕ_0 of U(0.94, 1.06) and no Allee effect or constant mortality effect; the solid line is the cumulative distribution (left axis). Note that there is less than 4% probability that ϕ_0 > 1.02; the vertical bars are the probabilities of values of ϕ_0 in 0.001 increments of the distribution (right axis).

As suggested by Figure 5.1.2-2, there was little variation in the fit of the different models to the time series data (1994 to 2007). In a closer examination of the baseline model results, the abundance in each year from the SIR resample of population trajectories provide posterior distributions for the abundance in each year that account for the population dynamics as well as the annual abundance estimates from surveys. Comparison of the median values and the 2.5 percentiles and 97.5 percentiles of these posterior distributions to the annual abundance estimates from surveys, indicates that the model has a smoothing effect on the time series and gives an indication of the measurement errors that occur in each year (Fig. 5.1.2-4) (Wade 2002). The median values are an estimate of abundance in each year with the 2.5 percentiles and 97.5 percentiles forming a 95% credibility interval which is narrower than the one standard error range in most years and much narrower than the 95% confidence intervals for the individual abundance estimates from the aerial surveys (Fig. 2.3.1-1) (Punt et al. 2004, Brandon and Wade 2006).

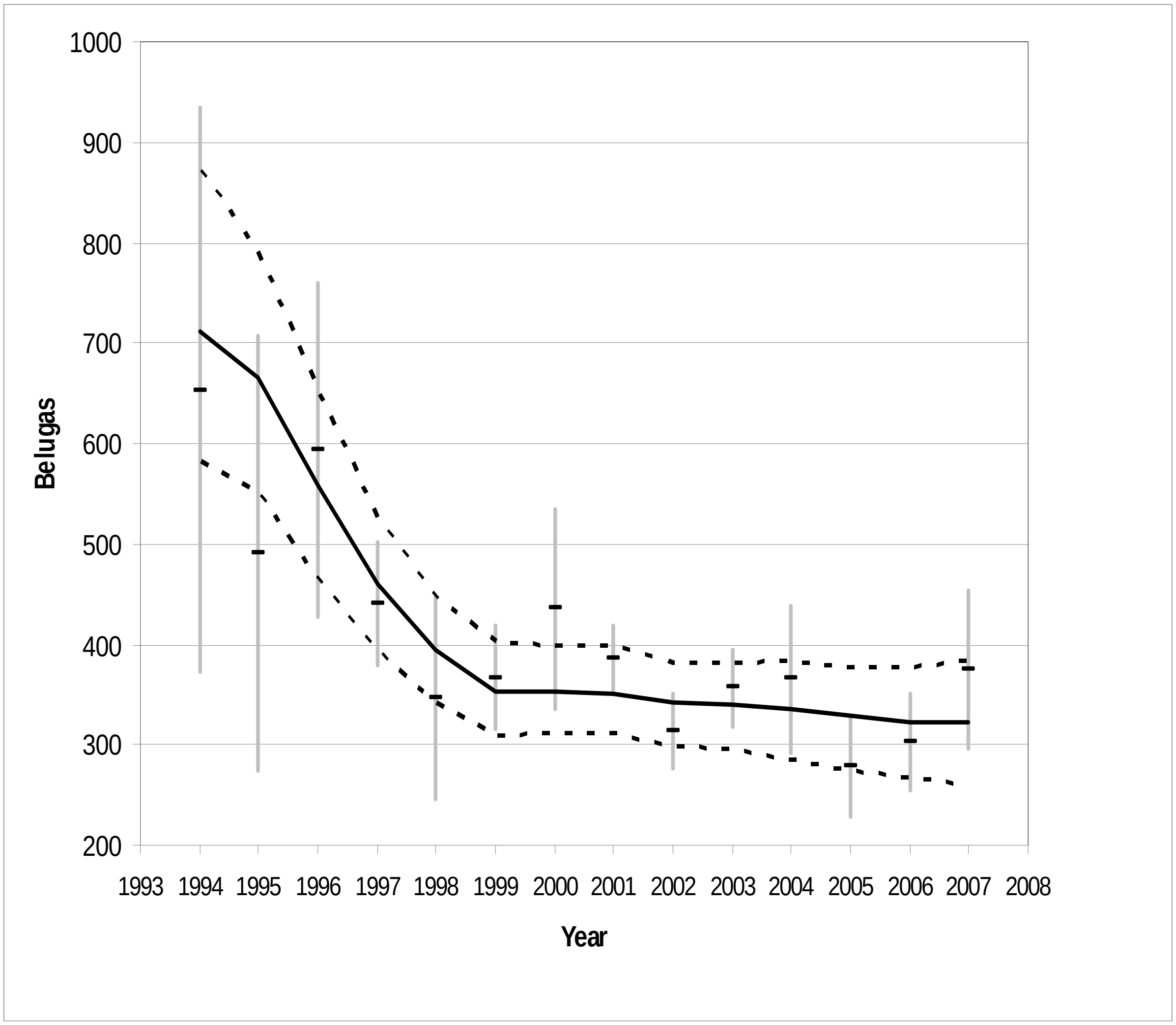


Figure 5.1.2-4. Posterior distributions of annual abundance for the years 1994-2007 from the Baseline model (a) The vertical gray bar is ± 1 standard error for each of the annual abundance estimates (black cross bar). The posterior distributions of the abundance from the population model are represented by the solid line connecting the median values and the dashed lines connecting the 2.5 percentile and 97.5 percentile values. The values between the dashed lines represent a 95% credibility interval.

Posterior distributions for abundance in 2007 from variations of the Baseline model were nearly identical with medians ranging between 322 and 328 (Table 5.1.2-1). The Healthy Population model indicated a somewhat higher median of 373 (Table 5.1.2-1, row b) but when combined with $P_{Me} = 0.05$ and $C = 5$ (Table 5.1.2-1, row j) the median value nearly fell in the range of the Baseline variations. The models with missed small, gray animals had higher medians for the 2007 population size because 15 to 20% of the populations were missed in the counts, these under estimates were compared to the abundance estimate data to test the model fit. All of the variations of the Baseline model considered had probabilities similar to the Baseline model itself with none being significantly better as indicated by the Bayes factor. The Healthy Population model fit to either the full time series or only years 1999-2007 had a much lower probability than any of the variations of the Baseline model considered and should not be considered viable models. However, the Healthy Population model with P_{Me} = 0.05 and C = 5 was significantly more likely than the Healthy Population model itself and had a probability similar to that of the variations of the Baseline model.

Table 5.1.2-1. Statistics for the posterior distributions of the population size in 2007 (N2007) and the Bayes factors for each model compared to the Baseline model. The dark shading (b, t) is the Healthy Population and the light shading (f, i - s) used parameters and model variations outside the range supported by the available Cook Inlet beluga predation mortality data and are meant for sensitivity analysis only. Note that the absolute value of the Bayes factor should be greater than 2.0 before a significant difference in probability is indicated. U = uniform distribution, C = constant mortality effect parameter, P_{Me} = unusual mortality event (with a 5% annual probability of 20% mortality).

Model ID	Variation from Baseline Model	N2007 Median	N2007 5th percentile	N2007 95th percentile	Probablility Relative to the Baseline	2 × Ln Bayes Comparison to the Baseline
a	ϕ_0 U(0.94, 1.06)	328	285	377.05	1.00	0.00
b	ϕ_0 U(1.02, 1.06)	373	329	419	0.26	-2.69
c	Allee	327	284	378	0.98	-0.03
d	$C = 1$	326	283	377	1.03	0.05
e	$C = 2$	325	281	375	0.99	-0.02
f	$C = 5$	320	276	371	1.04	0.08
g	P_{Me} = 0.05	323	270	376	1.08	0.16
h	P_{Me} = 0.05, C = 1	322	270	374	1.14	0.26
i	P_{Me} = 0.05, C = 5	316	264	367	1.12	0.23
j	Healthy Population, P_{Me} = 0.05, C = 5	340	281	392	1.16	0.30
k	Missed small, gray	414	340	505	1.22	0.39
l	Missed small, gray, P_{Me} = 0.05, C = 1	412	329	505	1.33	0.57
m	Missed small, gray, Decreasing 1994-2003	373	314	444	1.11	0.21
n	Missed small, gray, Decreasing '94-'03, P_{Me} = 0.05, C = 1	374	308	450	1.13	0.24
o	Variable environment	326	283	377	1.02	0.05
p	Var. environment, P_{Me} = 0.05, C = 1	322	269	374	1.12	0.23
q	Immature survival 95% of adult surv.	326	283	375	0.98	-0.04

Model ID	Variation from Baseline Model	N2007 Median	N2007 5^{th} percentile	N2007 95^{th} percentile	Probablility Relative to the Baseline	2 × Ln Bayes Comparison to the Baseline
r	Immature survival 95% of adult surv., P_{Me} = 0.05, C = 1	320	267	370	1.05	0.09
s	Baesline w/ 1999-2007 Likelihood	325	282	376	1.00	0.00
t	Healthy Population w/ 1999-2007 Likelihood	370	324	417	0.26	-2.70

During the projection from 2007 to 2307, considerable variation occurred within each model run and between models (Fig. 5.1.2-2; Tables 5.1.2-2 and 5.1.2-3). However, by the year 2307 in the projections, the majority of cases in each model had either gone extinct or recovered to a population size greater than 500. The Healthy Population models (b, t) were the only one which resulted in a majority of the cases recovering to a population size above 500 (Table 5.1.2-2). For the six unshaded models, the probability of extinction by 2307 was between 41% and 79% (Table 5.1.2-3). The probability of extinctions before 2057 (within 50 years) reached 1% in cases with stochastic mortality events. The probability of extinction in 100 years ranged from 1% to 42% for the unshaded models (Table 5.1.2-3).

The Allee effect had a limited impact on the probability of extinction in 300 years, increasing the probability by 1% over the Baseline. Where C was 1 or 2 animals per year (models d, e, h) there was a 20% to 39% probability of extinction in 100 years and 79% to 86% in 300 years. The effect of including $C = 1$ with $P_{Me} = 0.05$ (model h) is roughly equivalent to $C = 2$ (model e) (Table 5.1.2-3). As indicated in Figure 5.1.2-1, the $C = 1$ threshold was around 60 animals and the $C = 2$ threshold was around 120 animals. Three unusual mortality events in a short time span would nearly reduce the population by half making up the difference between the two thresholds. Increasing C to 5 mortalities per year (models f, i, j), raised the population size threshold to 200 belugas, increased the overall risk of decline and extinction in each model to the extent that a significant probability existed for extinction in 50 years (Table 5.1.2-3). Again this population size threshold was increased by including unusual mortality events (c.f. models i and j).

In general, unusual mortality events added 10% to 15% to the probabilities of extinction in 300 years in each variation of the Baseline model. In the Healthy Population model, unusual mortality events with $C = 5$ resulted in a probability of extinction in 300 years of 36% while $C = 5$ effect alone resulted in no extinctions, as the projected populations never fell below this parameter's threshold of 200 belugas. Variation in the model assumptions did not result in significant variation in the results for the similar unshaded models and, of particular note, models k and l which correspond to models a and h, but include the assumption of missed small gray animals, had similar but slightly reduced probabilities of extinction and nearly identical probabilities of decline.

Table 5.1.2-2. Outcomes of projections to year 2307 (300 years) for each of the models. The dark shading is the Healthy Population (b) and the light shading (i, j) used parameters outside the range supported by the available Cook Inlet beluga predation mortality data and are meant for sensitivity analysis only.

Model ID	Percent probability that the population will be:					Probability of extinction by 2307 (%)
	> 500	< 500 & > 350	< 350 & > 200	< 200 & > 100	< 100	
a	18	5	5	6	26	41
b	100	0	0	0	0	0
c	18	4	5	6	21	47
d	18	3	3	3	4	69
e	17	2	2	1	1	76
f	14	0	0	0	0	85
g	14	3	4	4	19	57
h	11	2	2	2	3	79
i	6	0	0	0	0	93
j	53	2	1	1	1	42
k	21	5	6	7	28	32
l	15	3	3	2	4	74
m	30	5	6	7	25	28
n	17	3	3	2	4	70
o	19	4	5	5	24	42
p	11	2	2	2	3	79
q	15	4	5	6	28	41
r	5	2	2	1	3	88
s	15	3	5	5	21	51
t	100	0	0	0	0	0

Table 5.1.2-3. Extinction risk for each of the models by 2057, 2107, 2207, and 2307. The dark shading is the Healthy Population (b) and the light shading (i, j) used parameters outside the range supported by the available Cook Inlet beluga predation mortality data and are meant for sensitivity analysis only. Probability of declining is the probability that $N_{2307} < N_{2007}$

Model ID	Percent probability of extinction by:				Probability of declining (%)
	2057 (50 years)	2107 (100 years)	2207 (200 years)	2307 (300 years)	
a	0	1	22	41	77
b	0	0	0	0	0
c	0	2	28	47	77
d	0	20	60	69	79
e	1	42	71	76	81
f	25	72	83	85	85
g	0	5	39	57	83
h	1	39	72	79	86
i	45	84	92	93	93
j	1	18	38	42	45
k	0	0	14	32	77
l	0	27	64	74	84
m	0	0	12	28	66
n	0	27	61	70	81
o	0	1	23	42	76
p	1	38	71	79	86
q	0	1	21	41	81
r	1	43	80	88	93
s	0	2	31	51	82
t	0	0	0	0	0

5.1.3. Discussion and Conclusions

Although the model structure and parameters had obvious effects on the distributions of predicted outcomes, even the best case scenario (model a) with no threshold effects resulted in population declines in 77% of the cases and extinction in 41% of the cases within 300 years. With this most optimistic scenario, with no harvest after 2005, only 18% of the cases resulted in a population above 500 animals in 2307. The distributions of possible outcomes were sensitive to a variety of poorly known small population effects; however, the data that we do have supports the choice of the Baseline (model h) as the best approximation of the current population with the estimated mortality due to killer whale predation averaging 1 per year (*C* = 1) and allowing for uncertainty with unusual mortality events occurring on average every 20 years. This model had a 39% probability of extinction in 100 years and an 86% probability of extinction in 300 years. Although there were no data to support higher predation rates or more frequent unusual mortality events, the examples given (models f, i, j) indicate the fragile nature of this population. Should the constant mortality level increase either by increased killer whale predation or other means, or if this mortality level has been underestimated, the population would have a very high probability of decline and a significant probability of extinction in 50 years (e.g., Table 5.1.2-3, row f, i).

A synergistic effect occurred when the Allee effect or constant mortality effect acted as traps for populations hit by a series of unusual mortality events, which hastened the extinction of declining populations and placed even populations with an otherwise healthy annual increase at risk. Several of the assumptions of this analysis have been questioned during reviews. To test these assumptions, we used models k-t, which indicate that the results are robust to relaxation of the assumptions. Of particular interest are the results for models k and l where over half of the small gray animals under 10 years of age are assumed to be missed during the aerial surveys. Models k and l were designed to test the results presented in models a and h, respectively, and ended up having nearly identical results to these models. In all but models o and p, the environment is assumed to be constant. Models o and p showed results similar to the constant environment models, however, without an environmental time series and mechanism forcing the population it was unlikely that environmental variability alone would do more than add to the existing variability. Taken as a whole, these modeling results indicate clearly that it is likely that the Cook Inlet beluga population will continue to decline or go extinct over the next 300 years unless factors determining its growth and survival are altered in its favor.

5.2. Application of IUCN Criteria

Information on application of IUCN criteria is provided in Hobbs et al. (2006).

6. CONCLUSIONS OF THE STATUS REVIEW

The small, isolated population of belugas in Cook Inlet has not shown appreciable signs of recovery since 1999 when hunting restrictions began. Prior to this, a significant decline in abundance was documented from 1994 to 1998, but there are little empirical data for the period between 1979 and 1994 to identify a mechanism for the apparent decline of this population from 1,300 to 650. Anecdotal reports suggest a Native subsistence hunt (enumerated through hunter interviews) was significant during the 1970s and 1980s and may have been at levels similar to the hunts reported in the mid-1990s. Also, commercial and sport hunts occurred during the 1960s and 1970s, so the highest available abundance estimate of 1,300, based on the 1979 ADF&G survey, may already represent a partially depleted population. With the very limited hunt between 1999 and 2007, NMFS anticipated that the population would begin to recover at a rate of 2% to 6% per year. However, a Bayesian analysis including the 2006 and 2007 estimates of abundance indicates that there is a probability of less than 4% that the annual increases of 2% or greater will occur and a probability of 77% or more that the population will decline further.

A population viability analysis was conducted to assess the extinction risks faced by this small population under a range of scenarios that considered density dependence, constant mortality, Allee effects, and catastrophes. The best case scenario, with no threshold effects, resulted in population declines in 77% of the cases and extinction within 300 years in 41%. Even with this most optimistic scenario, and with no harvest after 2007, only 18% of the cases resulted in a population above 500 animals in 2307. There is a significant probability that the Cook Inlet beluga population will continue to decline or go extinct over the next 300 years unless factors determining its growth and survival are altered in its favor. The contraction of the range of this population northward into the upper Inlet makes it far more vulnerable to catastrophic events with the potential to kill a significant fraction of the population. The probability of potential catastrophic events -- such as oil or toxic substance spills, failure of key fish runs, ice entrapments, or disease or parasitic introductions -- added about 30% to the probabilities of extinction in 300 years in the models. As the models demonstrate, killer whale predation which is documented on a near annual basis, could also significantly impact recovery. Since belugas spend much of their time in shallow waters, stranding is a constant risk. Prolonged stranding events more than a few hours, although not common, may under unusual circumstances such as unusual tidal cycles, storm surge, flooding, tsunami or earthquake uplift result in significant mortalities.

Belugas in Cook Inlet make up a small, genetically distinct population that appears to have strong site fidelity to the Inlet year-round. Should this population go extinct, it is highly unlikely that Cook Inlet would be repopulated with belugas in the foreseeable future. The closest large population is in Bristol Bay, 1,500 km away by sea and separated by the Alaska Peninsula that extends 3 degrees of latitude south of the southern limit of the Bristol Bay beluga population. It is highly probable that the loss of the Cook Inlet beluga population would result in a permanent loss of range for the beluga species.

7. CITATIONS

Abookire, A. A., and J. F. Piatt. 2005. Oceanographic conditions structure forage fishes into lipid-rich and lipid-poor communities in lower Cook Inlet, Alaska, USA. Mar. Ecol. Prog. Ser. 287:229-240.

ADF&G (Alaska Department of Fish and Game). 2004. Fish Distribution Database – Interactive Mapping. <www.sf.adfg.state.ak.us/sarr/FishDistrib/FDD_ims.cfm>. Accessed October 2006.

ADF&G (Alaska Department of Fish and Game). 2005. Voles. State of Alaska. Available at http://www.adfg.state.ak.us/pubs/notebook/smgame/voles.php. Accessed October 2007.

Allee, W. C., O. Park, A. E. Emerson, T. Park, and K. P. Schmidt. 1949. Principles of animal ecology. W. B. Saunders Co., Philadelphia, PA.

Allen, K., and T. Smith. 1978. A note on the relation between pregnancy rate, age at maturity and adult and juvenile mortality rates. Rep. Int. Whal. Comm. 28:477-478.

Anderson, K .L., J. E. Whitlock, and V. J. Harwood. 2006. Persistence and differential survival of fecal indicator bacteria in subtropical waters and sediments. Appl. Environ. Microbiol. 71(6):3041-3048.

Anderson, R. M., and R. M. May. 1992. Infectious diseases of humans. Dynamics and control. Oxford Univ. Press, New York, NY.

Angliss, R. P., and K. L. Lodge. 2004. Alaska marine mammal stock assessments, 2003. U.S. Dep. Commer., NOAA Tech. Memo. NMFS-AFSC-144, 230 p.

Armstrong, T. 1985. White whales trapped in sea ice. Polar Rec. 22:552.

Barr, B., J. L. Dunn, M. D. Daniel, and A. Banford. 1989. Herpes-like viral dermatitis in a beluga whale. J. Wildl. Dis. 25(4):609-611.

Becker, P. R., M. M. Krahn, E. A. Mackey, R. Demiralp, M. M. Schantz, M. S. Epstein, M. K. Donais, B. J. Porter, D. C. G. Muir, and S. A. Wise. 2000. Concentrations of polychlorinated biphenyls (PCB’s), chlorinated pesticides, and heavy metals and other elements in tissues of beluga whale, *Delphinapterus leucas*, from Cook Inlet, Alaska. Mar. Fish. Rev. 62 (3):81-98.

Becker, P. R., R. S. Pugh, M. M. Schantz, E. A. Mackey, R. Demiralp, M. S. Epstein, M. K. Donais, B. J. Porter, S. A. Wise, and B. A. Mahoney. 2001. Persistent chlorinated compounds and elements in tissues of Cook Inlet beluga whales, *Delphinapterus leucas*, banked by the Alaska marine mammal tissue archival project. U.S. Dep. Commer. and National Institute of Standards and Technology, NISTIR 6702, MMS 2000-067. 54 p.

Béland, P., S. De Guise, and R. Plante. 1992. Toxicologie et pathologie des mammiferes marins du Saint-Laurent. INELS, Montreal, QC for the Fond Mondial pour la Nature (Canada), Toronto.

Béland, P., S. De Guise, C. Girard, A. Lagacé, D. Martineau, R. Michaud, D. C. G. Muir, R. J. Norstrom, E. Pelletier, S. Ray, and L. R. Shugart. 1993. Toxic compounds and health and reproductive effects in St. Lawrence beluga whales. J. Great Lakes Res. 19(4):766-775.

Begon, M., J. L. Harper, and C. R. Townsend. 1996. Ecology: individuals, populations and communities. 3rd edition. Blackwell Science, Oxford, United Kingdom.

Blackburn, J. K. 2003. Characterizing spatially explicit patterns of antibiotic resistance in the marine environment using top-level marine predators. Master’s thesis, Louisiana State Univ., Baton Rouge, Louisiana. 120 p.

Blackwell, S. B., and C. R. Greene, Jr. 2002. Acoustic measurements in Cook Inlet, Alaska, during 2001. Report from Greeneridge Sciences, Inc., Aptos, CA, for NMFS, Alaska Region, Anchorage, AK. [Paper available from NMFS, Alaska Region, 222 W 7th Ave., Box 43, Anchorage, AK 99513].

Bossart, G. D., T. A. Brawner, C. Cabal, M. Kuhns, E. A. Eimstad, J. Caron, M. Trimm, and P. Bradley. 1990. Hepatitis B-like infection in a Pacific white-sided dolphin (*Lagenorhynchus obliquidens*). J. Amer. Vet. Med. Assoc. 196(1):127-130.

Bowenkamp, K. E., S. Frasca, Jr., A. Draghi, II, G. J. Tsongalis, C. Koerting, L. Hinckley, S. De Guise, R. J. Montali, C. E. C. Goertz, D. J. St. Aubin, and J. L. Dunn. 2001. *Mycobacterium marinum* dermatitis and panniculitis with chronic pleuritis in a captive white whale (*Delphinapterus leucas*) with aortic rupture. J. Vet. Diagn. Invest. 13:524-530.

Braham, H. W. 1984. Review of reproduction in the white whale, *Delphinapterus leucas,* narwhal, *Monodon monoceros,* and Irrawaddy dolphin, *Orcaella brevirostris,* with comments on stock assessment. Rep. Int. Whal. Comm. (Spec. Issue 6):81-89.

Brandly, P. J., and R. Rausch. 1950. A preliminary note on trichinosis investigations in Alaska. Arctic 3:105-107.

Brandon, J., and P. R. Wade. 2006. A stock assessment of the Bering-Chukchi-Beaufort Seas stock of bowhead whales using Bayesian model averaging. J. Cetacean Res. Manage. 8(3):225–239.

Breton-Provencher, M. 1981. Survey of the beluga whale population in the Poste-de-la-Baleine region. Unpubl. Doc. Submitted to Int. Whal. Comm. (SC/32/SM16) [Paper available from http://www.iwcoffice.org/].

Brodie, P. F. 1969. Mandibular layering in *Delphinapterus leucas* and age determination. Nature 221:956-968.

Brodie, P. F. 1971. A reconsideration of aspects of growth, reproduction, and behavior of the white whale (*Delphinapterus leucas*) with reference to the Cumberland Sound, Baffin Island, population. J. Fish. Res. Bd. Can. 28:1309-1318.

Brodie, P. F., J. L. Parsons, and D. E. Sergeant. 1981. Present status of the white whale (*Delphinapterus leucas*) in Cumberland Sound, Baffin Island. Rep. Int. Whal. Comm. 31:579-582.

Brodie, P. F. 1982. The beluga (*Delphinapterus leucas*): Growth at age based on a captive specimen and a discussion of factors affecting natural mortality estimates. Rep. Int. Whal. Comm. 32:445-447.

Brodie, P. F., J. R. Geraci, and D. J. St. Aubin. 1990. Dynamics of tooth growth in beluga whales, *Delphinapterus leucas*, and effectiveness of tetracycline as a marker for age determination, p. 144-168. *In* T. G. Smith, D. J. St. Aubin, and J. R. Geraci (editors), Advances in research on the beluga whale, *Delphinapterus leucas*. Can. Bull. Fish. Aquat. Sci. 224.

Brown, J. H. 1995. Macroecology. University of Chicago Press, Chicago, IL.

Brown Gladden, J. G., M. M. Ferguson, M. K. Friesen, and J. W. Clayton. 1999. Population structure of North American beluga whales (*Delphinapterus leucas*) based on nuclear DNA microsatellite variation and contrasted with the population structure revealed by mitochondrial DNA variation. Mol. Ecol. 8:347–363.

Buck, J.D., L.L. Shepard, P.M. Bubucis, S. Spotte, K. McClave and R.A. Cook. 1989. Microbiological characteristics of white whale (*Delphinapterus leucas*) from capture through extended captivity. Can. J. Fish. Aquat. Sci. 46:1914-1921.

Burek, K. 1999a. Biopsy report of beluga whale: Case No. 98V0581. Rep. to NMFS, Alaska Region, Anchorage, AK [Paper available from NMFS, Alaska Region, 222 W 7th Ave., Box 43, Anchorage, AK, 99513].

Burek, K. 1999b. Biopsy report of beluga whale: Case No. 99V0269. Rep. to NMFS, Alaska Region, Anchorage, AK [Paper available from NMFS, Alaska Region, 222 W 7th Ave., Box 43, Anchorage, AK, 99513].

Burns, J. J., and G. A. Seaman. 1986. Investigations of belukha whales in coastal waters of western and northern Alaska. II. Biology and ecology. U.S. Dep. Commer., NOAA, OCSEAP Final Rep. 56(1988): 221-357.

Calkins, D. G. 1989. Status of belukha whales in Cook Inlet, p. 109-112. *In* L. E. Jarvela and L. K. Thorsteinson (editors.), U.S. Dep. Commer., NOAA, OCSEAP Gulf of Alaska, Cook Inlet, and North Aleutian Basin information update meeting. Anchorage, AK, Feb. 7-8, 1989. Anchorage, AK.

Callan, R. J., G. Early, H. Kida, and V. S. Hinshaw. 1995. The appearance of H3N3 influenza viruses in seals. J. Gen. Virol. 76:199-203.

Calle, P. P., D. E. Kenny, and R. A. Cook. 1993. Successful treatment of suspected *Erysipelas septicemia* in a beluga whale (*Delphinapterus leucas*). Zoo Biol. 12:483-490.

Clausen, D. M. 1981. Summer food of Pacific cod, *Gadus macrocephalus*, in coastal waters of Southeastern Alaska. Fish. Bull., U.S. 78:968-973.

Cohen, D. M., T. Inada, T. Iwamoto, and N. Scialabba. 1990. FAO species catalogue. Vol. 10. Gadiform fishes of the world (Order Gadiformes). An annotated and illustrated catalogue of cods, hakes, grenadiers and other gadiform fishes known to date. FAO Fisheries Synopsis 10 (125):1-442.

Colborn, T., and M. J. Smolen. 1996. Epidemiological analysis of persistent organochlorine contaminants in cetaceans. Rev. Environ. Contamin. Toxicol. 146:91–172.

Conrad, P. A., M. A. Miller, C. Kreuder, E. R. James, J. Mazet, H. Dabritz, D. A. Jessup, F. Gulland, and M. E. Grigg. 2005. Transmission of Toxoplasma: clues from the study of sea otters as sentinels of *Toxoplasma gondii* flow into the marine environment. Int. J. Parasitol. 35(11-12):1155-1168.

Courchamp, F., T. Clutton-Brock, and B. Grenfell. 1999. Inverse density dependence and the Allee effect. Trends Ecol. Evol. 14(10):405-410.

Dailey, M. D. 2001. Parasitic diseases, p. 357-379. *In* L. A. Dierauf and F. M. D. Gulland (editors), CRC handbook of marine mammal medicine, 2nd Edition. CRC Press, Boca Raton, Florida.

Dailey, M. D., F. M. D. Gulland, L. J. Lowenstine, P. Silvangni, and D. Howard. 2000. Prey, parasites and pathology associated with the mortality of a juvenile gray whale (*Eschrichtius robustus*) stranded along the northern California coast. Dis. Aquat. Org. 42:111-117.

Das, K., U. Siebert, M. Fontaine, T. Jauniaux, L. Holsbeek, J. M. Bouquegneau. 2004. Ecological and pathological factors related to trace metal concentrations in harbour porpoises *Phocoena phocoena* from the North Sea and adjacent areas. Mar. Ecol. Prog. Ser. 281:283–295.

Daszak, P., A. A. Cunningham, and A. D. Hyatt. 2000. Emerging infectious diseases of wildlife – threats to biodiversity and human health. Science 287:443-449.

Daszak, P., L. Berger, A. A. Cunningham, A. D. Hyatt, D. E. Green, and R. Speare. 1999. Emerging infectious diseases and amphibian population declines. Emerg. Infect. Dis. 5:735-748.

Davis, R. A., and C. R. Evans. 1982. Offshore distribution and numbers of white whales in the eastern Beaufort Sea and Amundsen Gulf, summer 1981. Report for SOHIO Alaska Petroleum Co., Anchorage, and Dome Petroleum Ltd., Calgary, Alberta, by LGL Ltd. 76 p.

Davis, R. A., and K. J. Finley. 1979. Distribution, migrations, abundance and stock identity of eastern Arctic white whales. Unpubl. doc. Submitted to Int. Whal. Comm. (SC/31/SM10). [Paper available from http://www.iwcoffice.org/].

de Castro, F., and B. Bolker. 2005. Mechanisms of disease-induced extinction. Ecol. Lett. 8:117-126.

De Guise, S., A. Lagacé, and P. Béland. 1993 Intramuscular *Sarcocystis* in two beluga whales and an Atlantic white-sided dolphin from the St. Lawrence estuary, Quebec, Canada. J. Vet. Diagn. Invest. 5:296-300.

De Guise, S., A. Lagacé, and P. Beland. 1994. Tumors in twenty-four St. Lawrence beluga whales (*Delphinapterus leucas*). Vet. Pathol. 31:444-449.

De Guise, S., A. Lagacé, P. Béland, C. Girard, and R. Higgins. 1995a. Non-neoplastic lesions in beluga whales (*Delphinapterus leucas*) and other marine mammals from the St. Lawrence estuary. J. Comp. Pathol. 112:257-271.

De Guise, S., D. Martineau, P. Béland, and M. Fournier. 1995b. Possible mechanisms of action of environmental contaminants on St. Lawrence beluga whales (*Delphinapterus leucas*). Environ. Health Perspect. 103(S4):73-77.

de Swart, R. L. 1995. Impaired immunity in seals exposed to bioaccumulated environmental contaminants. Ph.D. thesis. Erasmus Univ., Rotterdam, The Netherlands.

de Swart R. L., T. C. Harder, P. S. Ross, H. W. Vos, and A. D. Osterhaus. 1995. Morbilliviruses and morbillivirus diseases of marine mammals. Infect. Agents Dis. 3:125-130.

de Swart, R. L., P. S. Ross, J. S. Vos, and A. D. M. E. Osterhaus. 1996. Impaired immunity in harbour seals (*Phoca vitulina*) exposed to bioaccumulated environmental contaminants – review of a long term feeding study. Environ. Health Perspect. 104:823-828.

Deem, S. L., W. B. Karesh, and W. Weisman. 2001. Putting theory into practice: Wildlife health in conservation. Conserv. Biol. 15:1224-1233.

Degollada, E., M. André, M. Arbelo, and A. Fernández. 2002. Incidence, pathology and involvement of *Nasitrema* species in odontocete strandings in the Canary Islands. Vet. Rec. 150:81-82.

DFO (Department of Fisheries and Oceans). 1995. Saint Lawrence beluga whale recovery plan. Saint Lawrence beluga whale recovery team. Dept. Fish. Oceans and World Wildlife Fund, Canada. 73 p.

EBASCO Environmental. 1990a. Summary report: Cook Inlet discharge monitoring study: produced water (discharge number 016) Sept. 1988-Aug. 1989. Prepared for Amoco Production Co., ARCO Alaska Inc., Marathon Oil Co., Phillips Petroleum Co., Shell Western E&P Inc., Texaco Inc., Unocal Corp., Anchorage, Alaska and U.S. Environmental Protection Agency, Region 10, Seattle, WA.

EBASCO Environmental. 1990b. Comprehensive report: Cook Inlet discharge monitoring study: Apr. 1987-Jan. 1990. Prepared for Amoco Production Co., ARCO Alaska Inc., Marathon Oil Co., Phillips Petroleum Co., Shell Western E&P Inc., Texaco Inc., Unocal Corp., Anchorage, Alaska and U.S. Environmental Protection Agency, Region 10, Seattle, WA.

Eschmeyer, W. N., E. S. Herald, and H. Hammann. 1983. A field guide to Pacific coast fishes of North America. Houghton Mifflin Company, Boston, MA.

Fay, J. A., D .J. Foster, and R. T. Stanek. 1984. The use of fish and wildlife resources in Tyonek, Alaska. Alaska Dep. Fish Game, Div. Subsistence, Anchorage, AK, Tech. Rep. Ser. 105.

Fayer, R., J. P. Dubey, and D. S. Lindsay. 2004. Zoonotic protozoa: From land to sea. Trends Parasitol. 20(11):531-536.

FHWA (Federal Highways Administration) and KABATA (Knik Arm Bridge and Toll Authority). 2006. Knik Arm Crossing Project, Draft Environmental Impact Statement and Draft Section 4(f) Evaluation. Prepared by HDR, Inc., for KABATA, Anchorage, Alaska.

Forbes, L. B. 2000. The occurrence and ecology of *Trichinella* in marine mammals. Vet. Parasitol. 93:321-334.

Ford, J. K. B., G. M. Ellis, and K. Balcomb. 2000. Killer whales: The natural history and genealogy of *Orcinus orca* in British Columbia and Washington. UBC Press, Vancouver, British Columbia, Canada.

Funk, D.W., T.M. Markowitz, and R. Rodrigues (editors). 2005. Baseline studies of beluga whale habitat use in Knik Arm, Upper Cook Inlet, Alaska, July 2004-July 2005. Rep. from LGL Alaska Research Associates, Inc., Anchorage, AK, in association with HDR Alaska, Inc., Anchorage, AK, for Knik Arm Bridge and Toll Authority, Anchorage, AK, Department of Transportation and Public Facilities, Anchorage, AK, and Federal Highway Administration, Juneau, AK. 232 p.

Gaydos, J. K., K. C. Balcomb, R. W. Osborne, and L. Dierauf. 2004. Evaluating potential infectious disease threats for southern resident killer whales, *Orcinus orca*: a model for endangered species. Biol. Conserv. 117:253-262.

Gaydos, J. K., W. A. Miller, K. V. Gilardi, A. Melli, H. Schwantje, C. Engelstoft, H. Fritz, and P. A. Conrad. 2007. Cryptosporidium and Giardia in marine-foraging river otters (*Lontra canadensis*) from the Puget Sound Georgia Basin ecosystem. J. Parasitol. 93(1):198-202.

Geraci, J. R., and V. J. Lounsbury. 2005. Marine mammals ashore: a field guide for strandings. 2nd ed. National Aquarium in Baltimore, Baltimore, MD.

Geraci, J. R., and D. J. St. Aubin. 1987. Effects of parasites on marine mammals. Int. J. Parasitol. 17:407-414.

Geraci, J. R., D. J. St. Aubin, I. K. Barker, R. G. Webster, V. S. Hinshaw, W. J. Bean, H. L. Ruhnke, J. H. Prescott, G. Early, A. S. Baker, S. Madoff, and R. T. Schooley. 1982. Mass mortality of harbor seals: Pneumonia associated with influenza A virus. Science 215:1129-1131.

Goetz, K. T., D. J. Rugh, A. J. Read, and R. C. Hobbs. 2007. Habitat use in a marine ecosystem: Beluga whales *Delphinapterus leucas* in Cook Inlet, Alaska. Mar. Ecol. Prog. Ser. 330:247-256.

Goren, A. D., P. F. Brodie, S. Spotte, G. C. Ray, W. H. Kaufman, A. J. Gwinnett, J. J. Sciubba, and J. D. Buck. 1987. Growth layer groups (GLGs) in the teeth of an adult belukha whale (*Delphinapterus leucas*) of known age: Evidence for two annual layers. Mar. Mammal Sci. 3:14-21.

Hall, A. J., K. Hugunin, R. Deaville, R. J. Law, C. R. Allchin, and P. D. Jepson. 2006. The risk of infection from polychlorinated biphenyl exposure in harbor porpoise (*Phocoena phocoena*): a case-control approach. Environ. Health Perspect. 114:704-711.

Hanni, K. D., J. A. K. Mazet, F. M. D. Gulland, J. Estes, M. Staedler, M. J. Murray, M. Miller, and D. A. Jessup. 2003. Clinical pathology and assessment of pathogen exposure in southern and Alaskan sea otters. J. Wildl. Dis. 39(4):837-850.

Harvell, C. D., C. E. Mitchell, J. R. Ward, S. Altizer, A. P. Dobson, R. S. Ostfield, and M. D. Samuel. 2002. Climate warming and disease risks for terrestrial and marine biota. Science 296:2158–2162.

Hazard, K. 1988. Beluga whale, *Delphinapterus leucas,* p. 195-235. *In* J. W. Lentfer (editor), Selected marine mammals of Alaska: Species accounts with research and management recommendations. Mar. Mammal Comm., Washington, D.C.

Heide-Jørgensen, M. P., J. Jensen, A. H. Larsen, J. Teilmann, and B. Neurohr. 1994. Age estimation of white whales (*Delphinapterus leucas*) from Greenland. Meddr Gronland, Biosci. 39:187-193.

Heide-Jørgensen M. P., P. Richard, M. Ramsay, and S. Akeeagok. 2002. Three recent ice entrapments of Arctic cetaceans in West Greenland and the eastern Canadian High Arctic. NAMMCO Sci. Publ. 4:143-148.

Helle, E., M. Olsson, and S. Jensen. 1976. PCB levels correlated with pathological changes in seal uteri. Ambio 5:261-263.

Higgins, R. 2000. Bacteria and fungi of marine mammals: a review. Can. Vet. J. 41(2):105-116.

Hinshaw, V. S., W. J. Bean, R. G. Webster, J. E. Rehg, P. Fiorelli, G. Early, J. R. Geraci, and D. J. St. Aubin. 1984. Are seals frequently infected with avian influenza viruses? J. Virol. 51:863-865.

Hobbs, R. C., K. L. Laidre, D. J. Vos, B. A. Mahoney, and M. Eagleton. 2005. Movements and area use of belugas, *Delphinapterus leucas*, in a subarctic Alaskan estuary. Arctic 58(4): 331-340.

Hobbs, R. C., D. J. Rugh, and D. P. DeMaster. 2000a. Abundance of beluga whales, *Delphinapterus leucas,* in Cook Inlet, Alaska, 1994-2000. Mar. Fish. Rev. 62(3):37-45.

Hobbs, R. C., J. M. Waite, and D. J. Rugh. 2000b. Beluga, *Delphinapterus leucas*, group sizes in Cook Inlet, Alaska, based on observer counts and aerial video. Mar. Fish. Rev. 62(3): 46-59.

Hobbs, R. C., K. E. W. Shelden, D. J. Vos, K. T. Goetz, and D. J. Rugh. 2006. Status review and extinction assessment of Cook Inlet belugas (*Delphinapterus leucas*). AFSC Processed Rep. 2006-16, 74 p. Alaska Fish. Sci. Cent., Natl. Mar. Fish. Serv., NOAA, 7600 Sand Point Way NE., Seattle, WA 98115-6349.

Hohn A. A., and C. Lockyer. 1999. Growth layer patterns in teeth from two known-history beluga whales: Reconsideration of deposition rates. Unpubl. doc. Submitted Int. Whal. Comm. (SC/51/SM4). 12 p + figures. [Paper available from http://www.iwcoffice.org/].

Houde, M., L. N. Measures, and J. Huot. 2003. Lung worm (*Pharurus pallasii*: Metastrongyloidea: Pseudaliidae) infection in the endangered St. Lawrence beluga whale (*Delphinapterus leucas*). Can. J. Zool. 81:543-551.

Howard, E. B., J. O. Britt, G. K. Marsumoto, R. Itahara, and C. N. Nagano. 1983. Bacterial Diseases, p. 70-118. *In* E. B. Howard (editor), Pathology of marine mammal diseases, Vol. 1. CRC Press, Boca Raton, FL.

Hughes-Hanks, J. M., L.G. Rickard, C. Panuska, J. R. Saucier, T. M. O'Hara, L. Dehn, and R. M. Rolland. 2005. Prevalence of *Cryptosporidium* spp. and *Giardia* spp. in five marine mammal species. J. Parasitol. 91(5):1225-1228.

Huntington, H. P. 2000. Traditional knowledge of the ecology of beluga whale, *Delphinapterus leucas*, in Cook Inlet, Alaska. Mar. Fish. Rev. 62(3):134-140.

IUCN (World Conservation Union). 2004. Red list of threatened species. IUCN, Gland, Switzerland. Available from http://www.iucn.org/themes/ssc/redlist.htm

Ivashin, M. V., and K. V. Shevlagin. 1987. The white whale (*Delphinapterus leucas* Pallas, 1776): Entrapment and escape in the ice of Senjavin Strait, USSR. Rep. Int. Whal. Comm. 37:357-359.

IWC (International Whaling Commission). 2000. Annex I: report of the sub-committee on small cetaceans. J. Cetacean Res. Manage. 2 (Suppl.): 235-264.

IWC (International Whaling Commission). 2007. Appendix K, Report of the Standing Working Group on Environmental Concerns. Scientific Committee Report, The International Whaling Commission's 59th annual meeting in Anchorage, Alaska, USA 2007. IWC, Cambridge, United Kingdom.[Available from http://www.iwcoffice.org/sci_com/screport.htm].

Jepson, P. D. S., Brew, A. P. MacMillan, J. R. Baker, J. Barnett, J. K. Kirkwood, T. Kuiken, I. R. Robinson, and V. R. Simpson. 1997. Antibodies to *Brucella* in marine mammals around the coast of England and Wales. Vet. Rec. 141:513-515.

Jepson, P. D., P. M. Bennett, C. R. Allchin, R. J. Law, T. Kuiken, J. R. Baker, E. Rogan, and J. K. Kirkwood. 1999. Investigating potential associations between chronic exposure to polychlorinated biphenyls and infectious disease mortality in harbor porpoises from England and Wales. Sci. Total Environ. 243-244:339-348.

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. Environ. Toxicol. Chem. 24:238-248.

Johnson, S. P., S. Nolan, and F. M. D. Gulland. 1998. Antimicrobial susceptibility of bacteria isolated from pinnipeds stranded in central and northern California. J. Zoo Wildl. Med. 29(3):288-294.

Kannan, K., J. L. Reiner, S. H. Yun, E. E. Perrotta, L. Tao, B. Johnson-Restrepo, and B. D. Rodan. 2005. Polycyclic musk compounds in higher trophic level aquatic organisms and humans from the United States. Chemosphere 61(5):693-700.

Kass, R. E., and A. E. Raftery. 1994. Bayes Factors. Tech. Rep. 254, Dept. Stat., Univ. Washington, and Tech. Rep. 571, Dept. Stat., Carnegie-Mellon Univ. 56 p.

Kennedy, S., I. J. Lindstedt, M. M. McAliskey, S. A. McConnell, and S. J. McCullough. 1992. Herpesviral encephalitis in a harbor porpoise (*Phocoena phocoena*). J. Zoo Wildl. Med. 23:374-379.

Kennedy-Stoskopf, S. 2001. Viral diseases, p. 285–307. *In* L. A. Dierauf and F. M. D Gulland (editors), CRC handbook of marine mammal medicine, 2nd Edition, CRC Press, Boca Raton, FL.

Kenyon, A. J., and B. J. Kenyon. 1977. Prevalence of *Pharurus pallasii* in the beluga whale (*Delphinapterus leucas*) of Churchill River Basin, Manitoba. J. Wildl. Dis. 13:338-340.

Khuzin, R. S. 1961. The procedure used in age determination and materials relating to reproduction of the beluga. Nauchno-Tekh. Byull. TINRO 1(15):58-60.

Kleinenberg, S. E., A. V. Yablokov, B. M. Bel'kovich, and M. N. Tarasevich. 1964. Beluga (*Delphinapterus leucas*). Investigations of the species. Izdatel'stvo Nauka, Moscow. [Translated from Russian by the Israel Program for Scientific Translation, Jerusalem, 1969]. 376 p.

Klinkhart, E. G. 1966. The beluga whale in Alaska. Alaska Dep. Fish. Game Fed. Aid in Wildlife Restoration Proj. Rep. Vol. VII.

Lafferty, K. D., and L. R. Gerber. 2002. Good medicine for conservation biology: the intersection of epidemiology and conservation theory. Conserv. Biol. 16(3):593-604.

Laidre, K. L., K. E. W. Shelden, D. J. Rugh, and B. A. Mahoney. 2000. Beluga, *Delphinapterus leucas*, distribution and survey effort in the Gulf of Alaska. Mar. Fish. Rev. 62(3): 27-36.

Lambertsen, R. 1986. Disease of the common fin whale crassicaudiosis of the urinary system. J. Mammal. 67(2):353-366.

Lambertsen, R. 1992. Crassicaudiosis: a parasitic disease threatening the health and population recovery of large baleen whales. Rev. Sci. Tech. Off. Int. Epizool. 11(4):1131-1141.

Lenghaus, C., M. J. Studdert, and D. Gavier-Widén. 2001. Calicivirus infections, p. 280-291. *In* E. S. Williams and I. K. Barker (editors), Infectious diseases of wild mammals. Iowa State Univ. Press, Ames, IA.

Lentfer, J. W. (editor). 1988. Selected marine mammals of Alaska: Species accounts with research and management recommendations. Mar. Mammal Comm., Washington, D.C.

Lerczak, J. A., K. E. W. Shelden, and R. C. Hobbs. 2000. Application of suction-cup attached VHF transmitters to the study of beluga, *Delphinapterus leucas,* surfacing behavior in Cook Inlet, Alaska. Mar. Fish. Rev. 62(3): 99-111.

Lesage, V., and M. C. S. Kingsley. 1998. Updated status of the St. Lawrence River population of the beluga, *Delphinapterus leucas*. Can. Field-Nat. 112(1): 98-114.

Lewis, R. J., and K. Berry. 1988. Brain lesions in a Pacific white-sided dolphin (*Lagenorhynchus obliquidens*). J. Wildl. Dis. 24(3):577-581.

Litzky, L. K. 2001. Monitoring recovery status and age structure of Cook Inlet, Alaska beluga whale by skin color determination. M.S. thesis, Univ. Washington. 76 p.

Litzow, M. A., K. M. Bailey, F. G. Prahl, and R. Heintz. 2006. Climate regime shifts and reorganization of fish communities: the essential fatty acid limitation hypothesis. Mar. Ecol. Prog. Ser. 315: 1-11

Lockyer, C., A. A. Hohn, W. D. Doidge, M. P. Heide-Jørgensen, and R. Suydam. 2007. Age determination in belugas (*Delphinapterus leucas*): A quest for validation of dentinal layering. Aquat. Mammals 33:293-304

Loughlin, T. R. 1994. Marine mammals and the *Exxon Valdez*. Academic Press, San Diego, CA.

MacNeil, A. C., T. A. Gornall, W. E. Giddens, and J. Boyce. 1978. Evidence of *Nocardia* sp. in a captive-born beluga whale. Aquat. Mammals 6:50-53.

Mahoney, B. A., and K. E. W. Shelden. 2000. Harvest history of beluga whale, *Delphinapterus leucas*, in Cook Inlet, Alaska. Mar. Fish. Rev. 62(3): 124-133.

Margolis, H. S., J. P. Middaugh, and R. D. Burgess. 1979. Arctic trichinosis: Two Alaskan outbreaks from walrus meat. J. Infect. Dis. 139:102–105.

MMC (Marine Mammal Commission). 1989. Annual report of the Marine Mammal Commission, calendar year 1988: a report to Congress. Marine Mammal Commission, Washington, D.C. 232 p.

Martin, W. E., C. K. Haun, H. S. Barrows, and H. Cravioto. 1970. Nematode damage to brain of striped dolphin, *Lagenorhynchus obliquidens*. Trans. Amer. Microsc. Soc. 89(2): 200-205.

Martineau, D., A. Lagacé, P. Béland, R. Higgins, D. Armstrong, and L. R. Shugart. 1988. J. Comp. Pathol. 98(3):287-311.

Martineau, D., S. De Guise, M. Fournier, L. Shugart, C. Girard, A. Lagacé, and P. Béland. 1994. Pathology and toxicology of beluga whales from the St. Lawrence Estuary, Quebec, Canada. Past, present and future. Sci. Total Environ. 154:201-215.

Martineau, D., S. Lair, S. De Guise, T. P. Lipscomb, and P. Béland. 1999. Cancer in beluga whales from the St. Lawrence estuary, Quebec, Canada: A potential biomarker of environmental contamination. J. Cetacean Res. Manage. 1:249-265.

Martineau, D., D. Lemberger, A. Dallaire, P. Labelle, T. P. Lipscomb, P. Michel, and I. Mikaelian. 2002. Cancer in wildlife, a case study: Beluga from the St. Lawrence estuary, Québec, Canada. Environ. Health Perspect. 110(3):285-292.

Mazzone, W. S. 1987. Walrus, *Odobenus rosmarus*, and whale interactions: an eyewitness account. Can. Field-Nat. 101(4):590-591.

McCallum, H. I., and A. Dobson. 1995. Detecting disease and parasite threats to endangered species and ecosystems. Trends Ecol. Evol. 10(5):190-194.

Measures, L. 2001. Lung worms of marine mammals, p. 279-300. *In* W. M. Samuel, M. J. Pybus, and A. A. Kocan (editors), Parasitic diseases of wild mammals. Iowa State Univ. Press, Ames, IA.

Measures, L. N., and M. Olson. 1999. *Giardiasis* in pinnipeds from eastern Canada. J. Wildl. Dis. 35(4):779-782.

Measures, L. N., P. Béland, D. Martineau, and S. De Guise. 1995. Helminths of an endangered population of belugas, *Delphinapterus leucas*, in the St. Lawrence estuary, Canada. Can. J. Zool. 73:1402–1409.

Mikaelian I, J. Boisclair, J. P. Dubey, S. Kennedy, D. Martineau. 2000. Toxoplasmosis in beluga whales (*Delphinapterus leucas*) from the St. Lawrence estuary: Two case reports and a serological survey. J. Comp. Pathol. 122:73-76.

Mikaelian, I., M. P. Tremblay, C. Montpetit, S. V. Tessaro, H. J. Cho, C. House, L. Measure, D. Martineau. 1999. Seroprevalence of selected viral infections in a population of beluga whales (*Delphinapterus leucas*) in Canada. Vet. Rec. 144(2):50-51.

Miller, M. A., I. A. Gardner, C. Kreuder, D. M. Paradies, K. R. Worcester, D. A. Jessup, E. Dodd, M. D. Harris, J. A. Ames, A. E. Packham, and P. A. Conrad. 2002. Coastal freshwater runoff is a risk factor for *Toxoplasma gondii* infection of southern sea otters (*Enhydra lutris nereis*). Int. J. Parasitol. 32:997-1006.

MMS (Minerals Management Service). 1996. Cook Inlet planning area oil and gas lease sale 149. Final Environmental Impact Statement. U.S. Dept. Interior Alaska OCS Region.

Minnette, H. 1986. Salmonellosis in the marine environment: a review and commentary. Int. J. Zoonoses 13:71-75.

Montgomery, R. A., J. M. Ver Hoef, and P. L. Boveng. 2005. Spatial modeling of haul-out site use by harbor seals in Cook Inlet, Alaska. Mar. Ecol. Prog. Ser. 341:257-264.

Moore, S. E., K. E. W. Shelden, L. K. Litzky, B. A. Mahoney, and D. J. Rugh. 2000. Beluga, *Delphinapterus leucas,* habitat associations in Cook Inlet, Alaska. Mar. Fish. Rev. 62(3): 60-80.

Moore, S. E., D. Rugh, K. Shelden, B. Mahoney, and R. Hobbs (editors). 1999. Synthesis of available information on the Cook Inlet stock of beluga whales. AFSC Processed Rep. 99-06, 22 p. Alaska Fish. Sci. Cent., NOAA, Natl. Mar. Fish. Serv., 7600 Sand Point Way NE, Seattle, WA 98115.

Mörner, T. 2001. Salmonellosis, p. 505-506. *In* E. S. Williams and I. K. Barker (editors), Infectious diseases of wild mammals. Iowa State University Press, Ames, IA.

Morris, B. F. 1988. Cook Inlet beluga whales. Unpubl. paper. Alaska Region, NMFS, Anchorage, Alaska. 29 p. [Paper available from NMFS, Alaska Region, 222 W 7th Ave., Box 43, Anchorage, AK, 99513].

Morris, W. F., and D. F. Doak. 2002. Quantitative conservation biology: Theory and practise of population viability analysis. Sinauer Associates, Inc., Sunderland, MA.

Morrow, J. E. 1980. The freshwater fishes of Alaska. Univ. B.C. Animal Resources Ecology Library. 248 p.

Moulton, L. L. 1997. Early marine residence, growth, and feeding by juvenile salmon in northern Cook Inlet, Alaska. Alaska Fish. Res. Bull. 4(2):154-77.

MOA (Municipality of Anchorage). 2006. 2005 Annual report, NPDES Permit No. AKS05255-8. Document No. WMP PMr05001. Anchorage, AK.

Munn, C.B. 2006. Viruses as pathogens of marine organisms-from bacteria to whales. J. Mar. Biol. Assoc. UK 86:453-467.

Murray, N. K., and F. H. Fay. 1979. The white whales or belukhas, *Delphinapterus leucas*, of Cook Inlet, Alaska. Unpubl. Doc. Submitted to Int. Whal. Comm. (SC/31/SM12). [Paper available from http://www.iwcoffice.org/].

NMFS (National Marine Fisheries Service). 1992. Status report on Cook Inlet belugas (*Delphinapterus leucas*). Unpublished report prepared by the Alaska Region, NMFS, 222 W 7th Ave., Box 43, Anchorage, AK 99513.

NMFS (National Marine Fisheries Service). 2005. Conservation plan for the Cook Inlet beluga whale (*Delphinapterus leucas*). Natl Mar. Fish. Serv., Juneau, Alaska. [available from http://www.fakr.noaa.gov/protectedresources/whales/beluga/mmpa/draft/conservationplan032005.pdf].

NOAA (National Oceanic and Atmospheric Administration). 2003. Subsistence harvest management of Cook Inlet beluga whales. Final Environmental Impact Statement, Alaska Region, National Marine Fisheries Service, July 2003. 178 p.

Neff, J. M., and G. S. Douglas. 1994. Petroleum and hydrocarbons in the water and sediments of upper Cook Inlet, Alaska, near a produced water outfall. Submitted to Marathon Oil Co., Anchorage, AK, by Battelle Ocean Sci. Lab., Duxbury, MA. 30 p.

Newman, S. J., and S. A. Smith. 2006. Marine mammal neoplasia: a review. Vet. Pathol. 43:865-880.

Nielsen O., R. E. A. Stewart, L. Measures, P. Duignan, and C. House. 2000. A morbillivirus antibody survey of Atlantic walrus, narwhal and beluga in Canada. J. Wildl. Dis. 36:508-517.

Nielsen O., A. Clavijo, and J. A. Boughen. 2001a. Serologic evidence of influenza A infection in marine mammals of arctic Canada. J. Wildl. Dis. 37(4):820-825.

Nielsen O., R. E. A. Stewart, K. Nielsen, L. Measures, and P. Duignan. 2001b. Serologic survey of *Brucella* spp. antibodies in some marine mammals of North America. J. Wildl. Dis. 37:89-100.

Nollens, H. H., J. A. Hernandez, E. R. Jacobson, M. Haulena, and F. M. D. Gulland. 2005. Risk factors associated with development of poxvirus lesions in hospitalized California sea lions. J. Amer. Vet. Med. Assoc. 227(3):467-473.

O'Brien, S. J., and J. F. Evermann. 1988. Interactive influence of infectious disease and genetic diversity in natural populations. Trends Ecol. Evol. 3:254-259.

O'Corry-Crowe, G. M. 2002. Beluga whale (*Delphinapterus leucas*), p. 94-99. *In* W. F. Perrin, B. Wursig, and J. G. M. Thewissen (editors), Encyclopedia of marine mammals. Academic Press, New York, NY.

O'Corry-Crowe, G. M., R. S. Suydam, A. Rosenberg, K. J. Frost, and A. E. Dizon. 1997. Phylogeography, population structure and dispersal patterns of the beluga whale *Delphinapterus leucas* in the western Nearctic revealed by mitochondrial DNA. Mol. Ecol. 6:955-970.

O'Corry-Crowe, G. M., A. E. Dizon, R. S. Suydam, and L. F. Lowry. 2002. Molecular genetic studies of population structure and movement patterns in a migratory species: The beluga whale, *Delphinapterus leucas*, in the western Nearctic, p. 53-64. *In* C. J. Pfeiffer (editor), Molecular and cell biology of marine mammals. Krieger Publishing Company, Malabar, FL.

O'Corry-Crowe, G., W. Lucey, C. Bonin, E. Henniger, and R Hobbs. 2006. The ecology, status and stock identity of beluga whales, *Delphinapterus leucas,* in Yakutat Bay, Alaska. Report to the U.S. Mar. Mammal Commission, February 2006. 22 p.

O'Corry-Crowe, G., C. Bonin, and A. Frey. 2007. Molecular genetic analysis of population structure, dispersal and gene flow of beluga whales, *Delphinapterus leucas,* in the western nearctic: New findings on the Cook Inlet population. Interim report to the National Marine Fisheries Service – Alaska Region, Juneau, October 1, 2007. 25 p.

O'Corry-Crowe, G. M., R. S. Suydam, R. Hobbs, L. Quakenbush, and B. Mahoney. 2008. Molecular genetic analysis of population structure, dispersal and gene flow of beluga whales, *Delphinapterus leucas,* in the western nearctic: New findings on the Cook Inlet population, p. 136. *In* Alaska Marine Science Symposium 2008, book of abstracts for oral presentations and posters. January 20-23, 2008, Anchorage, Alaska. [Available at: http://www.alaskamarinescience.org/Abstract%20Book%202008.pdf].

Ognetov, G. N. 1981. Studies on the ecology and the taxonomy of the white whale (*Delphinapterus leucas* Pall., 1776) inhabiting the Soviet Arctic. Rep. Int. Whal. Comm. 31:515-520.

O'Hara, T., and V. Woshner. 2006. Personal communication via K. Burek, AVPS, Eagle River, AK.

O'Hara, T. M., and T. J. O'Shea. 2001. Toxicology, p. 471-520. *In* L.A. Dierauf and F.M.D. Gulland (editors), CRC handbook of marine mammal medicine, 2nd Edition. CRC Press, Boca Raton, FL.

Ohishi, K., T. Maruyama, N. Kishida, H. Kida, A. Ninomiya, Y. Takada, N. Miyazaki, and A. N. Boltonov. 2004. Antibodies to human-related H3 influenza A virus in Baikal seals (*Phoca sibirica*) and ringed seals (*Phoca hispida*) in Russia. Microbiol. Immunol. 48:905-909.

Ohishi, K., A. Ninomiya, H. Kida, C. H. Park, T. Maruyama, T. Arai, E. Katsumata, T. Toboyama, A. N. Boltonov, L .S. Khuraskin, and N. Miyazaki. 2002. Serological evidence of transmission of human influenza A and B viruses to Caspian seals (*Phoca caspica*). Microbiol. Immunol. 46:639-644.

Ohsumi, S. 1979. Interspecies relationships among some biological parameters in cetaceans and estimation of the natural mortality coefficient of the southern hemisphere minke whale. Rep. Int. Whal. Comm. 29:397-406.

O'Shea, T. J., B. L. Homer, E. C. Greiner, and W. A. Layton. 1991. *Nasitrema* sp. associated encephalitis in a striped dolphin (*Stenella coeruleoalba*) stranded in the Gulf of Mexico. J. Wildl. Dis. 27:706-709.

Olson, M. E., P. D. Roach, M. Stabler, and W. Chen. 1997. *Giardiasis* in ringed seals from the western Arctic. J. Wildl. Dis. 33:646-648.

Osterhaus, A. D. M. E., G. F. Rimmelzwaan, B. E. E. Martina, T. M. Bestebroer, and R .A. M. Fouchier. 2000. Influenza B virus in seals. Science 288:1051-1053.

Perrin, W. F. (editor). 1982. Annex H: Report of the sub-committee on small cetaceans. Rep. Int. Whal. Comm. 32:113-125.

Philippa, J. D. W., F. A. Leighton, P. Y. Daoust, O. Nielsen, M. Pagliarulo, H. Schwantje, T. Shury, R. Van Herwijnen, B. E. E. Martina, T. Kuiken, M. W. G. Van de Bildt, and A. D. M. E. Osterhaus. 2004. Antibodies to selected pathogens in free-ranging terrestrial carnivores and marine mammals in Canada. Vet. Rec. 155:135-140.

Pierce, G. J., M. B. Santos, S. Murphy, J. A. Learmonth, A. F. Zuur, E. Rogan, P. Bustamante, F. Caurant, V. Lahaye, V. Ridoux, B. N. Zegers, A. Mets, M. Addink, C. Smeenk, T. Jauniaux, R. J. Law, W. Dabin, A. López, J. M. Alonso Farré, A. F. González, A. Guerra, M. García-Hartmann, R. J. Reid, C. F. Moffat, C. Lockyer, and J. P. Boon. 2007. Bioaccumulation of persistent organic pollutants in female common dolphins (*Delphinus delphis*) and harbour porpoises (*Phocoena phocoena*) from western European seas: Geographical trends, causal factors and effects on reproduction and mortality. Environ. Pollut. [available online]:1-15. doi:10.1016/j.envpol.2007.08.019

Punt, A. E., C. Allison, and G. Fay. 2004. An examination of assessment models for the eastern North Pacific gray whale based on inertial dynamics. J. Cetacean Res. Manage. 6:121-32.

Rausch, R. L. 1970. Trichinosis in the Arctic, p. 348-373. *In* S. E. Gould (editor), Trichinosis in man and animals. C. C. Thomas Publ., Springfield, IL.

Ray, G. C., D. Wartzok, and G. Taylor. 1984. Productivity and behavior of bowheads, *Balaena mysticetus*, and white whale, *Delphinapterus leucas*, as determined from remote sensing. Rep. Int. Whal. Comm. (Spec. Issue 6):199-209.

Reidarson, T. H., J. F. McBain, L. M. Dalton, and M. G. Rinaldi. 1999. Diagnosis and treatment of fungal infections in marine mammals, p. 478-485. *In* M. E. Fowler and R. E. Miller (editors), Zoo and wild animal medicine. W. B. Saunders, Philadelphia, PA.

Reidarson, T. H., J. F. McBain, L. M. Dalton, and M. G. Rinaldi. 2001. Mycotic diseases, p. 337-355. *In* L. A. Dierauf and F. M. D. Gulland (editors), CRC handbook of marine mammal medicine, 2nd Edition. CRC Press, Boca Raton, FL.

Robeck, T. R., and L. M. Dalton. 2002. *Saksenaea vasiformis* and *Apophysomyces elegans* zygomycotic infections in bottlenose dolphins (*Tursiops truncatus*), a killer whale (*Orcinus orca*), and Pacific white-sided dolphins (*Lagenorhynchus obliquidens*). J. Zoo Wildl. Med. 33(4):356-366.

Robeck, T. R., S. L. Monfort, P. P. Calle, J. L. Dunn, E. Jensen, J. R. Boehm, S. Young, and S. T. Clark. 2005. Reproduction, growth and development in captive beluga (*Delphinapterus leucas*). Zoo Biol. 24:29-49.

Ross, P. S. 1995. Seals, pollution and disease: Environmental contaminant-induced immuno-suppression. Ph.D. thesis, Univ. Utrecht, Utrecht, The Netherlands.

Ross, P. S., R. L. de Swart, R. Addison, H. V. Loveren, J. G. Vos, and A. D. M. E. Osterhaus. 1996. Contaminant-induced immunotoxicity in harbour seals – wildlife at risk. Toxicol. 112:157-169.

Rubin, D. B. 1988. Using the SIR algorithm to simulate posterior distributions, p. 395-402. *In* J. M. Bernardo, M. H. DeGroot, D. V. Lindley, and A. F. M. Smith (editors), Bayesian statistics 3. Oxford Univ.Press.

Rugh, D. J., K. E. W. Shelden, and B. A. Mahoney. 2000. Distribution of belugas, *Delphinapterus leucas*, in Cook Inlet, Alaska, during June/July, 1993-2000. Mar. Fish. Rev. 62(3): 6-21.

Rugh, D. J., K. E. W. Shelden, C. L. Sims, B. A. Mahoney, B. K. Smith, L. K. Litzky, and R. C. Hobbs. 2005a. Aerial surveys of belugas in Cook Inlet, Alaska, June 2001, 2002, 2003, and 2004. U.S. Dep. Commer., NOAA Tech Memo. NMFS-AFSC-149. 71 p.

Rugh, D. J., K. T. Goetz, B. A. Mahoney, B. K. Smith, and T. A. Ruszkowski. 2005b. Aerial surveys of belugas in Cook Inlet, Alaska, June 2005. Unpubl. doc.. Natl. Mar. Mammal Lab., NMFS, NOAA, Alaska Fish. Sci. Cent., 7600 Sand Point Way NE, Seattle, WA 98115. 17 p.

Rugh, D. J., K. L. Laidre, K. E. W. Shelden, and B. A. Mahoney. 2001. Distributional changes in a declining beluga population. Abstract in the Fourteenth Biennial Conf. on the Biology of Marine Mammals. Vancouver, B.C., Canada, Nov. 28-Dec. 3, 2001.

Samuels, W. M., M. J. Pybus, and A. A. Kocan (editors). 2001. Parasitic diseases of wild mammals, 2nd Edition. Iowa State University Press, Ames, IA.

Scholin, C. A., F. Gulland, G. J. Doucette, S. Benson, M. Busman, F. P. Chavez, J. Cordaro, R. DeLong, A. De Vogelaere, J. Harvey, M. Haulena, K. Lefebvre, T. Lipscomb, S. Loscutoff, L. J. Lowenstine, R. I. Martin, P. E. Miller, W. A. McLellan, P. D. R. Moeller, C. L. Powell, T. Rowles, P. Silvagni, M. Silver, T. Spraker, V. Trainer, and F. M. Van

Dolah. 2000. Mortality of sea lions along the central California coast linked to a toxic diatom bloom. Nature 403:80-84.

Scott, M. E. 1988. The impact of infection and disease on animal populations: implications for conservation biology. Conserv. Biol. 2(1):40-56.

Seaman, G. A., and J. J. Burns. 1981. Preliminary results of recent studies of belukhas in Alaskan waters. Rep. Int. Whal. Comm. 31:567-574.

Seaman, G. A., L. F. Lowry, and K. J. Frost. 1982. Foods of belukha whales (*Delphinapterus leucas*) in western Alaska. Cetology 44:1-19.

Sergeant, D. E. 1959. Age determination of odontocete whales from dentinal growth layers. Norsk Hvalfangst Tid. 6:273-288.

Sergeant, D. E. 1973. Biology of white whales (*Delphinapterus leucas*) in western Hudson Bay. J. Fish. Res. Bd Can. 30:1065-1090.

Shelden, K. E. W. 1994. Beluga whales (*Delphinapterus leucas*) in Cook Inlet – a review. Unpubl. doc. 10 p. [referenced as Appendix A in Withrow et al. (1994). Beluga whale distribution and abundance in Cook Inlet, 1993, p. 128-153. *In* MMAP Status of Stocks and Impacts of Incidental Take 1993, Office of Protected Resources, NMFS, Silver Spring, MD, June 1994.].

Shelden, K. E. W., D. J. Rugh, B. A. Mahoney, and M. E. Dahlheim. 2003. Killer whale predation on belugas in Cook Inlet, Alaska: Implications for a depleted population. Mar. Mammal Sci. 19(3): 529-544.

Shults, L. M., F. H. Fay, and J. D. Hall. 1982. Helminths from Stejneger's beaked whale *Mesoplodon stejnegeri* and Risso's dolphin *Grampus griseus* in Alaska. Proc. Helminthol. Soc. Wash. 49(1):146-147.

Silvagni, P. A., L. J. Lowenstine, T. Spraker, T. P. Lipscomb, and F. M .D. Gulland. 2005. Pathology of domoic acid toxicity in California sea lions (*Zalophus californianus*). Vet. Pathol. 42:184-191.

Smith, K. F., D. F. Sax, and K. D. Lafferty. 2006. Evidence for the role of infectious diseases in species extinction and endangerment. Conserv. Biol. 20(5):1349-1357.

Stephens, P. A., and W. J. Sutherland. 1999. Consequences of the Allee effect for behaviour, ecology and conservation. Trends Ecol. Evol. 14(10):401-405

Stewart, R. E. A., S. E. Campana, C. M. Jones, and B. E. Stewart. 2006. Bomb radiocarbon dating calibrates beluga (*Delphinapterus leucas*) age estimates. Can. J. Zool. 84:1840-1852.

Stoddard, R. A., F. M. D. Gulland, E. R. Atwill, J. Lawrence, S. Jang, and P. A. Conrad. 2005. *Salmonella* and *Campylobacter* spp. in northern elephant seals, California. Emerg. Infect. Dis. 11(12):1967-1969.

Sundberg, J. P., M. Van Ranst, and A. B. Jenson. 2001. Papillomavirus infection, p. 223-231. *In* E. S. Williams, and I. K. Barker (editors), Infectious diseases of wild mammals. Iowa State Univ. Press, Ames, IA.

Theriault, G., G. Gibbs, and C. Tremblay. 2002. Cancer in belugas from the St. Lawrence estuary. Environ. Health Perspect. 110:A562-A564.

Thompson, P. M. 2007. Developing water quality standards for coastal dolphins. Mar. Pollut. Bull. 54:123-127.

Thompson, P. M., H. Thompson, and A. J. Hall. 2002. Prevalence of morbillivirus antibodies in Scottish harbour seals. Vet. Rec. 151(20):609-610.

Treschev, V. V. 1968. The new campulid *Orthosplanchnus albamarinus* sp.n. (Trematoda, Campulidae)-parasite of the white whale. Zool. Zhurnal 47:937-940 (In Russian).

USCG (United States Coast Guard). 1988. Federal on-scene coordinator's report - major oil spill: M/V *Glacier Bay*. Cook Inlet, Alaska. 2 July to 3 August 1987.

Van Bressem, M. F., K. Van Waerebeek, and J. A. Raga. 1999. A review of virus infections in cetaceans and the potential impact of morbilliviruses, poxviruses and papillomaviruses on host population dynamics. Dis. Aquat. Org. 38:53-65.

Van Bressem, M. F., K. Van Waerebeek, P. D. Jepson, J. A. Raga, P. J. Duignan, O. Nielsen, A. P. DiBeneditto, S. Siciliano, R. Ramos, W. Kant, V. Peddemors, R. Kinoshita, P. S. Ross, A. Lopes-Ferandez, K. Evans, E. Crespo, and T. Barrett. 2001. An insight into the epidemiology of dolphin morbillivirus worldwide. Vet. Microbiol. 81:287-304.

Van Daele, L. J. 2007. Population dynamics and management of brown bears on Kodiak Island, Alaska. Ph.D. dissertation, Univ. Alaska, Fairbanks, Alaska. 81 p.

Vlasman, K. L., and G. D. Campbell. 2003. Field guide: Diseases and parasites of marine mammals of the Eastern Canadian Arctic. Canadian Cooperative Wildlife Health Centre and the Inuit Tapiriit Kanatami. University of Guelph, Guelph, Ontario. 109 p.

Vos, D. J. 2003. Cook Inlet beluga age and growth. M.S. thesis, Alaska Pacific University, Anchorage, Alaska. 69 p.

Vos, D. J., and K. E. W. Shelden. 2005. Unusual mortality in the depleted Cook Inlet beluga population. Northwest. Nat. 86(2):59-65.

Wade, P. R., and R. P. Angliss. 1997. Guidelines for assessing marine mammal stocks: Report of the GAMMS workshop April 3-5, 1996, Seattle, Washington. U.S. Dep. Commer., NOAA Tech. Memo. NMFS-OPR-12.

Wade, P. R. 2002. A Bayesian stock assessment of the eastern Pacific gray whale using abundance and harvest data from 1967 to 1996. J. Cetacean Res. Manage. 4:85-98.

Wazura, K. W., J. T. Strong, C. L. Glenn, and A. O Bush. 1986. Helminths of the beluga whale (*Delphinapterus leucas*) from the Mackenzie River Delta, Northwest Territories. J. Wildl. Dis. 22(3):440-442.

Williams, E. S., and I. K. Barker (editors). 2001. Infectious diseases of wild mammals, 3rd Ed. University of Iowa Press, Ames, IA.

Wong, S. 2002. Ocean sentinels: Marine mammals and antimicrobial resistance. Proceedings of the 42nd Interscience Conference on Antimicrobial Agents and Chemotherapy, September 27-30, 2002, San Diego, CA.

Woshner, V. M. 2000. Concentrations and interactions of selected elements in tissues of four marine mammal species harvested by Inuit hunters in arctic Alaska, with an intensive histologic assessment, emphasizing the beluga whale. Ph.D. dissertation, University of Illinois, Urbana, Illinois. 302 p.

Wynne, K. 1997. Guide to marine mammals of Alaska. Alaska Sea Grant Program, University of Alaska, Fairbanks. 75 p.

Young, S. J. F., D. G. Huff, and C. Stephen. 1999. A risk-management approach to a mycotic disease potential in captive beluga whales (*Delphinapterus leucas*). Zoo Biol. 18:5-16.