Exxon Valdez Oil Spill
Restoration Project Final Report

Effect of Disease on Recovery of Pacific Herring in Prince William Sound, Alaska

Restoration Project 030462
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Study History: This project continues the field component of project 98162 (the final report for 98162 is approved). Results for this project were reported in four annual reports: 1) 99462, "Effect of Disease on Recovery of Pacific Herring in Prince William Sound, Alaska, Spring 1999"; and 2) 00462, "Effect of Disease on Recovery of Pacific Herring in Prince William Sound, Alaska, Fall 1999 and Spring 2000"; 3) 01462, "Effect of Disease on Recovery of Pacific Herring in Prince William Sound, Alaska, Fall 2000 and Spring 2001"; and 4) 02462, "Effect of Disease on Pacific Herring Population Recovery in Prince William Sound." Detailed histopathologic examination, blood analysis, and a modeling component were supported by a grant from the National Science Foundation (project #9871982, "Role of parasites and disease in health and population abundance of adult Pacific herring"). Results specific to the National Science Foundation part of the study are not reported here. Publications that preceded this final report:

1. Marty, G. D., T. J. Quinn, II, G. Carpenter, T. R. Meyers, and N. H. Willits. 2003. Role of disease in abundance of a Pacific herring (*Clupea pallasi*) population. Canadian Journal of Fisheries and Aquatic Sciences 60:1258-1265.

2. Quinn, T. J., II, G. D. Marty, J. Wilcock, and M. Willette. 2001. Disease and population assessment of Pacific herring in Prince William Sound, Alaska. Pages 363–379 *in* F. Funk, J. Blackburn, D. Hay, A. J. Paul, R. Stephensen, R. Toreson, and D. Witherell, editors Herring: Expectations for a new millennium. University of Alaska Sea Grant, AK-SG-01-04, Fairbanks.

Abstract: Due to disease-related declines in population biomass, Pacific herring (*Clupea pallasi*) fisheries in Prince William Sound have been closed since 1999. Detailed disease study from 1994 through 2002 included samples collected in the spring (n = 233–300) and fall (n = 40–160) and analyzed using consistent methods. During the first 7 years of study, viral hemorrhagic septicemia virus and ulcers covered by filamentous bacteria were the most significant causes of diseases; *Ichthyophonus hoferi* varied little (16–24%) and prevalence was highly correlated with fish age. In spring 2001, however, prevalence of *I. hoferi* (38%) was 50% greater than it had been in any of the previous 7 years. Prevalence of *I. hoferi* in 2002 returned to baseline levels (15%), and the drop in *I. hoferi* prevalence by 2002 was associated with increased mortality of adult fish during the previous year. The best model for estimating population biomass includes variable mortality derived from a virus-ulcer index for younger fish (ages 3 and 4) and the *I. hoferi* prevalence in older fish (ages 5+). Virus-ulcer outbreaks have cycled through the population in roughly 4-year cycles since 1989, but the severity of the outbreaks has steadily decreased since 1993.

Key Words: *Clupea pallasi*, disease, *Exxon Valdez*, *Ichthyophonus hoferi*, Pacific herring, Prince William Sound, viral hemorrhagic septicemia virus (VHSV).

Project Data: Data include date, location, and time of capture; sex, age, standard length, body weight, gonad weight, and liver weight; gross necropsy findings; and results from virus analysis (viral hemorrhagic septicemia virus, VHSV), histopathology, and blood analysis. All disease data are stored in an Excel spreadsheet (191 columns and 3933 rows). The spreadsheet is stored and maintained by Gary D. Marty, VM:APC, Univ. of CA, 1 Shields Ave., Davis, CA 95616; 530-754-8062; e-mail: gdmarty@ucdavis.edu. The model is contained in an Excel spreadsheet stored and maintained by author Quinn. Data will be freely available after they are published.

Citation: Marty, G. D., T. J. Quinn II, S. A. Miller, T. R. Meyers, and S. D. Moffitt. 2004. Effect of Disease on Recovery of Pacific Herring in Prince William Sound, Alaska, *Exxon Valdez* Oil Spill Restoration Project Final Report (Restoration Project 030462), University of California, Davis, California.

TABLE OF CONTENTS

Executive Summary

Introduction

Due to low population biomass, Pacific herring (*Clupea pallasi*) fisheries in Prince William Sound have been close since 1999. Studies of the Prince William Sound Pacific herring population since 1994 provided evidence that disease is a significant variable in population fluctuations. Before 2001, ulcers and viral hemorrhagic septicemia virus were associated with acute mortality that was significant on a population scale (Marty et al. 2003). The other major disease—caused by the primitive protist *Ichthyophonus hoferi*—is chronic and decreases the life span of affected fish, but it did not play a significant role in unexpected population fluctuations before 2001 (Marty et al. 2003). In 2001, however, the prevalence of *Ichthyophonus hoferi* (38%) was 50% greater than it had been in any previous year of study. Prevalence of *I. hoferi* in 2002 returned to baseline levels (15%). We provide evidence that the drop in *I. hoferi* prevalence by 2002 was associated with increased mortality of older fish during the previous year, presumably as a result of infection with *I. hoferi*.

Objectives

Our study had three objectives: 1) determine the prevalence of major diseases in Pacific herring; 2) determine the interaction of gender, age, and season on disease prevalence; 3) determine if disease prevalence correlates with population trends.

Methods

Adult Pacific herring from Prince William Sound were sampled at random and subjected to complete necropsy in April 1999, 2000, 2001, and 2002 (n = 300), and in October/November 1999, 2000, and 2001 (n = 40–100). All fish collected before 2001 were sampled from bays near the north end of Montague Island. During the spring of 2001 and 2002, fish numbers were more evenly distributed between the Northeast and Montague areas of PWS; therefore, sampling was split between these areas. Analysis of all fish included determination of age, weight and length, gross examination, and culture of head kidney and spleen for virus isolation. In fish with severe external lesions, kidney was cultured for bacteria (all were negative). A project supported by the National Science Foundation included analysis of blood, complete histopathology, and mathematical modeling of the role of disease on population biomass. Results from study supported solely by the National Science Foundation are not reported here.

An age-structured assessment model was modified using disease information to estimate the biomass of Prince William Sound Pacific herring. The original model, developed more than a decade ago, set natural mortality M at a constant 0.25. In this report, we examine alternative models that allow natural mortality to vary based on prevalence of important diseases. We stratify mortality by two age groups, ages 3–4 and ages 5+. For each age group and for pooled ages, we test model fit using the spring virus-ulcer index (Marty et al. 2003) and the spring prevalence of *I. hoferi*.

Results

Disease prevalence in the Pacific herring population was low in both 1999 and 2000. In spring 2001, prevalence of *I. hoferi* increased to 38% in April 2001: more than 50% greater than in any year since population-level disease study began in 1994. By 2002, prevalence of *I. hoferi* (15%) was back to historical levels. Prevalence of viral hemorrhagic septicemia virus was less than 2% in all spring samples from 1999 – 2001, but increased to 14% in 2002. Prevalence of ulcers was less than 1% in all spring samples; therefore, the impact of the virus outbreak in 2002 was minimal among the adult population. None of the fall samples had evidence of viral hemorrhagic septicemia, and this finding is consistent with fall samples in all other years studied (Marty et al. 2003). Mathematical modeling provides evidence that the *I. hoferi* outbreak was associated with increased mortality in 2001. The best model uses constant baseline mortality M_0=0.25 for all ages; the virus-ulcer index is used for the younger age group (ages 3–4) and *I. hoferi* prevalence is used for the older age group (ages 5+).

Discussion

Viral hemorrhagic septicemia virus, associated ulcers, and *I. hoferi* are the most important identifiable variables limiting recovery of the Pacific herring population of Prince William Sound, Alaska. Outbreaks of viral hemorrhagic septicemia and ulcers have been cycling through the population every 4–5 years since 1989. The severity of each outbreak has decreased in each cycle since 1993, and the last outbreak in 2002 was relatively mild. The decrease in *Ichthyophonus hoferi* prevalence between 2001 and 2002 was a result of increased mortality of adult fish in the population and strong recruitment of the lightly infected 1999 year-class into the population. Evidence from continued annual disease surveys conducted by ADFG in Prince William Sound indicates that the prevalence of *I. hoferi* has increased through 2004. If this pattern continues, we can expect another *I. hoferi* outbreak in 2005 or 2006.

Conclusion

Recovery of the Pacific herring population was significantly impaired by an outbreak of *I. hoferi* in 2001. Although strong recruitment of the 1999 year-class provided some hope for population recovery, increased prevalence of *I. hoferi* in this year class through spring 2004 will impair recovery. Also, recruitment of the 2000 and 2001 year classes into the population has been poor. The virus outbreak in 2002 seemed to have little effect on adults in the population, but it might have increased mortality of juveniles (i.e., the 2000 year class as 2-year-olds, and the 2001 year class as 1-year-olds). The Pacific herring population of Prince William Sound was significantly impaired by outbreaks of virus in 1993/1994 and 1998. Decrease in the severity of these outbreaks over the past decade seems to have been replaced by outbreaks of *I. hoferi*, first in 2001, and hypothesized for 2005 or 2006. We predict that recovery of the Pacific herring population of Prince William Sound will not occur until both viral hemorrhagic septicemia and *Ichthyophonus hoferi* remain at background levels for several years.

Introduction

When the *Exxon Valdez* oil spill occurred in March 1989, the biomass of spawning Pacific herring (*Clupea pallasi*) in **Prince William Sound** (**PWS**), Alaska, was the highest in 20 years of reliable estimates (more than $100 \bullet 10^6$ kg; Figure 1), and the population remained near record levels through 1992. Pacific herring in PWS first spawn when they are 3 or 4 years old. They rarely live more than 12 years, and before the spill abundant year classes recruited into the fishery about once every 4 years. In 1993, recruitment from the 1988 year-class was expected to be excellent, and fisheries biologists predicted a near-record spawning biomass of $110 \bullet 10^6$ kg before the spawning season (Figure 1). However, when the 1993 spawning season commenced, only 17% of the expected biomass appeared, fish were lethargic, and many had external hemorrhages. Therefore, PWS Pacific herring fisheries were severely curtailed in 1993, and were never opened in 1994, 1995, or spring 1996. In PWS before 1993, Pacific herring supported 5 commercial fisheries, with an average annual ex-vessel value of $8.3 million. Roe fisheries, the most valuable, are harvested in April just before spawning. **Viral hemorrhagic septicemia virus (VHSV)** was first isolated from groups of Pacific herring in 1993, but the significance of these isolates could not be determined (Meyers et al. 1994).

Disease study supported by the Trustee Council from 1994–2000 identified a virus (VHSV) and a protistan parasite (*Ichthyophonus hoferi*) that were important causes of disease (Marty et al. 1998). Virus prevalence was highly variable and associated with ulcers, acute disease, and unexpected population decline (Quinn et al. 2001). By comparison, *I. hoferi* prevalence was fairly constant and associated with chronic disease that probably decreased the life span of affected fish, but *I. hoferi* was not correlated with unexpected population decline (Marty et al. 2003).

Pacific herring population biomass increased enough in PWS so that roe fisheries were re-opened in 1997 and 1998. However, in 1998 high virus prevalence (14%) coupled with high ulcer prevalence (3.2%) provided evidence that the population was at risk of another disease-related decline. Therefore, this project was initiated to determine the effect of disease on recovery of Pacific Herring in PWS. Initial modeling efforts revealed that population biomass was best estimated through use of an age-structured assessment model modified with a disease index that incorporated the spring prevalence of VHSV and ulcers (Marty et al. 2003).

The estimated spawning biomass of Pacific herring in PWS decreased from about 26,000 metric tons in 1998 to only 13,000 metric tons in 2001. Fisheries in PWS were severely curtailed in 1999, and all Pacific herring fisheries have been closed since 1999. This report describes the major disease-related findings in Pacific herring sampled from PWS from 1994–2002, with special emphasis on effects of the unprecedented increase in the *I. hoferi* prevalence in 2001.

Objectives

Our study had three objectives:

1) determine the prevalence of major diseases in Pacific herring;
2) determine the interaction of gender, age, and season on disease prevalence; and
3) determine if disease prevalence correlates with population trends.

Methods

Necropsy and histopathology

Pacific herring were captured using a commercial purse seine in sets of 20 fish each. Fall samples were all collected from near the north end of Montague Island. During the spring of 2001 and 2002, fish numbers were more evenly distributed between the Northeast and Montague areas of PWS (unpublished ADFG population estimates); therefore, sampling was split between the Northeast region (2001, $n = 220$; 2002, $n = 200$) and the north end of Montague Island (2001, $n = 80$; 2002, $n = 100$). Fish were subjected to complete necropsy on board a contracted vessel at the site of capture. After capture, fish were held in plastic fish totes filled with about 300 L of seawater for no more than 5 hours before necropsy. Herring were anesthetized in tricaine methane sulfonate (Finquel®), assigned a unique necropsy number, weighed and measured (standard length), and a scale was removed for age determination. Several diagnostic procedures were done on each fish:

1) External lesions were scored as none (0), mild (1), moderate (2), or severe (3). For spring samples, gonadal fullness was estimated and scored as 3 (75–100% full), 2 (50–74% full), 1 (25–49% full), or 0 (0–25% full).

2) About 1.5 mL of blood was drawn from the caudal vein into 3-mL syringes that contained 0.1 mL of lithium heparin (1,000 IU•mL^{-1}); a capillary tube was filled and centrifuged (5500•*g* for 5 min) for determination of **packed cell volume (PCV)**, a blood smear was made and air-dried, and remaining blood was centrifuged (13,600•*g* for 5 min) and plasma was immediately decanted and frozen. Analysis of these samples was not part of this project, and results will not be reported here.

3) For virus isolation, head kidney and spleen from each fish were pooled in a plastic bag (one fish per bag) and shipped on ice to the ADFG Fish Pathology Laboratory in Juneau, Alaska; skin lesions, if present, were sampled and bagged separately for individual virus assay. Propagation of 1 cell line (EPC), media formulation, and tissue preparation for cell line inoculation were as previously described (Meyers et al. 1994).

4) For histopathology, samples of gill, liver, gonad, spleen, trunk kidney, gastrointestinal tract, heart, skin, skeletal muscle, and brain were fixed in 10% neutral buffered formalin. Analysis of these samples was not part of this project, and results will not be reported here (except for the overall *I. hoferi* prevalence scores, which are reported here). For each organ, severity of *I. hoferi* was scored as none (0), mild (1), moderate (2), or severe (3). Also, a sum-*Ichthyophonus* score was calculated for each fish by adding the *I. hoferi* score for each of 10 organs. Actual sum-*Ichthyophonus* scores ranged from 0 to 25. For modeling the effects of *I. hoferi* on population biomass, the prevalence of sum-*Ichthyophonus* score >0 was used.

5) Bacterial isolation was attempted from herring with severe external lesions; kidney tissues were aseptically inoculated onto trypticase soy agar (TSA) and marine agar and plates were incubated at 23° C for at least 5 days (all were negative);

6) Liver and gonads were weighed;

7) Herring worms (Anisakidae) were counted (herring worms are coiled parasites that commonly line the outside of the intestine in marine fish.); and

8) Opercular copepods were scored as other external lesions (these parasites are common on the inside of the operculum: the plate that covers the gill).

Population modeling of disease

The age-structured assessment model (Marty et al. 2003) was updated with more recent information. The basic model provides an estimation framework to integrate the various sources of information about Pacific herring in Prince William Sound from 1980 – 2003, including age compositions from the purse-seine fishery and spawning surveys, egg production estimates, and mile-days of milt from aerial surveys (Quinn et al. 2001). These observations are compared to comparable model quantities in a least squares setting to obtain parameter estimates of recruitment, abundance, and biomass.

The age-structured assessment model contains information about the Pacific herring fisheries in Prince William Sound, Alaska, which include purse-seine, gillnet, and pound fisheries in the spring (mainly for roe), and a food and bait fishery in the summer and fall. Recruitment occurs at age 3 and there are parameters for recruit abundance, {N3,t}, for all years (and for all abundances in the first year, 1980). From these parameters and the survival model (1), abundance at each subsequent age a+1 and year t+1 is estimated from the equation

$$N_{a+1,t+1} = \left[\left(N_{a,t} - C_{\text{seine}} - C_{\text{gill}} - C_{\text{pound}}\right) \times S_t^{1/2} - C_{\text{food}}\right] \times S_{t+1}^{1/2}, \quad (1)$$

in which N = abundance, C = annual catch-at-age, and $S^{1/2}$ = half-year survival, calculated as the square root of equation (3) below. In equation (1), we assume that natural mortality at time t affects the population in the last half-year of year t (before recruitment, spawning, and spring fisheries) and the first half-year of year $t+1$. Another assumption is that total catch for the seine fishery and catch-at-age for the other fisheries are assumed measured without error in the model. The estimated number of spawners is the mature abundance after the spring fisheries, or

$$\text{Spawners}_t = \sum_{\text{a}} \text{mat}_a \left(N_{a,t} - C_{\text{seine}} - C_{\text{gill}} - C_{\text{pound}}\right) \quad (2)$$

in which mat is the proportion mature. Spawning biomass is similar to equation (2) but also multiplies by weight-at-age.

The maturity variable mat_a in (2) is set to 1 for ages 5+ and estimated for ages 3 and 4 separately. In preliminary analyses of the extended dataset, we noticed a positive trend in the spawning age

composition residuals for ages 3 and 4 after 1997. This trend could only be removed by estimating separate maturity parameters for the time period 1980 – 1997 and 1998 – 2003. In addition, the parameter mat_4 for 1980 – 1997 was constrained to be above 0.60 to produce plausible estimates of population parameters, in accord with standard practice by ADFG.

The other parameters in the model are parameters for the purse seine fishery (which is assumed to be a logistic relationship with age), natural mortality for the plus group (aged 9+) relative to other ages, a calibration coefficient that converts the milt index into total egg production, and the disease parameters described below.

We assume that disease increases the natural mortality M for all adult ages (ages 3 and older), or equivalently, that disease lowers the corresponding natural survival S. For simplicity, S_t in year t is assumed to decrease linearly as a function of disease prevalence variables $\{x_{it}\}$, where natural survival S_0 from sources other than disease is constant. Expressed mathematically,

$$S = S_0\left(1 - \sum_i \beta_i x_{it}\right) \tag{3}$$

where x_{it} is the sampled prevalence for pathogen i in year t and β_i represents the proportion of the infected population that dies from the particular pathogen. We stratify mortality by two age groups, ages 3 – 4 and ages 5+. For each age group, the first prevalence variable is a VHSV-ulcer index (calculated by multiplying the relative frequency of VHSV+ fish with the relative frequency of fish with ulcers, with a lower bound of 0.5%; see Marty et al. 2003). The second is *I. hoferi* prevalence. Also, a parameter for disease prevalence in 1992–1993 is estimated for each disease variable (because there is only very limited information from this period; Quinn et al. 2001). Values for the VHSV indices and *I. hoferi* prevalences are given in Table 1.

Five combinations of disease variables are examined; the first four are stratified by age group (i.e., ages 3 and 4 in one group; ages 5+ in the other group): 1) both disease variables are included; 2) just the VHSV-ulcer index is used; 3) just the *I. hoferi* prevalence is used; 4) VHSV-ulcer index is used for ages 3 and 4, and *I. hoferi* is used for ages 5+; and 5), the VHSV-disease index pooled over all ages is used. Because the VHSV-disease index could not be calculated in 2003 (VHSV prevalence was not determined in 2003), natural mortality in 2003 is assumed equal to that in 2002.

Background natural mortality M_0 is treated in three different ways: (A) Different M_0 parameters are used for the two age groups and estimated from the data; (B) M_0 is assumed constant over age and estimated from the data; or (C), M_0 is assumed constant over age and set equal to 0.25, in accord with Quinn et al. (2001) and Marty et al. (2003). In these studies, it was not possible to estimate M_0 confidently, so it was set to the value used in earlier ADFG assessments. However, ADFG now attempts to estimate M_0, so we also attempt to do so.

In all, 15 models are examined. Combinations of the 5 disease treatments with the 3 background mortality choices are labeled A1, A2, …, C5.

The objective function includes residual least squares (or negative likelihood) components RSS_i for the purse seine age composition (in years when the fishery was open: 1980 – 1988, 1990 – 1992, 1997 – 1998, $n_1 = 88$ observations), spawning age composition (since 1982, $n_2 = 151$ observations), egg survey estimates on a logarithmic scale (from 10 years between 1984 and 1997, $n_3 = 10$ observations), and the egg milt index on a logarithmic scale (all years 1980 – 2003, $n_4 = 24$ observations), for a total of $n = 273$ observations. Seine and spawning age composition received a weighting factor of $\lambda_1 = \lambda_2 = 1$, and the egg survey and milt index were weighted by $\lambda_3 = \lambda_4 = 0.5$ to avoid unwelcome patterns in the residuals, on accord with current ADFG practice. The model is contained in a spreadsheet and available from author Quinn.

Model selection followed established procedures (Burnham and Anderson 1998), including the use of an information criterion, AICc (Akaike Information Criterion, corrected) as a model comparison statistic. In order to calculate AICc, the likelihood must be calculated first, from the equation

$$\ln L = \sum_i -\frac{1}{2}\left[n_i \ln(2\pi\sigma_i^2) + \frac{RSS_i}{\sigma_i^2} \right] \tag{4}$$

in which σ_i^2 is the unexplained variance for the *i*th dataset. The weighting terms λ_i for each dataset *i* are the variances relative to the first dataset (seine age composition), or $\lambda_i = \sigma_1^2 / \sigma_i^2$. The maximum log likelihood can be shown to be

$$\begin{aligned} \max \ln L &= \sum_i -\frac{n_i}{2}\left[\ln(2\pi\hat{\sigma}_1^2 / \lambda_i) + 1\right], \text{ in which} \\ \hat{\sigma}_1^2 &= \sum \lambda_i RSS_i / \sum n_i, \text{ and } \hat{\sigma}_i^2 = \hat{\sigma}_1^2 / \lambda_i. \end{aligned} \tag{5}$$

The maximum likelihood corresponds to the minimum residual weighted sum of squares. The Akaike information criterion AIC and its corrected version for small sample sizes AICc are then obtained from

$$\begin{aligned} AIC &= -2\ln L + 2p \\ AICc &= AIC + 2p(p+1)/(n-p-1) \end{aligned}, \tag{6}$$

where *p* is the number of parameters. "Unless the sample size is large with respect to the number of estimated parameters, use of AICc is recommended [over AIC]" (Burnham and Anderson 1998, p. 51).

The difference Δ between a given model and the model with the lowest AICc value is the primary statistic for choosing appropriate models. For model selection we used the following guidelines:

"For any model with $\Delta \le 2$ there is no credible evidence that the model should be ruled out … For a model with $2 \le \Delta \le 4$ there is weak evidence that the model is not the K-L [Kullback-Leibler] best model. If a model has $4 \le \Delta \le 7$ there is definite evidence that the model is not the

K-L best model, and if $7 \leq \Delta \leq 10$, there is strong evidence that the model is not the K-L best model. Finally, if $\Delta > 10$, there is very strong evidence that the model is not the K-L best model" (Burnham and Anderson 1998, p.128).

Therefore, we immediately rejected models with $\Delta > 4$ and included candidate models with $\Delta \leq 4$. We further eliminated models if they were not biologically realistic.

Historical disease index

Complete data to generate the VHSV-ulcer disease index are available for only 9 years, 1994 – 2002. However, limited data are available from 1989, 1992, 1993, 2003, and 2004 to assign reasonable values of the disease index for these years; for these years, assigned values are used for graphical analysis of trends, but not for mathematical modeling. For 1989, 10% of 20 Pacific herring from oiled sites had hepatocellular necrosis, but none of the 20 fish from reference sites had hepatocellular necrosis (Marty et al. 1999), for an overall PWS estimate of 5% necrosis prevalence; this is equivalent to the prevalence of ulcers in samples from 1997, so the disease index for 1989 is assigned the same value as for 1997. In 1992, only 1% of 82 Pacific herring livers had hepatocellular necrosis (Kocan et al. 1996), and 3-year-old recruitment was low; this is equivalent to 1999, so the disease index for 1992 is assigned the same value as for 1999. For 1993, VHSV prevalence was somewhere between 15% and 75% based on data from 4 pooled samples, and the combination of moderate and severe focal skin reddening prevalence varied from 15% to 43% (Meyers et al. 1994); assigning a conservative value for VHSV prevalence (30%), an ulcer prevalence (5%) yields a disease index for 1993 of 150. Ulcer prevalence in 2003 (0.3%) and 2004 (0.4%) were low, recruitment was low both years, and the disease index was assigned the same value as for 1999.

Results

Major diseases

A major epizootic caused by *I. hoferi*, affected the health of Pacific herring in PWS beginning in fall 2000. Prevalence of *I. hoferi* in spring 2001 (38%) was more than 50% greater than it had been in any previous year of study (Figure 2), or in any other Alaskan population studied in the past decade (e.g., populations from Sitka, Craig, and Auke Bay; Carls et al. 1998, Davis et al. 1999; G.D. Marty, unpublished observations). By spring 2002, prevalence of *I. hoferi* had returned to historical levels (Figure 2).

Sample prevalence of viral hemorrhagic septicemia virus and skin ulcers in spring 2001 was slightly higher than in 2000, but still at historically low levels (Figure 3). In 2002, the prevalence of VHSV (14%) was among the highest of the 9 years studied, but relatively low ulcer prevalence (0.7%) provided evidence that adult fish were not significantly impaired by the VHSV outbreak.

Over time, VHSV-ulcer associated disease in PWS Pacific herring is oscillating in a roughly 4-year cycle, the amplitude of which is decreasing with each cycle since 1993 (Figure 4). VHSV-ulcer outbreaks tend to be more common when the prevalence of young fish in the population is

high (Figure 4). PWS Pacific herring had a major disease outbreak in 1993, moderate disease in 1997–1998, and mild disease in 2002. As the amplitude of VHSV-ulcer outbreaks decreases over time, we have preliminary evidence that *I. hoferi* outbreaks may also be cyclic. According to studies conducted by ADFG in PWS Pacific herring through spring 2003, the *I. hoferi* prevalence has doubled since 2002, but it is not yet as high as in 2001. If *I. hoferi* prevalence peaks in 2005, it would complete a 4-year cycle.

Other parasites

Numbers of herring worm parasites (Anisakidae) per fish peaked in 1999 and then hit an all-time low in 2002 (Figure 5). This pattern was most obvious among fish first recruiting to the adult population (i.e., 2-year-olds in the fall, and the same year class as 3-year-olds the next spring). From spring 1994 through spring 1998, these young fish averaged 8 to 17 herring worms per fish. By fall 1998, 2-year-olds (1996 year class) averaged more than 40 herring worms per fish. Numbers of herring worms also increased in the 1994 and 1995 year-classes between spring 1998 and spring 1999. After spring 1999, numbers of herring worms in young fish steadily decreased, but numbers of herring worms remained relatively constant in the 1994 and 1995 year-classes (Figure 5). Anisakis remain as larvae in fish; they develop into adults and lay eggs only in the intestines of piscivorous birds and mammals. Little is known about changes in numbers of Anisakis over time, but the increase in 1999 might be correlated with increased predation on the sick population in 1998. As evidence against this hypothesis as a general trend, numbers of herring worms did not increase dramatically after the disease outbreak of 1993–1994. The persistence of herring worms in the 1994 and 1995 year-classes for several years after 1999 provides evidence that once fish are infected, the worms remain with the fish for many years.

Prevalence of copepod parasites on the medial operculum was usually greater in the fall than in the following spring, and the proportion of fish affected in spring samples increased from 1999 to 2002 (Figure 6). Opercular copepods do not seem to be related to mortality patterns at the population scale

Population modeling of disease

Summary statistics of the total weighted residual sums of squares and numbers of parameters show that models with more parameters have lower RSS values (Table 2) as expected, because more parameters are used. The numbers of parameters ranged from 36 to 44. Models with more restrictions on background mortality M_0 (C versus B versus A) have higher RSS values, because fewer parameters are used. Unweighted RSS components show that there are small differences in the fits to the various data components (seine, spawning, egg survey, and milt index), pending statistical tests of significance. Estimates of unexplained variance for the 4 datasets also do not differ much among models.

The likelihood values show the same relationships among models as RSS, except that higher values indicate better fitting models (Table 2). The AICc values used for model selection indicate substantial differences among models (Table 2). The most parsimonious model is C3, in which just *I. hoferi* data are used for the two age groups and M_0 is set to 0.25. Other candidate models (with Δ values less than or equal to 4) are A3, A4, and A5 (separate M_0's estimated by

age group, and either just *I. hoferi* data, VHSV-ulcer data for younger and *I. hoferi* data for older ages, or pooled VHSV-ulcer data), B3 and B5 (common M_0 estimated for all ages, and either just *I. hoferi* data or pooled VHSV-ulcer data), and C4 (M_0=0.25, VHSV-ulcer data for younger and *I. hoferi* data for older ages). Models A1, B1, and C1 with both disease datasets for both age groups and Models A2, B2, and C2 with just VHSV-ulcer data by age group were significantly poorer.

Estimates of disease parameters, baseline mortalities, and resultant natural mortalities are shown in Table 3. Models A1, B1, and C1, which use both disease variables, have positive β estimates for both age groups, suggesting that both disease sources may contribute to natural mortality at all ages. This finding differs from that in Marty et al. (2003), in which the *I. hoferi* disease effect was estimated to be 0. In other models, parameter estimates were also positive, with no convergence to 0 values, again suggesting that disease is important in determining natural mortality. Parameter estimates are comparatively larger in models with only one disease source, suggesting some confounding between the two diseases. Estimated prevalence in 1992–1993 was large compared to the observed values in later years (Table 1), because the disease event was significant, as was earlier shown in Quinn et al. (2001).

Baseline natural mortality was substantially larger for ages 3–4 than for ages 5+ (models A1 to A5). Comparison of AICc values (Table 2) among A, B, and C models for each disease treatment shows differences of less than 4, except for disease treatment 2. This result suggests that there is only weak evidence that the age differences are real.

Natural mortality for ages 3–4 is generally higher and more variable than for ages 5+ (Table 3, and plotted in Figure 7). Because the difference in natural mortality is directly a function of the disease information, this result means that disease appears to have more of an effect at younger ages. Disease events clearly stand out in years 1992–93, 1998, and 2001 for both age groups and in 1994 for ages 3–4 (Figure 7). Years 1996 and 1999 are important for some treatment combinations. Overall, the effect of disease depends on interactions among disease variables, treatment of background mortality, and age group.

The proportion of age 3 and 4 fish that are mature was clearly different before and after 1998 (Table 4). This signifies that there has been greater proportion of younger ages that engage in spawning in more recent times. Recruitment estimates vary among models, with higher recruitment estimates associated with models with higher natural mortality. Consequently, differences in 2004 spawning biomass vary only a small amount: from 17,000 to 22,000 mt.

Using the principle of parsimony, the AICc values indicate that models A3, A4, A5, B3, B5, C3, and C4 could be the best. On biological grounds, we tend to disfavor disease treatment 3, because its use of only *I. hoferi* information seems contrary to the strong evidence in favor of VHSV being a major contributor to the events of 1992–93 and 1998 (Quinn et al. 2001, Marty et al. 2003). On the basis that there is not clear evidence that background natural mortality M_0 varies by age or is much different from 0.25, we tend to reject treatments A and B. That leaves model C4 in which M_0=0.25 for all ages and VHSV information is used for the younger age group and *I. hoferi* is used for the older age group. Because that model is consistent with observations of the disease effect with age, we choose it as our best model.

The fit of the model C4 to the datasets has no unwelcome patterns in the seine or spawning age composition residuals (Figure 8). The fit to the egg survey information is poor in the late 1980's, but this is true of all stock assessment models that have been developed, including all 15 models in this study. The fit to the milt information is fairly good, except for some lack of fit in the early 1990s.

One remaining question is whether disease affected recruitment at age 3 since the early 1990s. We correlated recruitment estimates with the 5 disease time series in Table 1 to answer this question. Correlation analysis was first performed with no lag using data from 1994 to 2002. Then correlation was performed with a lag of 1 year in the disease information (recruitments from 1995 to 2003 versus disease from 1994 to 2002). Correlations between *I. hoferi* and recruitment with no lag are negative and significant, while correlations with lag 1 are positive and not significant (Table 5). The explanation for the significant negative relationship is that the large increase in *I. hoferi* prevalence in 2001 coincided with the smallest estimated recruitment on record. Also, prevalence of *I. hoferi* tends to be higher in older fish, and the population age is relatively older when recruitment is poor. Because this estimate is highly uncertain until more years of data have been collected, the biological significance of this result is still of question. In contrast, correlations between the VHSV-ulcer index (pooled, ages 3–4, ages 5+) and recruitment with no lag are positive, while correlations with lag 1 are positive and negative, and none is significant.

Discussion

Spring 2001 marked the beginning of the third major disease epizootic in the PWS Pacific herring population in the last decade. In the early and late 1990s, high prevalence of VHSV and ulcers was associated with significant decline in population biomass (Marty et al. 2003). By comparison, the epizootic of 2001 was associated with unusually high prevalence of *I. hoferi*. The cause of the increase in prevalence of *I. hoferi* is unknown, but it might be associated with high mortality in previous years. As evidence, prevalence of *I. hoferi* in 1993—at the height of the most severe epizootic in the past decade—was only about 5% (Marty et al. 1998), but *I. hoferi* prevalence increased to 23% in 1994 (Quinn et al. 2001). We also have good evidence of VHSV-related population decline from 1998–1999 that predated the 2001 increase in *I. hoferi* by two to three years.

Based on modeling results, however, there appears to be no compelling biological relationship between ln recruitment and disease over from 1994 to 2003. Previously, Marty et al. (2003) did find a significant relationship between VHSV over all ages lagged one year and recruitment. If it turns out that recruitment in 2001 is really as high as 30 million fish (instead of the current estimate near 0), which is on the low end of the historical range, then the VHSV correlations with ln recruitment becomes negative and significant. Otherwise, the previous relationship detected by Marty et al. (2003) was either spurious, or else there has been a change to no effect of VHSV on recruitment after 2000. Our original hypothesis was that disease was a sporadic event associated with exceeding carrying capacity, but the 1998, 2001, and 2002 disease events occurred when the population was relatively low.

There is also ambiguity in whether or not background mortality from other sources (such as predation) differs among age groups. While our data suggest that smaller, younger herring are more susceptible to predation, there is not much information in Prince William Sound to validate this hypothesis. From the results in this study, it cannot be ruled out that background natural mortality has become higher more recently and is greater for younger ages. The fact that Pacific herring abundance decreased dramatically in the mid-1990s raises the possibility that density-dependent increases in natural mortality have occurred with the potential decline in the ratio of herring as prey to the predator populations.

Also interesting is the apparent increase in the maturity on the spawning grounds inferred from the model fits to the spawning age composition data. It is possible that the reduction in herring abundance has increased either the rate of maturation or the migration of mature fish to the spawning areas. Nevertheless, it would be valuable in the future to investigate the maturity of herring in the population as a whole through field studies outside the spawning season.

How and at what age Pacific herring are naturally infected with *I. hoferi* is not known. Prevalence within a year class consistently increases with age over time (Hershberger et al. 2002, Marty et al. 2003), and minimal change in age-class distribution over time is consistent with infected fish living for several years with *I. hoferi*. This pattern of increased prevalence with age could be a result of small numbers of fish in a year class being infected every year. Alternatively, large numbers of fish might be infected when young, but growth of *I. hoferi* in the host increases to diagnosable levels in only a small percentage of the fish each year; the relative number of fish reaching diagnosable levels of infection each year varies, perhaps as a result of different environmental conditions.

Nine years of intensive disease study have dramatically increased our understanding of the interaction of disease with changes in population biomass. Indeed, this is the most comprehensive study of disease ever conducted in a marine fish population. We have clearly established that disease information improves our ability to estimate population abundance, and that prevalence of any single pathogen is not sufficient to significantly improve estimates of population abundance except in catastrophic disease episodes like 1993. The advantage of long-term study was clearly apparent with *I. hoferi*, which did not significantly alter population biomass estimates until the 8^{th} year of the study (2001).

Disease prevalence determined only after an epidemic has been detected often provides the wrong information on the cause of population decline. In PWS, disease was twice quantified 1 year before changes in population abundance were detected by traditional abundance estimates. First, high virus and ulcer prevalence in April 1998 were documented a full year before population decline was detected in 1999 (Marty et al. 2003). And second, as shown here, effects of the *I. hoferi* outbreak in 2001 were not detected at the population level until 2002. By the time population decline was detected in the field in 1999 and 2002, disease prevalence had changed enough that the cause of population decline could no longer be determined.

Disease may significantly affect recruitment (Marty et al. 2003). Two of the lowest recruitment estimates on record, in 1994 and 1999, followed increased natural mortality of adults in 1993 and 1998. Because adults are spatially separated from juveniles, our original hypothesis was that

disease in adults would not significantly affect recruitment. Our current mathematical model does not provide strong support for the relation of disease and recruitment, but as ADFG continues to monitor I. hoferi and ulcer prevalence in PWS, the relation can be tested again.

Disease in PWS Pacific herring is oscillating in a roughly 4-year cycle, the amplitude of which is decreasing with each cycle since 1993. PWS Pacific herring had a major VHSV-ulcer disease outbreak in 1993, moderate disease in 1997–1998, and mild disease in 2002. Our original hypothesis was that disease was a sporadic event associated with exceeding carrying capacity, but the 1998, 2001, and 2002 disease events occurred when the population was relatively low.

Conclusions

Three major diseases are limiting recovery of the Pacific herring population of Prince William Sound, Alaska. Ulcers and viral hemorrhagic septicemia were the major diseases limiting population recovery in the late 1990s, and VHSV prevalence was again relatively high in 2002. *I. hoferi* did not cause unexpected mortality in the 1990s, but caused significant mortality in 2001. Although detailed disease study ceased after 2002, evidence from ongoing limited disease studies by ADFG indicates that the *I. hoferi* prevalence has doubled from 2002 to 2004, but it is not yet as high as in 2001. As the 1999 year class hits 6 years old in 2005, prevalence of *I. hoferi* may again peak as it did in 2001 when the dominant 1994 and 1995 year classes were 6 and 7 years old. Because of the increasing proportion of the population infected with *I. hoferi*, it will be many years before the population recovers to levels before the spill.

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Tables

Table 1. Time series of a VHSV-ulcer index (calculated by multiplying the relative frequency of VHSV+ fish with the relative frequency of fish with ulcers, with a lower ulcer bound of 0.5%) and *I. hoferi* prevalence for spring samples of Prince William Sound Pacific herring. Data are stratified by age groups 3–4 and 5+. Also given is the VHSV-ulcer index pooled over all ages, as used in Marty et al. (2003).

	ages 3–4		ages 5–9		pooled ages
Year	VHSV-ulcer index	*I. hoferi*	VHSV-ulcer index	*I. hoferi*	VHSV-ulcer index
1994	1.04%	21.1%	0.06%	23.6%	0.14%
1995	0.14%	7.6%	0.03%	26.5%	0.06%
1996	0.00%	13.6%	0.00%	26.4%	0.00%
1997	0.09%	6.8%	0.05%	25.9%	0.07%
1998	0.60%	9.6%	0.16%	35.4%	0.44%
1999	0.01%	11.6%	0.00%	30.2%	0.01%
2000	0.00%	7.5%	0.00%	23.9%	0.00%
2001	0.01%	23.2%	0.01%	48.6%	0.01%
2002	0.14%	8.9%	0.04%	32.9%	0.10%

Table 2. Statistics for model fits of Prince William Sound Pacific herring, including weighted residual sums of squares (RSS), number of parameters, unweighted (unwt) sums of squares components for each dataset, unexplained variance components for each dataset, and likelihood statistics. Models are designated by background natural mortality M_0 (A: estimated by age groups 3–4 and 5+, B: estimated as a constant over all ages, C: set equal to 0.25) and by disease treatment type: 1) VHSV-ulcer index+*I. hoferi* (age); 2) VHSV-ulcer index (age); 3) *I. hoferi* (age); 4) VHSV-ulcer index (ages 3–4) + *I. hoferi* (ages 5+); and 5) VHSV-ulcer index (ages pooled).

Model	A1	A2	A3	A4	A5	B1	B2	B3	B4	B5	C1	C2	C3	C4	C5
weighted RSS	2.465	2.528	2.512	2.519	2.566	2.497	2.575	2.534	2.559	2.588	2.506	2.628	2.534	2.563	2.649
#parameters	44	40	40	40	38	43	39	39	39	37	42	38	38	38	36
unwt.RSS Seine	0.241	0.237	0.314	0.323	0.314	0.266	0.273	0.329	0.346	0.311	0.271	0.236	0.328	0.342	0.263
unwt.RSS Spawn	0.362	0.365	0.363	0.350	0.370	0.352	0.362	0.360	0.352	0.385	0.357	0.412	0.360	0.357	0.419
unwt.RSS Egg	2.050	2.170	2.035	2.071	2.182	2.060	2.184	2.022	2.082	2.186	2.006	2.086	2.012	2.044	2.069
unwt.RSS Milt	1.675	1.683	1.635	1.621	1.583	1.697	1.697	1.668	1.639	1.598	1.750	1.874	1.681	1.684	1.866
Unexplained variance components															
σ^2_Seine	0.0090	0.0093	0.0092	0.0092	0.0094	0.0091	0.0094	0.0093	0.0094	0.0095	0.0092	0.0096	0.0093	0.0094	0.0097
σ^2_Spawn	0.0090	0.0093	0.0092	0.0092	0.0094	0.0091	0.0094	0.0093	0.0094	0.0095	0.0092	0.0096	0.0093	0.0094	0.0097
σ^2_Egg	0.0181	0.0185	0.0184	0.0185	0.0188	0.0183	0.0189	0.0186	0.0188	0.0190	0.0184	0.0192	0.0186	0.0188	0.0194
σ^2_Milt	0.0181	0.0185	0.0184	0.0185	0.0188	0.0183	0.0189	0.0186	0.0188	0.0190	0.0184	0.0192	0.0186	0.0188	0.0194
Likelihood statistics															
ln L	243.4	239.9	240.8	240.4	237.9	241.6	237.4	239.6	238.3	236.7	241.2	234.7	239.6	238.1	233.5
AIC	-398.7	-399.9	-401.7	-400.8	-399.8	-397.3	-396.8	-401.3	-398.5	-399.5	-398.3	-393.3	-403.2	-400.1	-395.1
AICc	-381.4	-385.7	-387.5	-386.7	-387.1	-380.8	-383.4	-387.9	-385.1	-387.5	-382.6	-380.7	-390.5	-387.5	-383.8
Δ	9.2	4.8	3.0	3.9	3.4	9.8	7.1	2.7	5.4	3.0	7.9	9.9	0.0	3.1	6.7

Table 3. Estimates of disease parameters, background mortality M_0, and resultant natural mortality M from 1992 – 2003 in Pacific herring from Prince William Sound. Models as described in Table 2.

Model	A1	A2	A3	A4	A5	B1	B2	B3	B4	B5	C1	C2	C3	C4	C5
Disease parameters															
β_VHSV(3-4)	0.6143	54.0817	0.0000	48.7129	0.0000	0.6246	60.296	0.000	54.607	0.000	0.598	49.967	0.000	51.337	0.000
β_*I. hoferi*(3-4)	2.6790	0.0000	1.9902	0.0000	0.0000	2.9669	0.000	2.412	0.000	0.000	2.872	0.000	2.410	0.000	0.000
β_VHSV(5+ or all)	0.5182	0.6538	0.0000	0.0000	67.9147	0.5010	0.629	0.000	0.000	81.060	0.457	0.679	0.000	0.000	49.373
β_*I. hoferi*(5+)	0.1583	0.0000	0.5444	0.5645	0.0000	0.1651	0.000	0.562	0.566	0.000	0.277	0.000	0.572	0.601	0.000
Estimated prevalence (%) in 1992-93 by disease category															
*x*_VHSV(3-4)	0.8	1.0	0.0	1.2	0.0	0.8	1.0	0.0	1.1	0.0	0.8	1.1	0.0	1.1	0.0
*x*_*I. hoferi*(3-4)	20.6	0.0	29.9	0.0	0.0	19.7	0.0	24.7	0.0	0.0	19.4	0.0	24.5	0.0	0.0
*x*_VHSV(5+ or all)	98.1	94.8	0.0	0.0	0.8	94.0	91.3	0.0	0.0	0.7	89.1	99.1	0.0	0.0	1.2
*x*_*I. hoferi*(5+)	85.7	0.0	100.0	100.0	0.0	90.1	0.0	100.0	100.0	0.0	88.2	0.0	100.0	100.0	0.0
Background mortality by age															
M_0(3-4)	0.553	0.601	0.528	0.570	0.250	0.294	0.343	0.259	0.282	0.353	0.250	0.250	0.250	0.250	0.250
M_0(5-8)	0.256	0.287	0.250	0.250	0.368	0.294	0.343	0.259	0.282	0.353	0.250	0.250	0.250	0.250	0.250
M_0(9+)	0.689	0.702	0.688	0.632	0.419	0.579	0.564	0.636	0.519	0.420	0.673	0.844	0.669	0.636	0.839
Natural mortality including disease (age, year)															
M(3-4,92-93)	1.366	1.403	1.432	1.400	1.210	1.182	1.278	1.167	1.154	1.243	1.074	1.064	1.142	1.069	1.185
M(3-4,94)	1.398	1.426	1.071	1.275	0.469	1.291	1.328	0.968	1.119	0.474	1.194	0.982	0.958	1.012	0.322
M(3-4,95)	0.780	0.681	0.691	0.642	0.409	0.548	0.433	0.460	0.363	0.402	0.495	0.324	0.451	0.326	0.280
M(3-4,96)	1.005	0.601	0.843	0.570	0.368	0.808	0.343	0.655	0.282	0.353	0.743	0.250	0.646	0.250	0.250
M(3-4,97)	0.754	0.653	0.673	0.617	0.419	0.518	0.402	0.437	0.334	0.414	0.467	0.298	0.428	0.299	0.287
M(3-4,98)	0.847	0.989	0.735	0.912	0.719	0.624	0.788	0.515	0.675	0.788	0.568	0.603	0.506	0.615	0.492
M(3-4,99)	0.925	0.607	0.790	0.575	0.372	0.715	0.350	0.587	0.287	0.358	0.654	0.255	0.577	0.255	0.253
M(3-4,00)	0.778	0.601	0.690	0.570	0.368	0.545	0.343	0.459	0.282	0.353	0.493	0.250	0.449	0.250	0.250
M(3-4,01)	1.525	0.605	1.148	0.573	0.376	1.460	0.347	1.079	0.285	0.362	1.347	0.253	1.069	0.253	0.256
M(3-4,02)	0.828	0.682	0.724	0.642	0.437	0.603	0.434	0.502	0.363	0.435	0.547	0.324	0.492	0.327	0.300
M(3-4,03)	0.828	0.682	0.724	0.642	0.437	0.603	0.434	0.502	0.363	0.435	0.547	0.324	0.492	0.327	0.300

Model	A1	A2	A3	A4	A5	B1	B2	B3	B4	B5	C1	C2	C3	C4	C5
M5+(92-93)	1.288	1.254	1.036	1.081	1.210	1.261	1.198	1.084	1.116	1.243	1.305	1.367	1.099	1.170	1.185
M5+(94)	0.294	0.287	0.387	0.393	0.469	0.334	0.344	0.401	0.425	0.474	0.318	0.250	0.395	0.403	0.322
M5+(95)	0.298	0.287	0.405	0.411	0.409	0.338	0.343	0.419	0.443	0.402	0.326	0.250	0.413	0.423	0.280
M5+(96)	0.298	0.287	0.404	0.410	0.368	0.338	0.343	0.419	0.442	0.353	0.325	0.250	0.413	0.422	0.250
M5+(97)	0.298	0.287	0.403	0.409	0.419	0.338	0.344	0.417	0.441	0.414	0.325	0.250	0.411	0.420	0.287
M5+(98)	0.314	0.288	0.464	0.473	0.719	0.355	0.344	0.481	0.505	0.788	0.354	0.251	0.477	0.490	0.492
M5+(99)	0.305	0.287	0.430	0.437	0.372	0.345	0.343	0.445	0.469	0.358	0.338	0.250	0.440	0.451	0.253
M5+(00)	0.294	0.287	0.389	0.395	0.368	0.334	0.343	0.403	0.426	0.353	0.318	0.250	0.397	0.405	0.250
M5+(01)	0.336	0.287	0.557	0.570	0.376	0.377	0.343	0.578	0.603	0.362	0.395	0.250	0.576	0.595	0.256
M5+(02)	0.309	0.287	0.447	0.455	0.437	0.350	0.343	0.464	0.487	0.435	0.346	0.250	0.458	0.470	0.300
M5+(03)	0.309	0.287	0.447	0.455	0.437	0.350	0.343	0.464	0.487	0.435	0.346	0.250	0.458	0.470	0.300

Table 4. Estimates of maturity (Mat) for ages 3 and 4 before 1998 (<98) and from 1998 – 2003 (≥98), recruitment (age 3 abundance in millions of fish), and spawning biomass (in metric tons, assuming median recruitment for 2004) for Pacific herring in Prince William Sound, Alaska. Models as described in Table 2.

Model	A1	A2	A3	A4	A5	B1	B2	B3	B4	B5	C1	C2	C3	C4	C5
Percentage of age spawning (=100% for ages 5+)															
Mat,<98-age 3	16.1%	15.8%	16.9%	16.6%	25.7%	24.1%	23.1%	26.7%	26.8%	22.3%	27.1%	29.6%	27.3%	29.2%	29.2%
Mat,≥98-age 3	50.1%	62.2%	55.5%	62.0%	82.3%	64.0%	82.5%	70.8%	85.3%	82.2%	69.0%	98.1%	71.6%	89.7%	100.0%
Mat,<98-age 4	60.0%	60.0%	60.0%	60.0%	68.1%	69.9%	65.5%	74.5%	72.8%	63.4%	75.5%	76.4%	75.7%	76.7%	75.3%
Mat,≥98-age 4	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%
Recruitments															
N3,1990	86.1	111.5	68.4	80.7	69.9	58.2	80.6	43.3	51.8	83.4	46.8	52.5	42.0	46.6	48.4
N3,1991	1732.2	1867.9	1670.1	1739.1	1163.0	1167.6	1321.3	1055.2	1093.9	1347.4	1030.9	1019.8	1028.7	1004.4	1062.5
N3,1992	129.6	155.5	76.2	85.4	58.6	82.7	105.9	47.7	51.9	68.3	72.0	94.7	47.8	51.5	78.0
N3,1993	239.6	255.3	196.1	218.1	109.8	169.9	193.1	132.2	144.4	119.0	149.0	144.7	129.5	131.3	99.2
N3,1994	67.6	72.1	68.2	77.3	35.3	48.2	54.0	46.2	51.4	39.2	44.2	39.2	45.3	47.0	30.0
N3,1995	293.6	197.2	276.6	220.9	148.4	206.1	133.4	186.6	135.2	151.4	184.1	99.2	182.5	125.1	106.8
N3,1996	179.2	134.9	179.6	158.3	110.4	129.1	96.7	123.6	101.1	113.8	115.7	67.7	120.8	92.5	72.0
N3,1997	257.0	248.0	246.1	263.6	194.2	187.8	181.3	176.8	178.6	205.4	172.8	131.5	173.8	164.3	126.7
N3,1998	168.3	133.8	145.3	133.3	103.0	123.1	91.5	109.2	88.7	105.6	116.0	74.7	108.1	83.9	75.6
N3,1999	11.0	3.6	7.7	5.0	4.6	7.0	1.2	5.4	2.1	4.9	7.3	1.1	5.4	2.1	3.2
N3,2000	44.9	28.3	42.2	32.5	22.8	36.3	21.0	34.4	23.3	22.3	33.2	15.7	33.8	22.0	15.6
N3,2001	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
N3,2002	234.3	196.0	222.8	201.2	154.3	186.5	151.4	176.1	148.9	154.6	174.8	131.3	173.9	142.1	129.0
N3,2003	20.8	12.9	26.0	21.6	11.3	16.5	9.9	19.7	14.9	10.9	15.2	6.4	19.4	13.8	6.4
Estimated spawning biomass															
2004	17,181	20,450	17,490	18,833	20,074	17,740	21,391	17,757	19,761	21,015	17,688	21,507	17,718	19,639	21,485

Table 5. Correlations among ln recruitment estimates from Model C4 and disease time series from Table 1. Correlations with no lag use recruitment estimates and disease data from 1994 – 2002; correlations with lag 1 use recruitment estimates from 1995 – 2003 and disease data from 1994 – 2002.

	ages 3–4		ages 5–9		pooled ages
	VHSV	*I. hoferi*	VHSV	*I. hoferi*	VHSV
Correlation (no lag)	0.268	-0.677	0.318	-0.774	0.315
P-value	0.486	0.045	0.404	0.014	0.409
Correlation (lag 1)	0.130	0.453	-0.056	0.197	-0.121
P-value	0.739	0.220	0.887	0.611	0.756

Figures

Figure 1. Biomass estimates of adult prespawning Pacific herring in Prince William Sound, Alaska. Unexploited spawning biomass projected in the year before spawning (○) and calculated after spawning (●) using an age-structured assessment model. After 2000, biomass estimates were not projected before the spawning season.

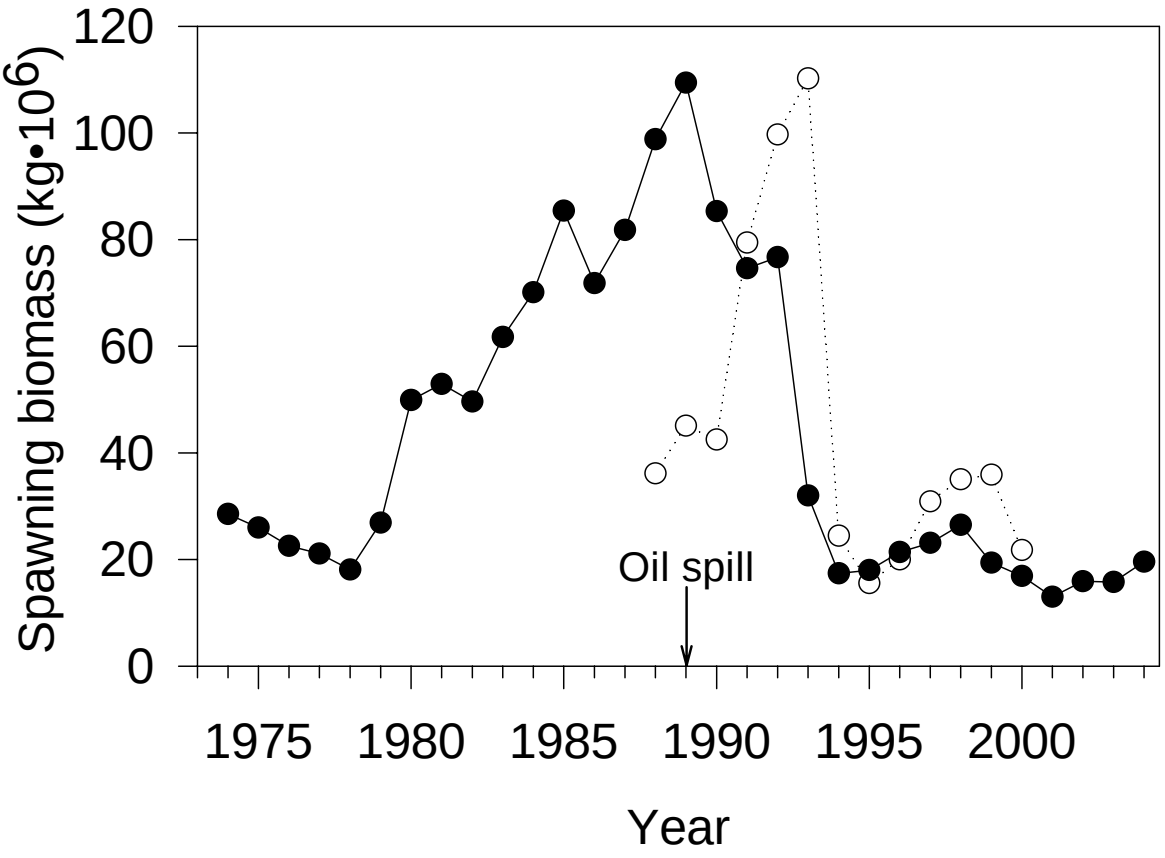


Figure 2. Mean fish age (●) and prevalence of *Ichthyophonus hoferi* (bars) in Pacific herring sampled from Prince William Sound, Alaska (spring samples only; n = 233–300 per year). Shaded part of bar is the prevalence of fish with sum-*Ichthyophonus* scores >10 (among all fish, sum-*Ichthyophonus* scores ranged from 0 to 25).

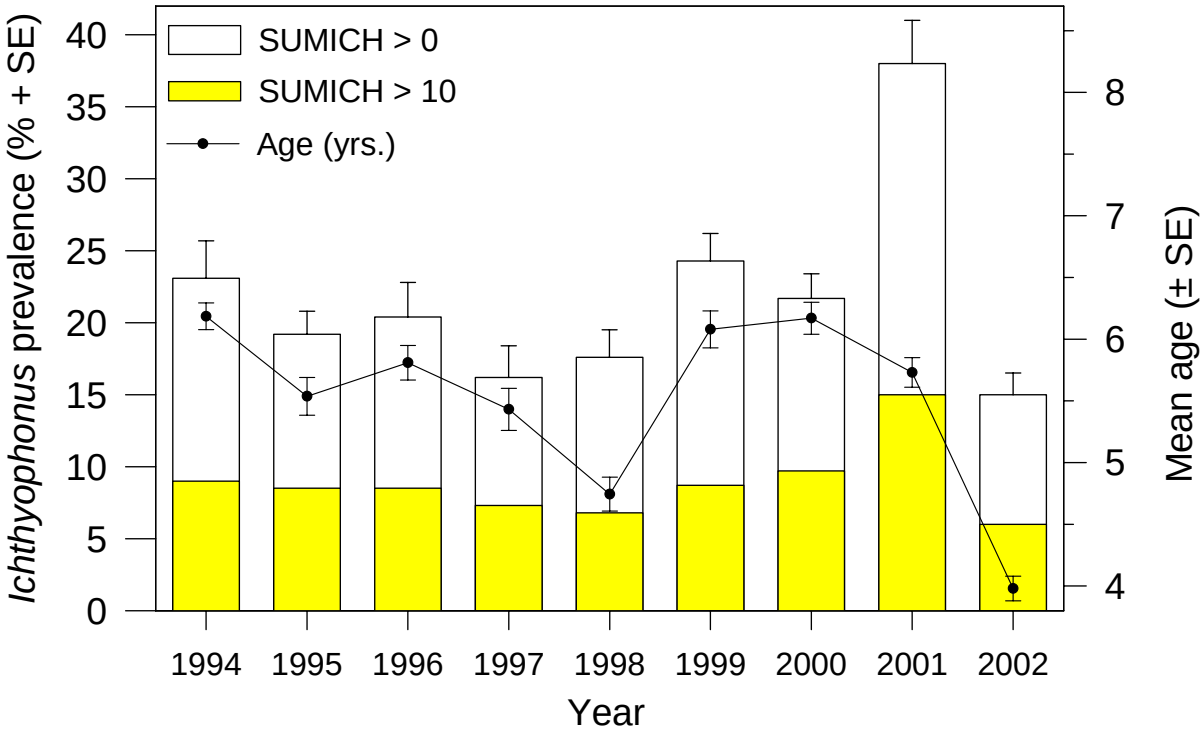


Figure 3. Prevalence of lesions and virus in adult Pacific herring sampled in the spring from Prince William Sound, Alaska (n = 233–300 per year); (a) external lesion focal skin reddening (moderate, light bars; severe, darker bars); and (b) viral hemorrhagic septicemia virus (VHSV).

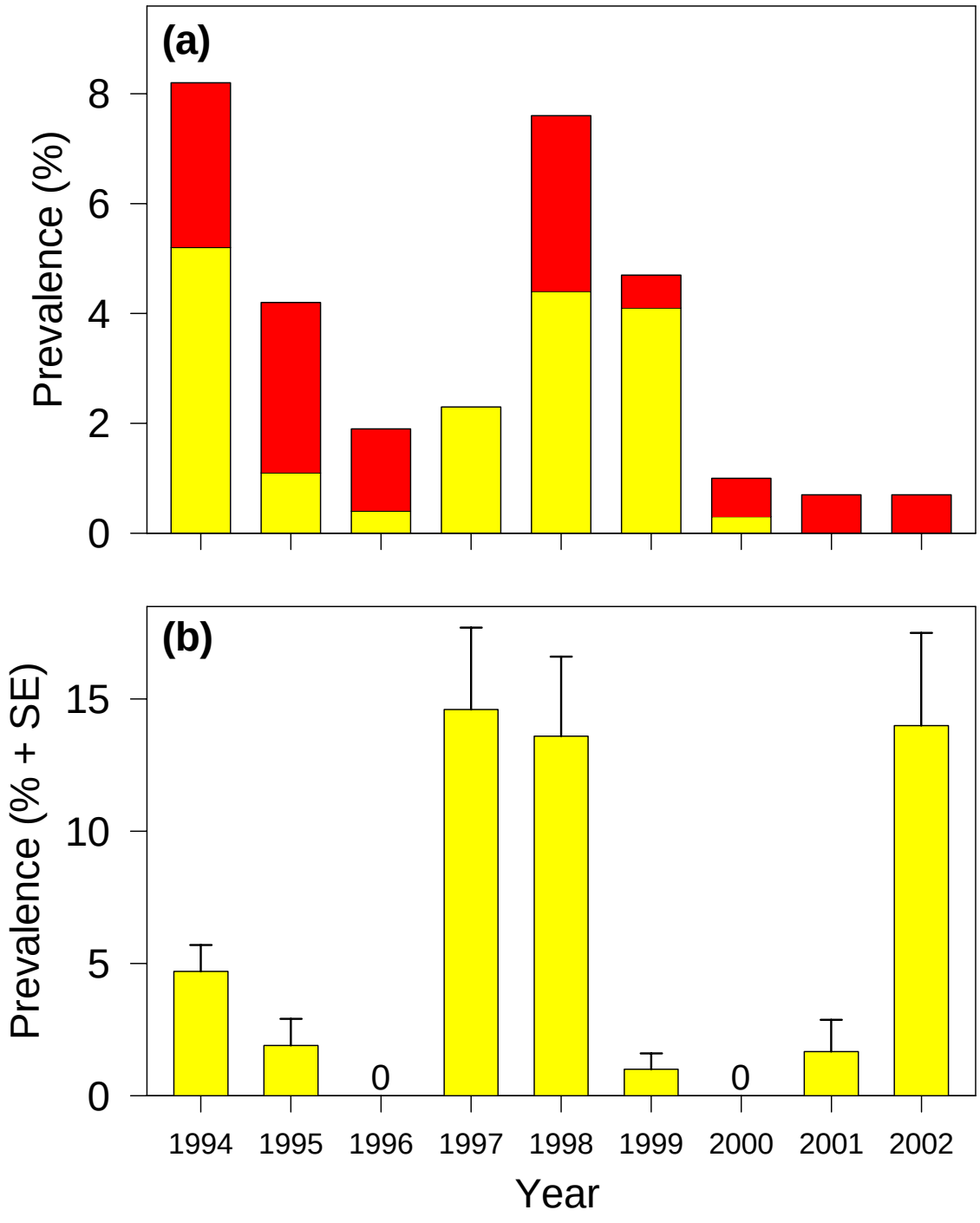


Figure 4. VHSV-ulcer disease index and proportion of 3-year-olds in samples of adult Pacific herring from Prince William Sound, Alaska. Arbitrary reference line highlights years with a high proportion of 3-year-olds. ND = no data.

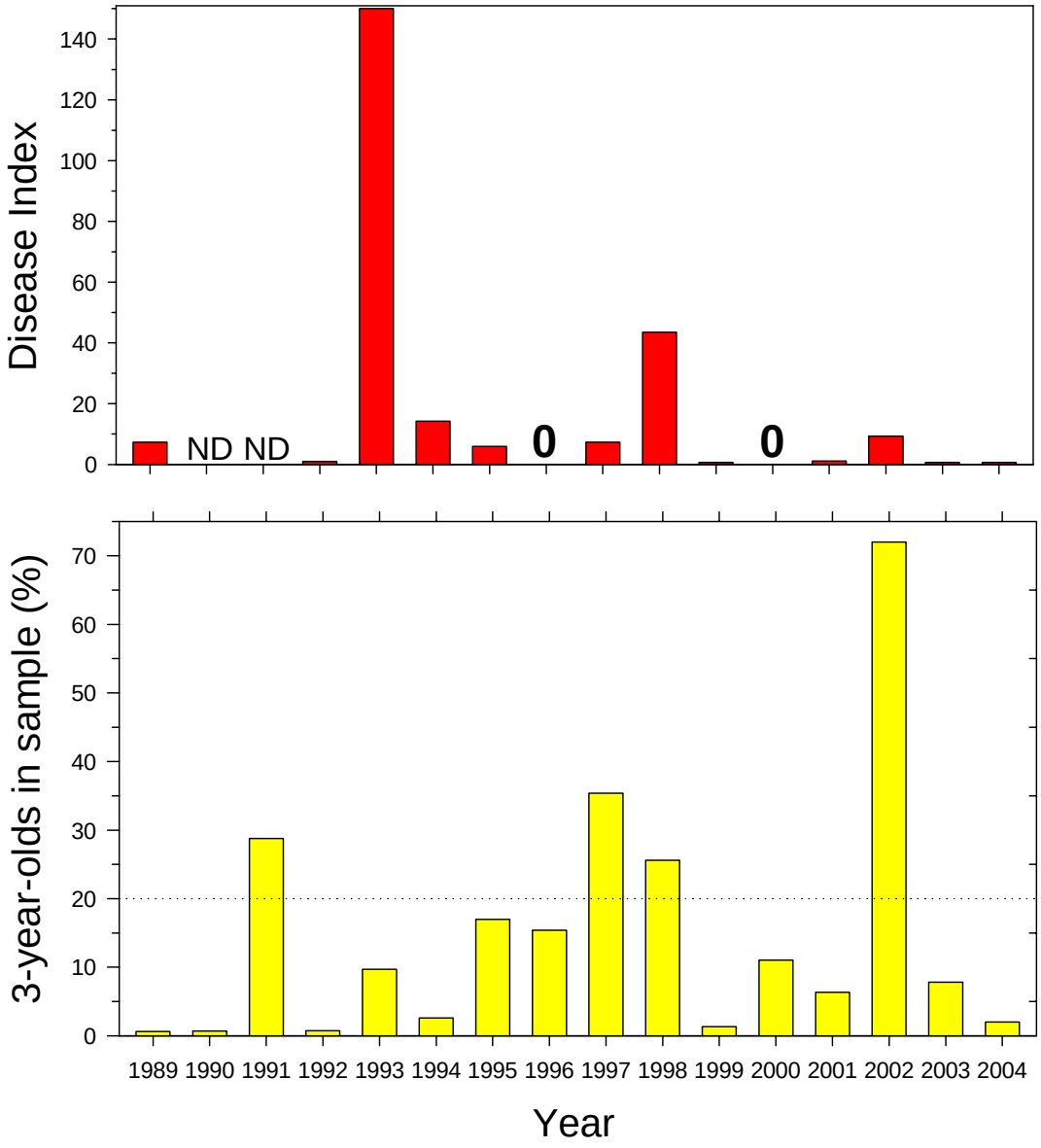


Figure 5. Prevalence of herring worms (Anisakidae) in the abdominal cavity of adult Pacific herring sampled from Prince William Sound, Alaska.

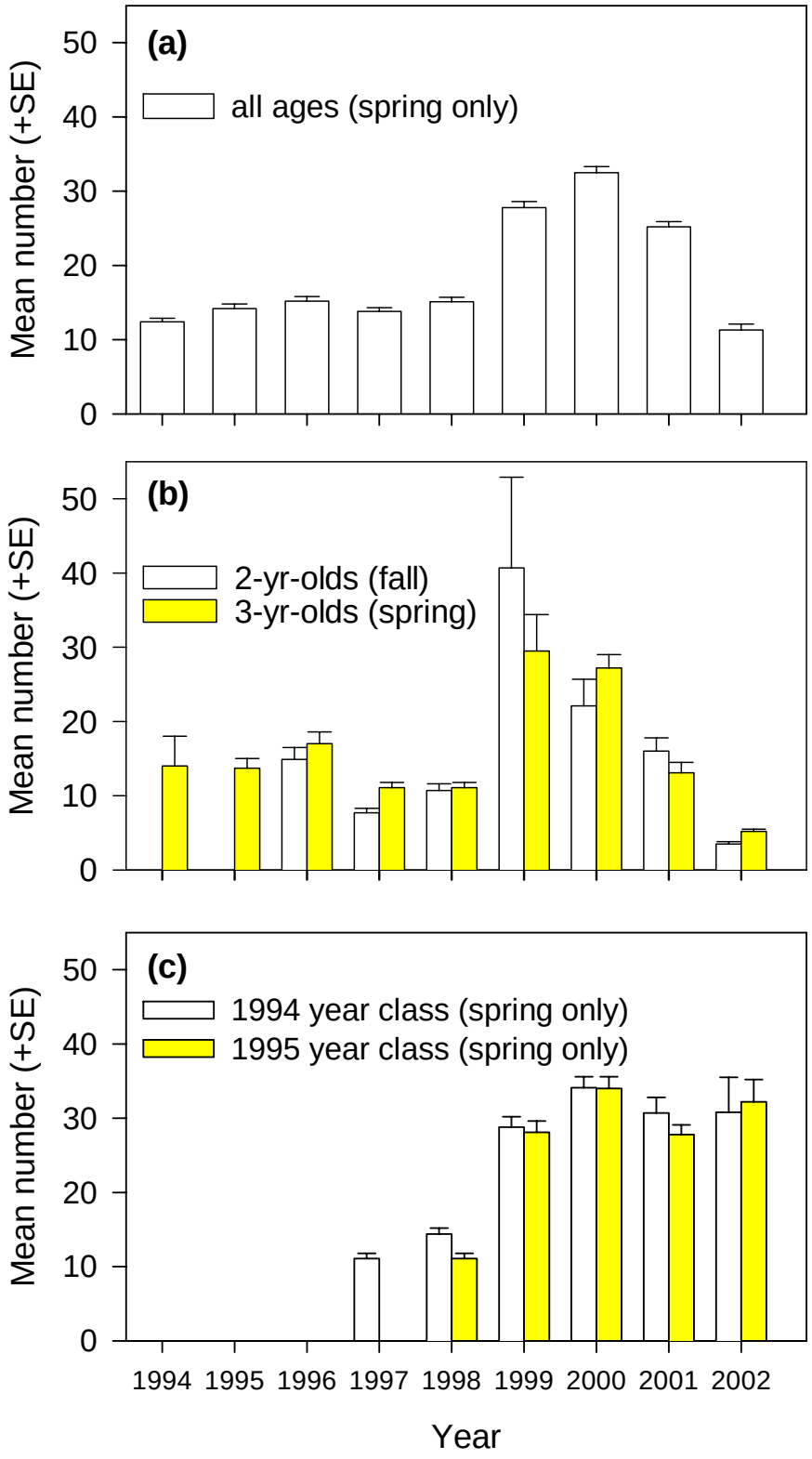


Figure 6. Prevalence of copepod parasites on the medial operculum (gill covering) of adult Pacific herring from Prince William Sound, Alaska. ND = no data.

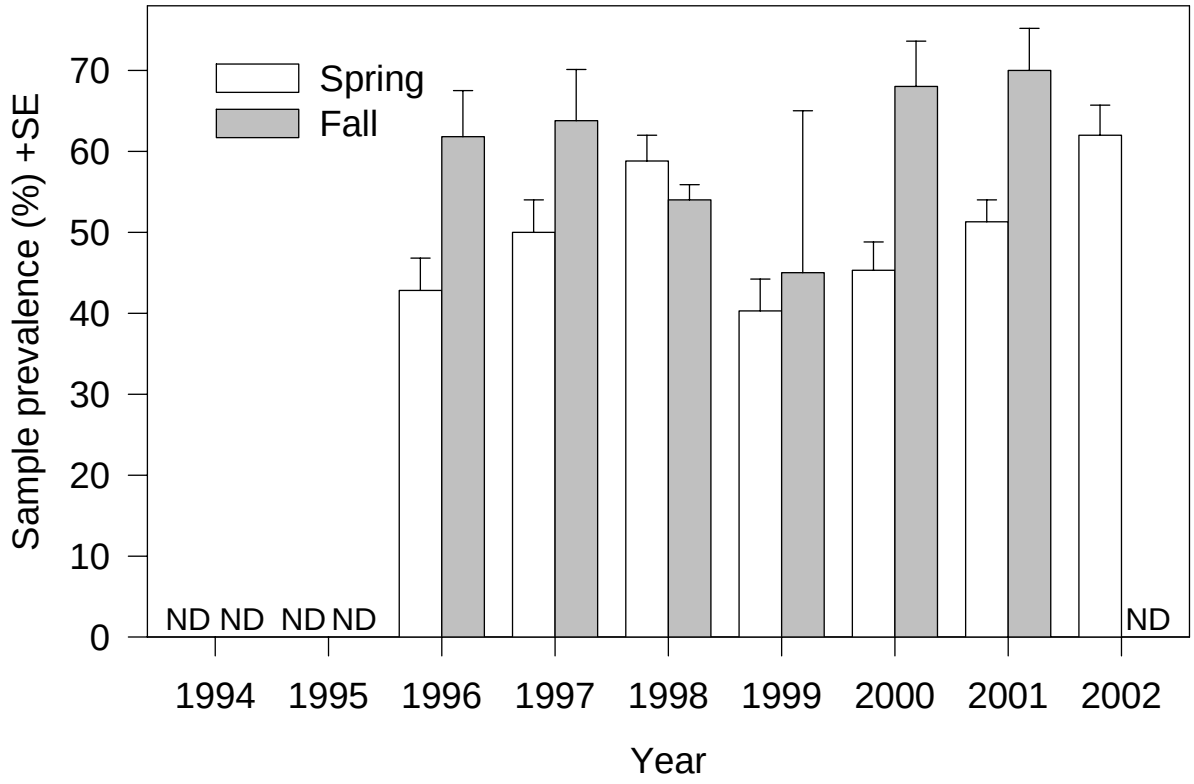


Figure 7. Estimates of natural mortality among Pacific herring in Prince Willaim Sound, Alaska, by age groups 3–4 and 5+ from 1992–2003 from disease models A1, A2, … C15 described in Table 2.

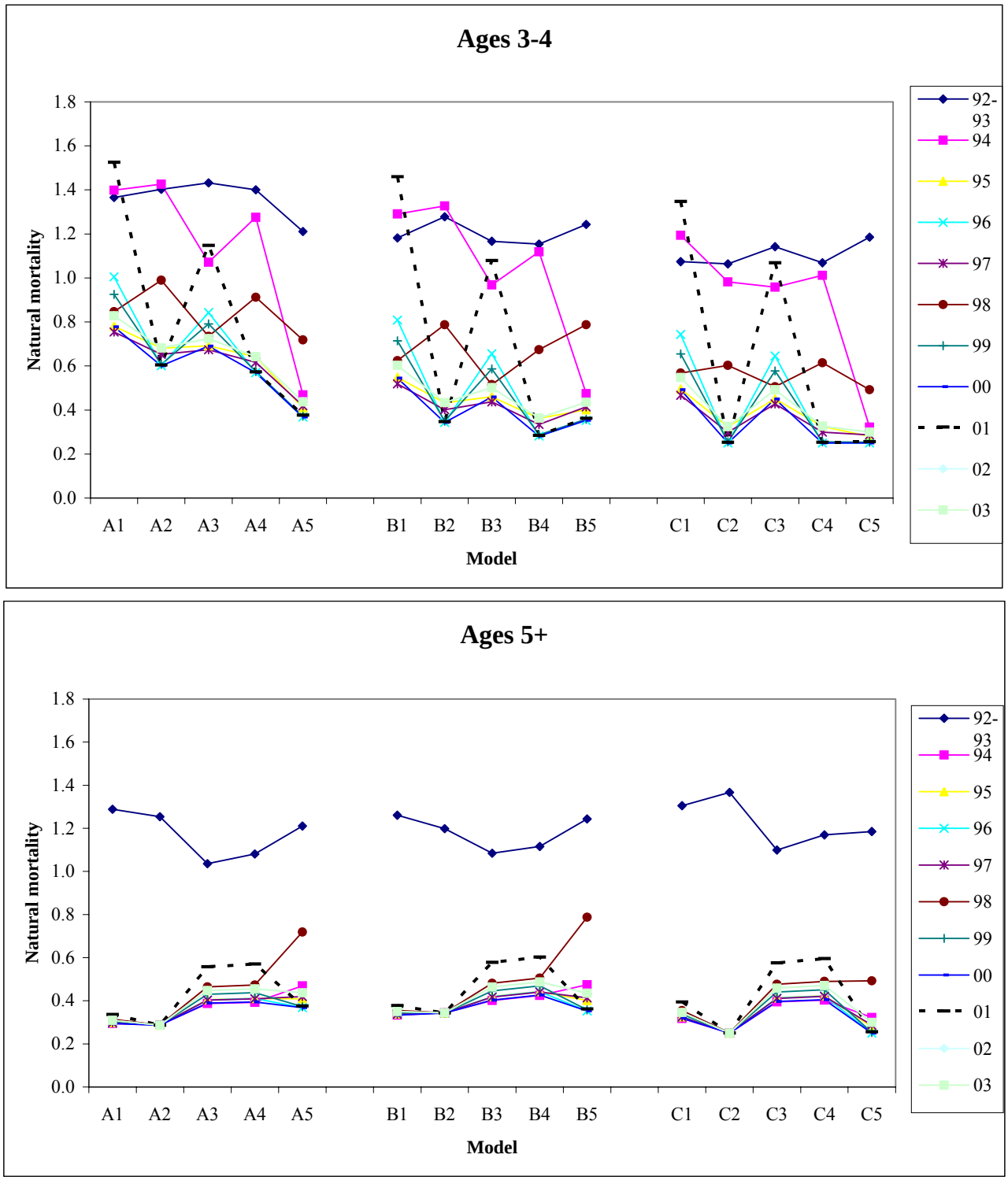
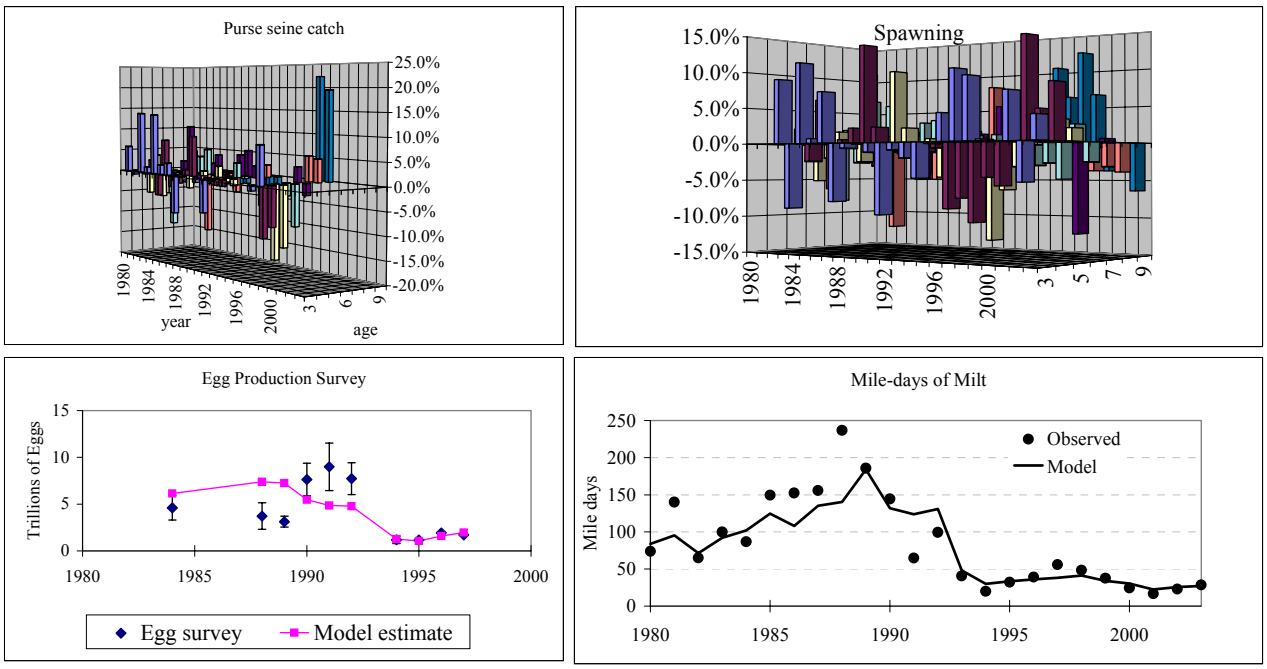


Figure 8. Residuals patterns for purse seine and spawning age composition and observed data compared with model estimates for egg survey data and mile-days of milt for the chosen best model C4. Model results for the Pacific herring population of Prince William Sound.



Appendix 1. Disease and population assessment of Pacific herring in Prince William Sound, Alaska.

The citation and abstract of the first publication resulting from this work is included here:

Quinn, T. J., II, G. D. Marty, J. Wilcock, and M. Willette. 2001. Disease and population assessment of Pacific herring in Prince William Sound, Alaska. Pages 363–379 *in* F. Funk, J. Blackburn, D. Hay, A. J. Paul, R. Stephensen, R. Toreson, and D. Witherell, editors Herring: Expectations for a new millennium. University of Alaska Sea Grant, AK-SG-01-04, Fairbanks.

Disease is rarely incorporated into fish stock assessment models because of a lack of information. A unique time series started in 1993–1994 on prevalence of two major diseases in Prince William Sound Pacific herring: viral hemorrhagic septicemia virus (VHSV) and the fungus-like organism *Ichthyophonus hoferi*. This research was prompted by a severe population decline in 1993 that shut down the fishery. Prevalence of ***I. hoferi*** appeared to be unrelated to population decline. We modified the herring assessment model to let natural survival be linearly and negatively related to disease prevalence. Eight models allowed various natural mortality changes. There was a clear increase in natural mortality in 1992–1993, and VHSV information enhanced model fit compared to a constant natural mortality model. Because of higher VHSV prevalence in 1993 and 1997–1998, estimated natural mortality was higher in these years and estimated spawning biomass was reduced. However, the true effect of higher VHSV prevalence on natural mortality and spawning biomass cannot yet be unambiguously determined, because there is limited information for 1992–1993 and six of the eight mortality models produced nearly identical fits. Nevertheless, using VHSV prevalence for modeling and forecasting is more conservative that using constant natural mortality. Neither disease series was significantly correlated with recruitment, suggesting that disease has its main effect at the adult stage. However, the presence of a negative correlation between recruits and VHSV prevalence lagged 1 year suggest that disease may affect juveniles.

Appendix 2. Role of disease in abundance of a Pacific herring (*Clupea pallasi*) population.

The citation and abstract of the second publication resulting from this work is included here:

Marty, G. D., T. J. Quinn, II, G. Carpenter, T. R. Meyers, and N. H. Willits. 2003. Role of disease in abundance of a Pacific herring (*Clupea pallasi*) population. Canadian Journal of Fisheries and Aquatic Sciences 60:1258-1265.

Disease significantly affects population abundance of Pacific herring (*Clupea pallasi*). Comprehensive epidemiological study of the Pacific herring population of Prince William Sound, Alaska, U.S.A., from 1994 to 2000 included complete necropsy examination of 230–500 fish each spring and 40–160 fish each fall (total n = 2983 fish). Mortality is best estimated, through modifications of an age structured assessment model, using a disease index that combines the prevalence of viral hemorrhagic septicemia virus (VHSV) with the prevalence of ulcers. Risk factors for an epidemic include poor body condition and abundant recruitment before spawning in the spring. Prevalence of the pathogen *Ichthyophonus hoferi* increased as fish aged, but changes in *I. hoferi* prevalence were not related to changes in population abundance. Disease that caused an epidemic in 1998 (VHSV and ulcers) nearly disappeared from the population when changes in abundance were detected by traditional stock assessment methods in 1999. Disease significantly affects recruitment—the two lowest recruitment estimates on record, in 1994 and 1999, followed increased natural mortality of adults in 1993 and 1998.