Exxon Valdez Oil Spill
Restoration Project Annual Report

Effect of Disease on Recovery of Pacific Herring in Prince William Sound, Alaska, Fall 1999 and Spring 2000

Restoration Project 00462
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 the first year of this project (99462), but results were included in the final report for 98162; therefore, results from fall 1998 samples were not repeated in the annual report for 99462, "Effect of Disease on Recovery of Pacific Herring in Prince William Sound, Alaska, Spring 1999." 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 were closed for the 2000 and 2001 seasons. Detailed disease study of Pacific herring sampled in November 1999 (n = 40) and April 2000 (n = 300) used the same methods as used every year since 1994. Disease prevalence in the Pacific herring population slightly decreased from spring 1999 to spring 2000, and disease probably did not significantly inhibit population recovery during this period. None of the Pacific herring sampled in fall 1999 or spring 2000 had evidence of viral hemorrhagic septicemia virus, compared with 1% prevalence in spring 1999. Prevalence of moderate+severe focal skin reddening decreased from 4.7% in 1999 to 1.0% in 2000. Prevalence of the fungus-like organism *Ichthyophonus hoferi* decreased from 24% in 1999 to 22% in 2000.

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). All project data is stored in an Excel spreadsheet (188 columns and 3276 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:

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Executive Summary

Introduction

The estimated spawning biomass of Pacific herring (*Clupea pallasi*) in Prince William Sound, Alaska, decreased from about 22,000 metric tons in 1999 to only 8,000 metric tons in 2000. Due to low population biomass, Pacific herring fisheries in Prince William Sound were closed for the 2000 and 2001 seasons. Studies of the Prince William Sound Pacific herring population since 1994 provided evidence that disease is a significant variable in population fluctuations. Ulcers and viral hemorrhagic septicemia virus are 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 fall of 1999 and spring of 2000.

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 November 1999 (n = 40) and April 2000 (n = 300). Fish were all sampled from near the north end of Montague Island (Stockdale Harbor, Rocky Bay, and Zaikof 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 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, except for total prevalence of *Ichthyophonus hoferi*, which was determined by histopathological examination.

Results

Disease prevalence in the Pacific herring population slightly decreased from spring 1999 to spring 2000. Prevalence of viral hemorrhagic septicemia virus decreased from 1% in 1999 to 0% in 2000. Prevalence of moderate+severe focal skin reddening decreased from 4.7% in 1999 to 1.0% in 2000, although prevalence of ulcers was the same in 1999 and 2000 (0.7%). Prevalence of the fungus-like organism *Ichthyophonus hoferi* decreased from 24% in 1999 to 22% in 2000. None of the fall samples had evidence of viral hemorrhagic septicemia, and this finding is consistent with fall samples in all other years studied. Fall sample size was too small to make valid comparisons of *Ichthyophonus* prevalence.

Discussion

Continuous study of the Pacific herring population of Prince William Sound, Alaska, provides detailed evidence of a disease outbreak that began in 1997, peaked in 1998, declined in 1999, and returned to baseline levels in 2000. Never before have we had disease information in a marine fish population that allowed us to follow a multiyear epizootic from baseline (1996) to peak (1998) and return to baseline (2000).

Conclusion

Further study, scheduled for April 2001 (project 01462) will provide more information on the effects of disease on herring population recovery. Because population biomass is very low, and recruitment of the 1997 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 1998 year class is strong, which based on low numbers in fall 2000 samples seems unlikely, population recovery based on restoration objectives cannot be confirmed until at least the year 2003.

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 100×10^6 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. 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.

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 associated with ulcers, acute disease, and unexpected population decline. By comparison, *Ichthyophonus* prevalence was fairly constant and associated with chronic disease that probably decreased the life span of affected fish, but *Ichthyophonus* did not result in unexpected population decline (Quinn et al. In press).

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.

The estimated spawning biomass of Pacific herring in PWS, Alaska, decreased from about 36,000 metric tons in 1998 to only 8,000 metric tons in 2000. Pacific herring fisheries in PWS were severely curtailed in 1999, and all Pacific herring fisheries were closed for the 2000 and 2001 seasons. This report describes the major disease-related findings in Pacific herring sampled from PWS during the fall of 1999 and spring of 2000. It also builds on data gathered from previous studies to provide evidence that the initial signs of disease-related population decline began in 1997, peaked in 1998, and then returned to baseline levels by 2000.

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 a commercial purse seiner in 2 sets of 20 fish each in November 1999, and by cast net (15 groups of 20 fish each) in April 2000. 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); 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 (one fish/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 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 *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, basic 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 20-fish sets.

Results

The health status of Pacific herring in PWS in spring 2000 returned to baseline levels. As evidence, prevalence of viral hemorrhagic septicemia virus and skin ulcers in spring 2000 were as low as they have ever been since study began in 1994 (Figure 2). Prevalence of *Ichthyophonus* in spring 2000 samples (24%) was similar to previous years, particularly when prevalence values are adjusted for age (Figure 3). Previous study has shown that *Ichthyophonus* is more common in older fish. Because the mean age of the sample population was not different from 1999 (6.1 years) to 2000 (6.2 years), no age adjustment is needed for comparing prevalence of *Ichthyophonus* between 1999 and 2000.

Prevalence of cysts of unknown etiology in the gills was similar in April 2000 and April 1999 (Figure 4). Prevalence of copepod parasites on the medial operculum was also similar in April 1999 and April 2000 (Figure 5).

Discussion

A multiyear disease outbreak first became evident in the Pacific herring population of PWS beginning in spring 1997. After VHSV was not isolated from any fish sampled in 1996, relatively good recruitment of the 1994 year-class in 1997 was associated with high viral prevalence in 1997. Ulcer prevalence in 1997 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 in Prince William Sound during the El Niño 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 (Foy and Norcross 2000). Because warmer water temperatures increased basal metabolism, and food resources were no greater than normal, fish were in poor condition by the spring of 1998. Poor condition in 1998 was associated with high VHSV prevalence among the relatively abundant 1995 recruiting year class, and ulcer prevalence was high among the entire population. 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 young fish in 1999), and viral and ulcer prevalence in 1999 were again low. Food availability was again good during 1999, the winter of 1999-2000 was not unusual, recruitment of the 1997 year class in 2000 was poor (i.e., few susceptible young fish in 2000),

and viral hemorrhagic septicemia virus was not isolated from any fish in 2000. Disease conditions in spring 2000 were consistent with baseline conditions in 1996, completing the disease outbreak in a 4-year cycle.

Combining our knowledge from study of two distinct disease outbreaks in the Pacific herring population of Prince William Sound, we have strong evidence that poor body condition in early spring is the most significant risk factor for an outbreak–regardless of the underlying mechanisms of poor condition. When the first epizootic occurred in the early 1990s, the Pacific herring population was high, summer growth was poor, and fish entered the winter of 92-93 with inadequate energy stores. By the time the second epizootic occurred in the late 1990s, the population was low, summer growth was excellent, and fish entered the winter of 97-98 in good condition. However, increased metabolism without increased food resources during the winter of 97-98 resulted in poor early spring condition and a second epizootic in 1998. Excellent summer growth in 1998 and 1999 with normal food resources during the winters of 98-99 and 99-00 restored the population to baseline health levels by the spring of 2000.

Conclusions

We are able to understand the timing and causes of multiyear disease outbreaks only through the availability of consistent long term data—information that until now has never been available to explain declines of wild fish populations. Further study, scheduled for April 2001 (project 01462) will provide more information on the effects of disease on herring population recovery. Because population biomass is very low, and recruitment of the 1997 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 1998 year class is strong, which seems unlikely based on their low numbers in the fall 2000 samples, population recovery based on restoration objectives cannot be confirmed until at least the year 2003.

Acknowledgments

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Literature Cited

Davis, C. R., G. D. Marty, M. A. Adkison, E. F. Freiberg, and R. P. Hedrick. 1999. Association of plasma IgM with body size, histopathologic changes, and plasma chemistries in adult Pacific herring *Clupea pallasi*. Diseases of Aquatic Organisms 38:125-133.

Foy, R. J., and B. L. Norcross. 2000. Temperature effects on zooplankton assemblages and juvenile herring feeding in Prince William Sound, Alaska. Pages *in* 18th Lowell Wakefield Fisheries Symposium: Proceedings of Herring 2000: Expectations for a New Millennium, February 22-26, 2000. Alaska Sea Grant Rep., no. 18.

Marty, G. D., E. F. Freiberg, T. R. Meyers, J. Wilcock, T. B. Farver, and D. E. Hinton. 1998. Viral hemorrhagic septicemia virus, *Ichthyophonus hoferi*, and other causes of morbidity in Pacific herring *Clupea pallasi* spawning in Prince William Sound, Alaska, USA. Diseases of Aquatic Organisms 32:15-40.

Meyers, T. R., S. Short, K. Lipson, W. N. Batts, J. R. Winton, J. Wilcock, and E. Brown. 1994. Association of viral hemorrhagic septicemia virus with epizootic hemorrhages of the skin in Pacific herring *Clupea harengus pallasi* from Prince William Sound and Kodiak Island, Alaska, USA. Diseases of Aquatic Organisms 19:27-37.

Quinn, T. F., G. D. Marty, J. Wilcock, and M. Willette. In press. Disease and population assessment of Prince William Sound Pacific herring. Pages *in* Proceedings of Herring 2000: Expectations for a New Millennium, Lowell Wakefield Fisheries Symposium, February 22-26, 2000. Alaska Sea Grant Rep., no. 18.

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 (Best estimate) using the age-structure assessment model. Estimates were made by Fritz Funk and Steve Moffitt, Alaska Department of Fish and Games, Juneau, Alaska; unpubl. data. Biomass estimates were not projected before the 2001 spawning season.

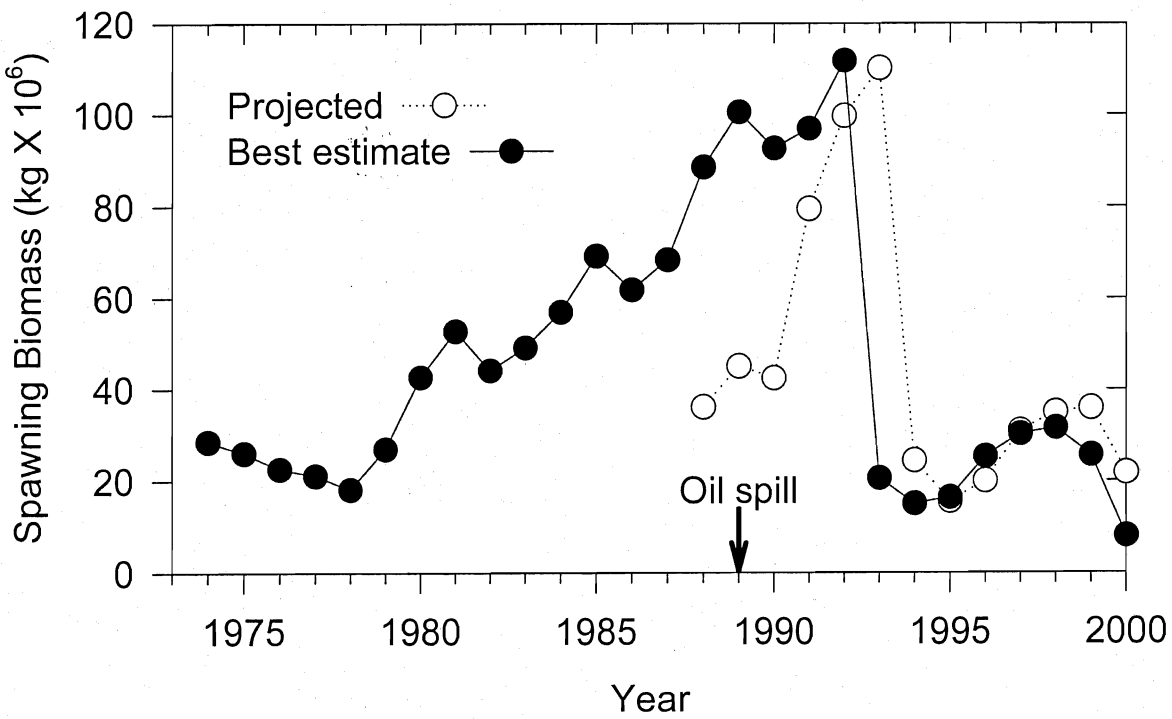

Figure 2. Prevalence of focal skin reddening (FSR) and viral hemorrhagic septicemia virus (VHSV) in adult Pacific herring sampled in the spring from Prince William Sound, Alaska (n = 233-300 per year).

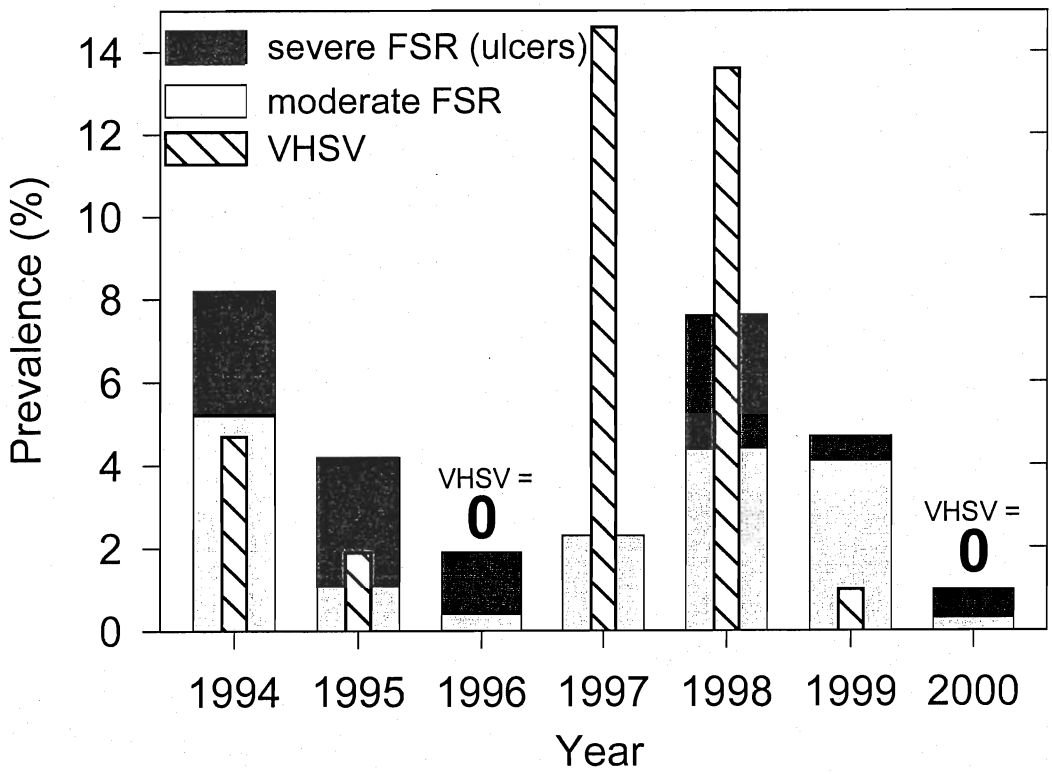

Figure 3. Mean fish age and prevalence of *Ichthyophonus hoferi* in Pacific herring sampled from Prince William Sound, Alaska (n = 233-300 per year).

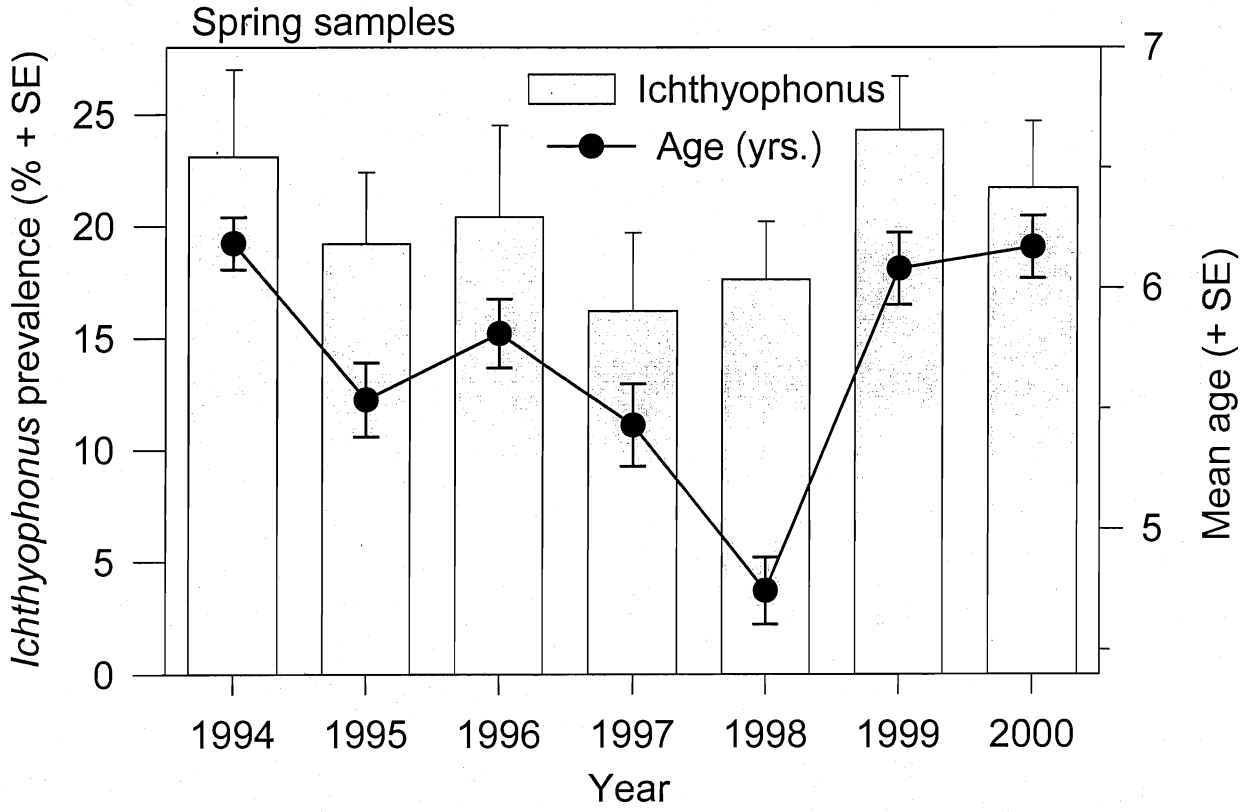

Figure 4. Prevalence of cysts of unknown etiology in the gills of adult Pacific herring from Prince William Sound, Alaska.

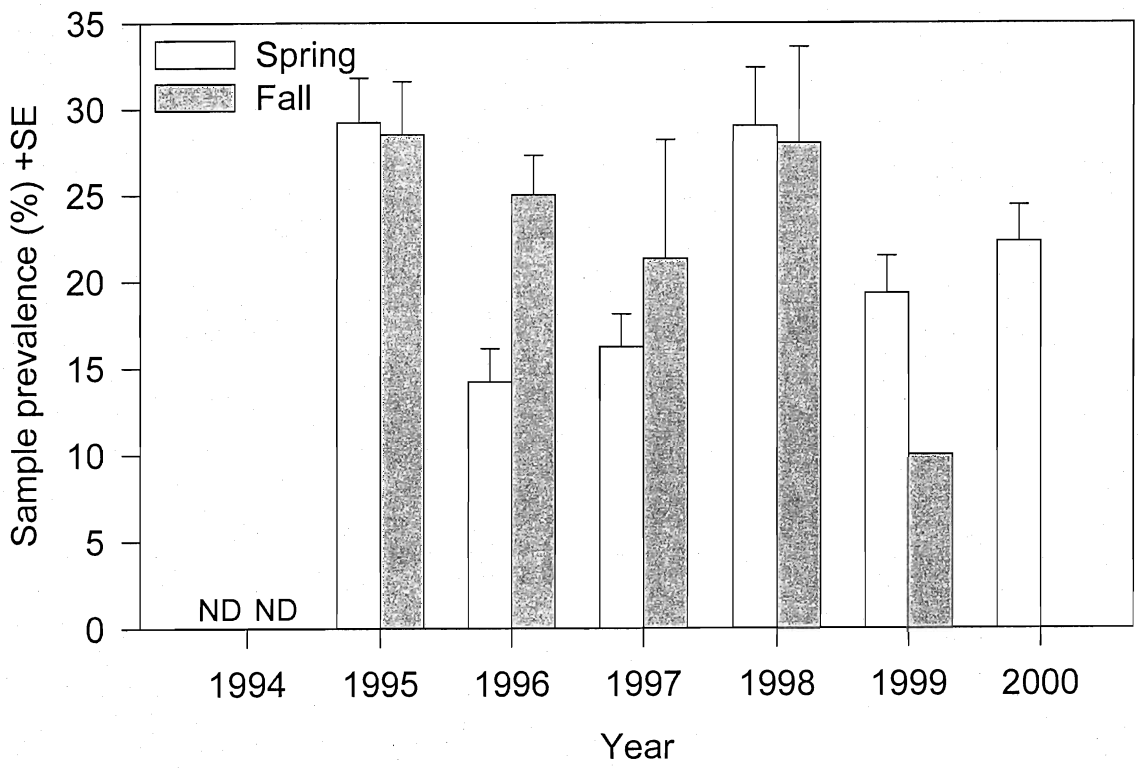

Figure 5. Prevalence of copepod parasites on the medial operculum (gill covering) of adult Pacific herring from Prince William Sound, Alaska.