Exxon Valdez Oil Spill
Restoration Project Annual Report

Effect of Disease on Recovery of Pacific Herring in Prince William Sound, Alaska, Spring 1999

Restoration Project 99462
Annual Report

This annual report has been prepared for peer review as part of the *Exxon Valdez* Oil Spill Trustee Council restoration program for the purpose of assessing project progress. Peer review comments have not been addressed in this annual report.

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Study History: This project continues the field component of project 98162 (the final report for 98162 has been approved). Sampling in fall 1998 was funded under this project (99462), but results were included in the final report for 98162; therefore, results from fall 1998 samples are not repeated here. Detailed histopathological examination, blood analysis, and a modeling component are 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.

Abstract: Due to low population biomass, Pacific herring (*Clupea pallasi*) fisheries in Prince William Sound were severely curtailed in 1999, and all Pacific herring fisheries are closed for the 2000 season. Detailed disease study on 300 Pacific herring in April 1999 used the same methods as used every year since 1994. The prevalence of viral hemorrhagic septicemia virus was significantly lower in spring 1999 (1%) than in 1998 (15%). Prevalence of the fungus-like organism *Ichthyophonus hoferi* was slightly higher in 1999 (24%) than in 1998 (18%). However, *Ichthyophonus* is more common in older fish, and differences in prevalence can be attributed to poor recruitment and older fish remaining in the population in 1999 (mean age = 6.1 yrs.) than in 1998 (mean age = 4.7 years). Also, unlike the virus, mortality resulting from *Ichthyophonus* infection is probably more constant over time. We conclude that most disease-related mortality occurred during and after spawning in 1998, when prevalence of ulcers and virus was high, and when fish behavior was abnormal. Resultant mortality was not documented until spring 1999 because Pacific herring disperse during the summer, and the most accurate population estimates were not made until early April when the fish congregated to spawn.

Key Words: *Clupea pallasi*, disease, *Exxon Valdez* oil spill, *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, and viral erythrocytic necrosis virus, VEN). All project data is stored in an Excel spreadsheet (188 columns and 2536 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. Data are available on a case by case basis.

Citation:

Marty, G.D., T.R. Meyers, and G. Carpenter. 2000. Effect of disease on recovery of Pacific Herring in Prince William Sound, Alaska, Spring 1999, *Exxon Valdez* Oil Spill Restoration Project Annual Report (Restoration Project 99462), Alaska Department of Fish and Game, Habitat and Restoration Division, Anchorage, Alaska.

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Executive Summary

Introduction

The estimated spawning biomass of Pacific herring (*Clupea pallasi*) in Prince William Sound, Alaska, decreased from about 36,000 metric tons in 1998 to only 22,000 metric tons in 1999. Due to low population biomass, Pacific herring fisheries in Prince William Sound were severely curtailed in 1999, and all Pacific herring fisheries are closed for the 2000 season. Studies of the Prince William Sound Pacific herring population since 1994 provided evidence that disease is a significant variable in population fluctuations. Viral hemorrhagic septicemia virus is associated with acute mortality that can be significant on a population scale. The other major disease—caused by the fungus-like organism *Ichthyophonus hoferi*—is chronic, decreases the life span of affected fish, but probably does not play a major role in unexpected population fluctuations. This report describes the major disease-related findings in Pacific herring sampled from Prince William Sound during the spring of 1999. It also builds on data gathered from previous studies to provide evidence to explain the fishery closures in 1999.

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

Three hundred adult Pacific herring from Prince William Sound were sampled at random and subjected to complete necropsy in April 1999. Fish were all sampled from the north end of Montague Island (Stockdale Harbor, Montague Point, and Rocky Bay). Analysis of all fish included determination of age, weight and length, gross examination, and culture of head kidney and spleen for virus isolation. In all fish with severe external lesions, kidney was cultured for bacteria (all were negative). A project supported by the National Science Foundation provided funds for 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, except for total prevalence of *Ichthyophonus hoferi*, which was determined by histopathological examination.

Results

Pacific herring in Prince William Sound were healthier in 1999 than in 1998. As evidence, prevalence of viral hemorrhagic septicemia virus in spring samples was significantly less in 1999 (1%) than in 1998 (14%), and prevalence of skin ulcers was significantly less in 1999 (0.6%) than in 1998 (3.2%). Prevalence of *Ichthyophonus* was slightly higher in 1999 (24%) than in 1998 (18%). However, previous study has shown that *Ichthyophonus* is more common in older fish.

Because of poor recruitment of the 1996 year class in 1999, mean age of the spawning population was significantly greater in 1999 (6.1 years) than in 1998 (4.7 years), and age-adjusted prevalence of *Ichthyophonus* was not different.

Discussion

Most of the mortality that led to closure of commercial herring fisheries in 1999 probably occurred during and immediately after spawning in mid-April 1998, when prevalence of ulcers and virus was high, and when fish behavior was abnormal. Resultant mortality was not documented until 1999 because Pacific herring disperse during the summer, and the most accurate population estimates are made in late March and early April when the fish congregate to spawn. By 1999, fish were relatively healthy.

Based on multiple years of data, several patterns are emerging with respect to parasites, disease, and Pacific herring. Viral hemorrhagic septicemia is more common in the young fish in the spring, and expression of virus is associated with significant lesions. Fish have no evidence of virus in the fall. *Ichthyophonus* prevalence and lesion severity are greater in mature fish than in new recruits, and fish are infected with *Ichthyophonus* year round. On a population scale, virus probably contributes more to mortality when the population is young, whereas *Ichthyophonus* may contribute more to mortality when the population is old.

Conclusion

We are able to understand the timing and causes of this population decline only through the availability of extensive pre-crash data—information that has never been available to explain the decline of a wild fish population. Further study, scheduled for April 2000 (project 00462) will provide more information on the effects of disease on herring population recovery. Because population biomass is very low, and recruitment of the 1996 year class was poor, it will be many years before the population recovers. According to the restoration objectives, a large year class must fully recruit into the fishery before a population can be reclassified as "recovered." Pacific herring do not fully recruit into the fishery until they are 5 years old. Even if initial recruitment of the 1997 year class is strong, which at present seems unlikely, population recovery based on restoration objectives cannot be confirmed until at least the year 2002.

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 (about 11×10^7 kg; Figure 1), and the population remained near record levels through 1992. Pacific herring in PWS first spawn when 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 110×10^6 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. Hence, 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.

Disease study supported by the Trustee Council from 1994-1998 identified a virus (viral hemorrhagic septicemia virus, VHSV) and a fungus-like organism (*Ichthyophonus hoferi*) that were important causes of disease on a population scale (Marty et al. 1998). Virus prevalence was highly variable and caused acute disease that could result in unexpected population decline. By comparison, *Ichthyophonus* prevalence was fairly constant and caused chronic disease that probably decreased the life span of affected fish, but did not result in unexpected population decline.

Pacific herring population biomass increased enough in PWS that roe fisheries were re-established 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.

The estimated spawning biomass of Pacific herring in PWS, Alaska, decreased from about 36,000 metric tons in 1998 to only 22,000 metric tons in 1999. Due to low population biomass, Pacific herring fisheries in PWS were severely curtailed in 1999, and all Pacific herring fisheries are closed for the 2000 season. This report describes the major disease-related findings in Pacific herring sampled from PWS during the spring of 1999. It also builds on data gathered from previous studies to provide evidence that the fishery closures in 1999 were a result of mortality during and immediately after spawning in 1998. Indeed, low virus prevalence in spring of 1999 (1%) provided strong evidence that fish in 1999 were relatively healthy and already on the road to recovery.

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

Pacific herring were captured by purse seine by a commercial seiner in 15 sets of 20 fish each in April 1999. 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 4 hours before necropsy. In groups of two, 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); 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 frozen for later analysis. A 100-µL plasma aliquot from each fish was frozen separately for IgM analysis (Davis et al. 1999). 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 and shipped on ice to the Alaska Department of Fish and Game's 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 was 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 *Ichthyophonus* prevalence scores, which are reported here).

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; and

7) herring worms (Anisakidae) in the peritoneal cavity were counted.

Statistical Analysis

Detailed statistical analysis will be included as part of the final report. Here, basis summary statistics such as sample size, sample mean, and standard error are reported for major findings. For major parasites, standard error was calculated from the mean prevalence of each organism within each of the 15 20-fish sets.

Results

Pacific herring in PWS were healthier in 1999 than in 1998. As evidence, prevalence of viral hemorrhagic septicemia virus and skin ulcers was significantly less in April 1999 than in April 1998 (Figure 2). Prevalence of *Ichthyophonus* was slightly higher in 1999 (24%) than in 1998 (18%). However, *Ichthyophonus* is more common in older fish (Figure 4). Because of poor recruitment of the 1996 year class in 1999, mean age of the population was significantly greater in 1999 than in 1998 (Figure 3), and age-adjusted prevalence of *Ichthyophonus* was not significantly increased in 1999.

Prevalence of cysts of unknown etiology in the gills was less in April 1999 than in April 1998 (Figure 5). Prevalence of copepod parasites on the medial operculum was also less in April 1999 than in April 1998 (Figure 6). Because copepod parasites are more common in young fish, the decrease in prevalence in 1999 is probably a reflection of poor recruitment and older fish in the population in 1999 than in 1998.

Discussion

Most of the mortality that led to closure of commercial herring fisheries in 1999 probably occurred during and immediately after spawning in 1998, when prevalence of ulcers and virus was high, and when fish behavior was abnormal. Resultant mortality was not documented until 1999 because Pacific herring disperse during the summer, and the most accurate population estimates are made in late March and early April when the fish congregate to spawn. By 1999, fish were relatively healthy and disease-related mortality in 1999 was probably minimal. Note, however, that recovery might still be slow because of higher predation mortality unrelated to disease.

Disease-related mortality in one year may significantly affect the expression of disease in following years. Disease develops through a complex interaction of the host (Pacific herring), the pathogens (VHSV, *Ichthyophonus*, et al.), and the environment (PWS and the northern Gulf of Alaska) (Hedrick 1998). On a population scale, virus probably contributes more to mortality when the population is young, whereas *Ichthyophonus* contributes more to mortality when the population is old. From laboratory studies, we know that fish that recover from infection with VHSV often have slow growth several weeks after other evidence of the virus is gone (Richard Kocan, University of Washington, Seattle, WA; personal communication). We also know that the major zooplankton bloom in PWS usually occurs May or early June, within a few weeks of the herring spawn. If fish are still recovering from an April viral infection and are unable to take full advantage of the short-lived bloom, they will enter in the winter in poor condition. Fish in poor condition are more likely to succumb to viral infection. Also, energy stores for overwinter survival tend to be less in young herring than in older herring. Therefore, young fish in poor condition are more likely to be infected with VHSV and die. Those that do survive are more likely to be in poor condition going into the following winter.

The multiyear pattern of disease expression was evident in the Pacific herring population of PWS beginning in 1997. Relatively strong recruitment of the 1994 year class in 1997 was associated with high viral prevalence in 1997. Ulcer prevalence was relatively low and disease-related mortality was low, but time needed to recover fully from the viral infection left a significant proportion of the population poorly prepared for the winter of 1997-1998. Water temperatures during the winter of 1997-1998 averaged 2°C greater than the winter of 1996-1997, but food availability was relatively poor during the winter of 1997-1998 (Robert Foy, University of Alaska, Fairbanks, Alaska; personal communication). Warm water contributed to the earliest herring spawning season on record in 1998, when the sac roe seiner fishery was completed on April 6, 1998. By the time disease study began on April 15, 1998, fish condition was poor, VHSV prevalence was high among the relatively strong 1995 recruiting year class, and ulcer prevalence was high. Fish behavior was very abnormal: we were able to catch nonspawning herring with a cast net deployed in shallow water near shore. It is highly likely that many of the VHSV-infected fish died in 1998 during and immediately after spawning. Food availability was good during 1998, the winter of 1998-1999 was not unusual, recruitment of the 1996 year class in 1999 was poor (i.e., few susceptible fish in 1999), and viral and ulcer prevalence in 1999 were again low. Good food availability and overwinter conditions for 1999-2000 should result in a relatively healthy population in April 2000.

Conclusions

We are able to understand the timing and causes of this population decline only through the availability of extensive pre-crash data—information that has never been available to explain the decline of a wild fish population. Further study, scheduled for April 2000 (project 00462) will provide more information on the effects of disease on herring population recovery. Because population biomass is very low, and recruitment of the 1996 year class was poor, it will be many years before the population recovers. According to the restoration objectives, a large year class

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Acknowledgments

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Figures

Figure 1. Biomass estimates of adult prespawning Pacific herring in Prince William Sound, Alaska. Unexploited spawning biomass projected in the year before spawning (Projected) and calculated after spawning (Actual) using the age-structure assessment model. Estimates were made by Mark Willette, Alaska Department of Fish and Games, Juneau, Alaska; unpubl. data.

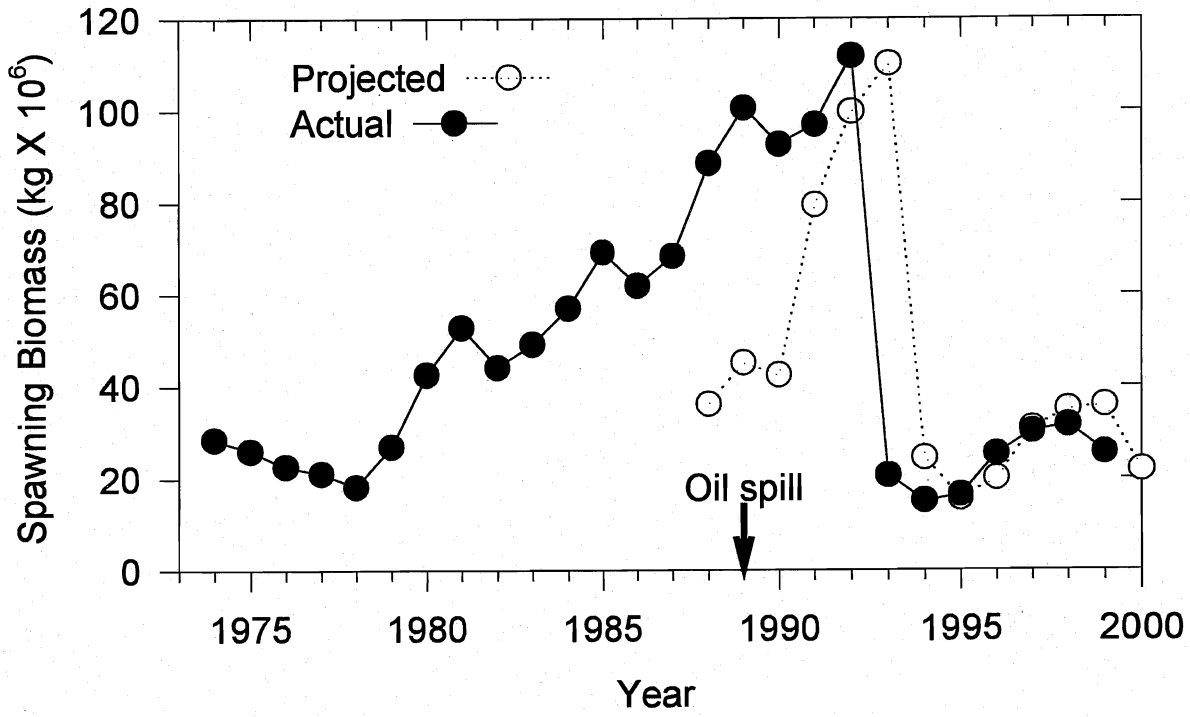

Figure 2. Prevalence of focal skin reddening (FSR) and viral hemorrhagic septicemia virus (VHSV) in adult Pacific herring sampled in the spring from Prince William Sound, Alaska (n = 233-3000 per year).

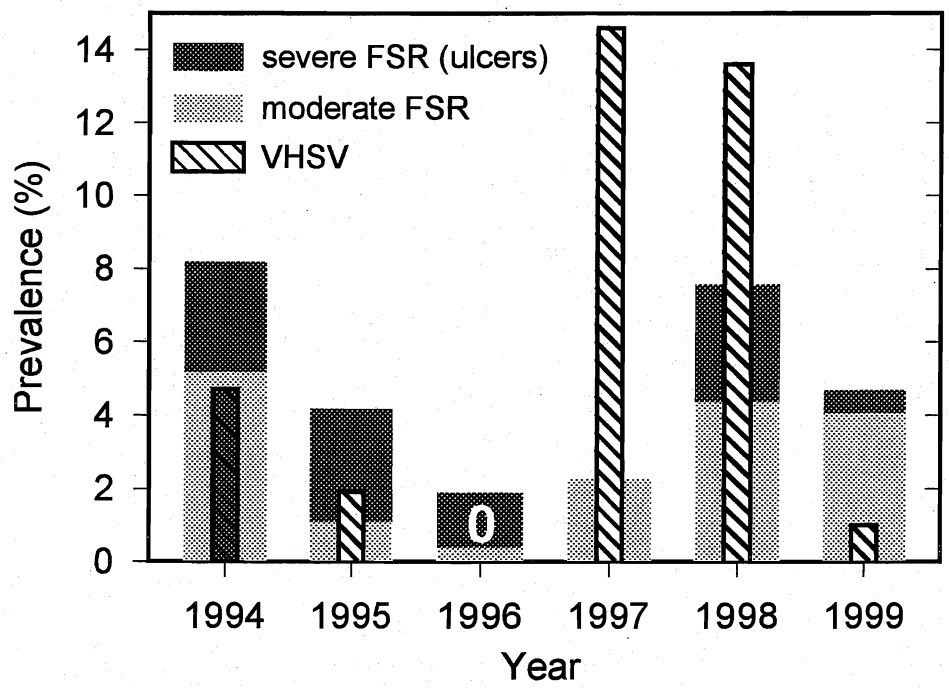

Figure 3. Mean age and prevalence of *Ichthyophonus hoferi* in Pacific herring sampled from Prince William Sound, Alaska (n = 233-300 per year).

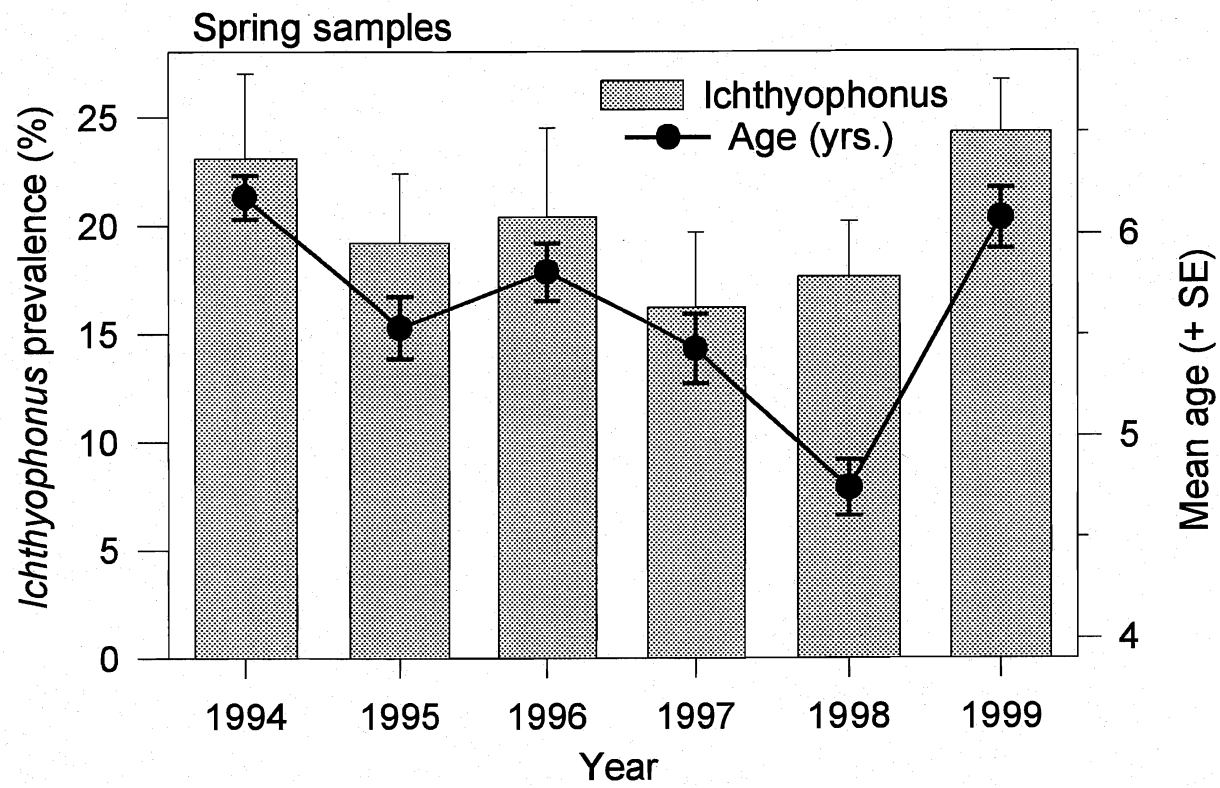

Figure 4. Mean prevalence of *Ichthyophonus hoferi* in the 1988 Pacific herring year class sampled in the spring from Prince William Sound, Alaska.

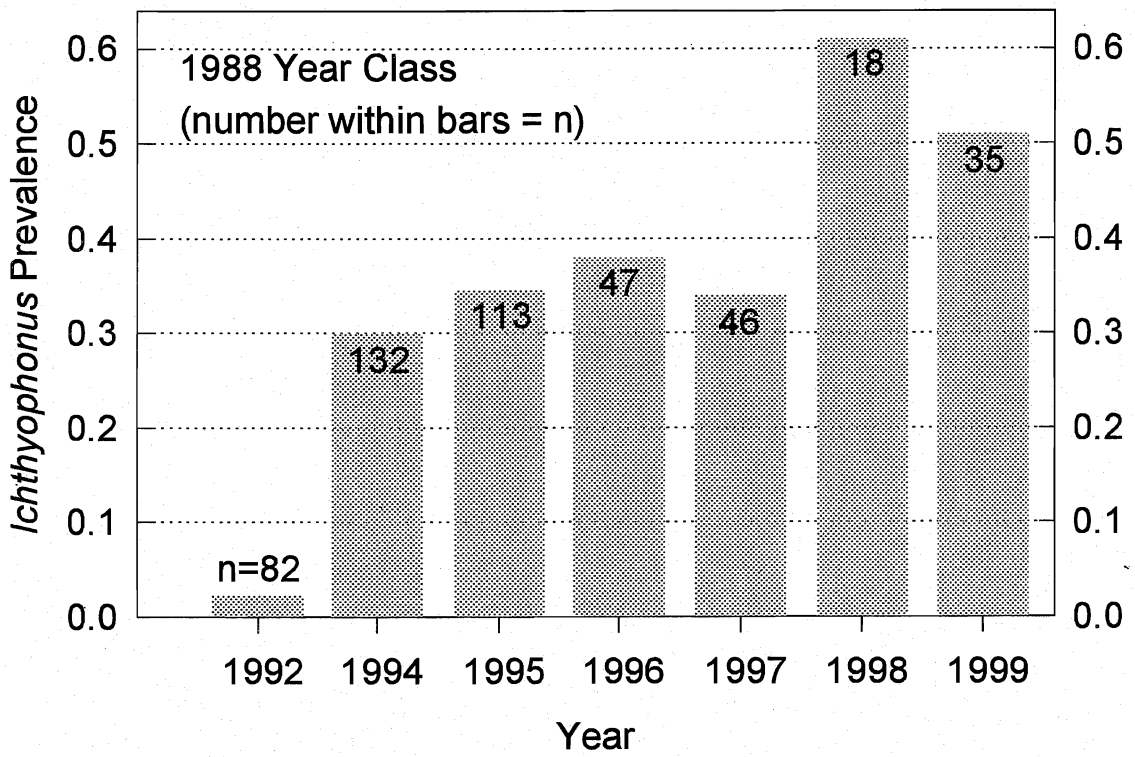

Figure 5. Prevalence of cysts of unknown etiology in the gills of adult Pacific herring from Prince William Sound, Alaska.

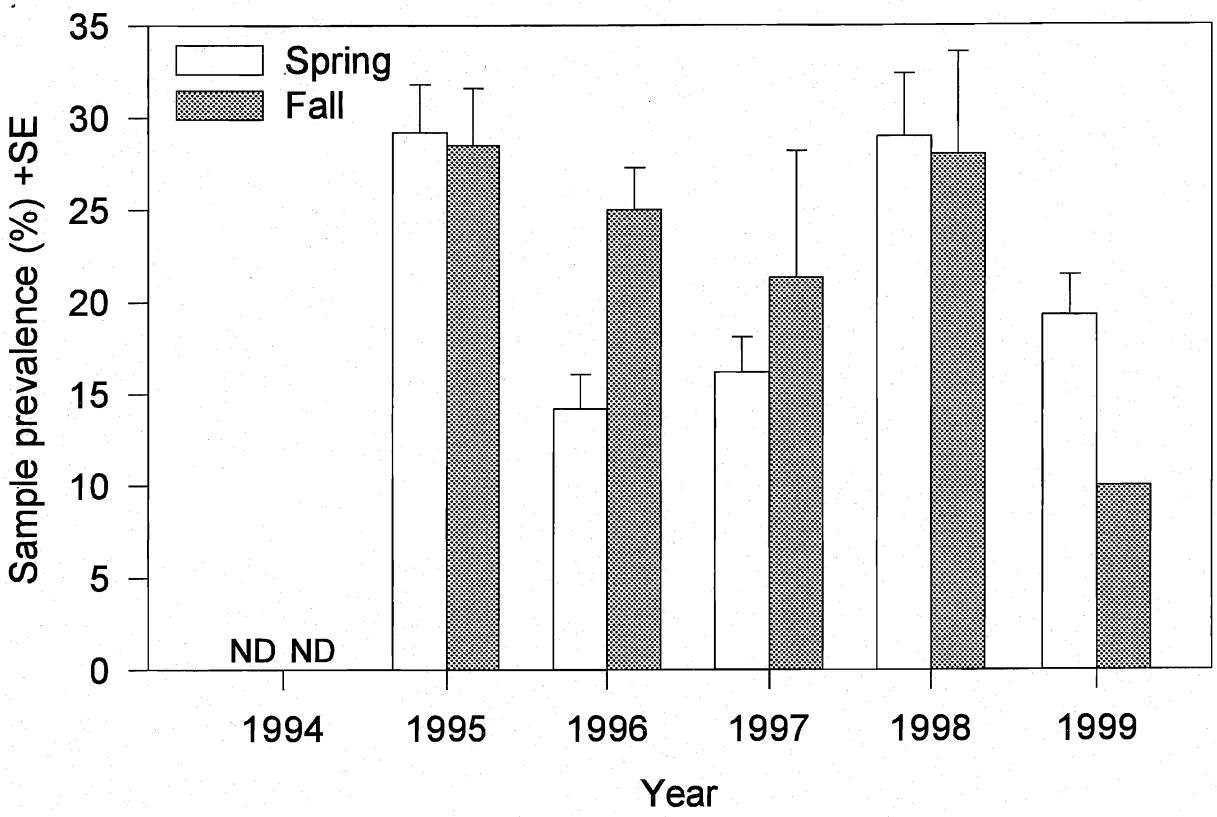

Figure 6. Prevalence of copepod parasites on the medial operculum of adult Pacific herring from Prince William Sound, Alaska.