Conceptual Model for Disease Effects in the Klamath River

J. S. Foott
USFWS, California-Nevada Fish Health Center

J. L. Bartholomew
Department of Microbiology
Oregon State University

R. W. Perry and C. E. Walker
USGS, Columbia River Research Laboratory

INTRODUCTION

This summary report describes a conceptual model for myxozoan disease effects on juvenile Chinook and coho salmon in the Klamath River under the scenarios of current conditions and removal of the four Klamath project dams. For reasons summarized in a previous document "Compilation of Information Relating to Myxozoan Disease Effects to Inform the Klamath Basin Restoration Agreement (Compilation Report; Bartholomew and Foott 2010), we use *Ceratomyxa shasta* infection as an indicator of disease mortality. A review of the parasite life cycle, parasite distribution in the Klamath River, assumptions, definitions and assessments of how specific physical and biological parameters affect disease is presented in that document. That report also prioritized the factors we believe are most important for predicting where disease effects will occur under current conditions and where future areas of high infection might occur.

The conceptual model for disease effects presented in this report builds on the *C. shasta* life cycle model (Figure 1), where the parasite has two life stages requiring fish and polychaete hosts. Because of the spatial and temporal differences in the life stages of salmon, we separate the cycle into two compartments. In one, the parasite infects adult salmon as they migrate into fresh water to spawn, thus releasing myxospore stages upriver to infect polychaetes. In the second, the polychaetes release actinospores in the spring, timed with out migration of juvenile salmon. The contribution of myxospores to the current infectious zone (Shasta River to Seiad Creek) by infected juvenile salmon is unknown but believed to be far less significant than adult carcasses.

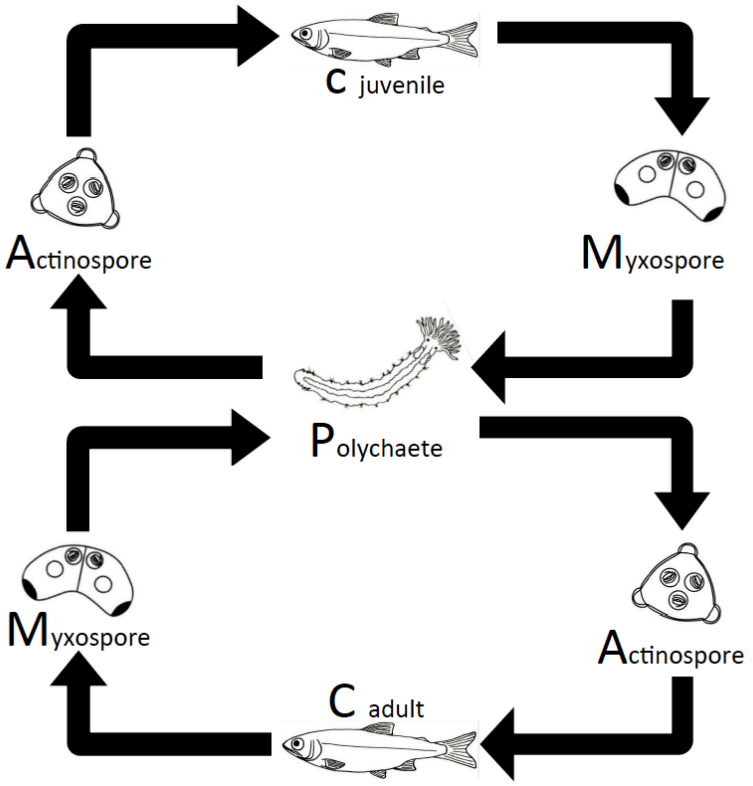

Figure 1. Life cycle model for *Ceratomyxa shasta* showing transmission of parasite life stages to both hosts: polychaete worms and salmon (juvenile and adult).

The conceptual model (Figure 2) is based on the assumption that disease occurs when the following parameters coincide:

- microhabitats with low velocity, stable flows (high density polychaete habitat)
- high numbers of spawning adult salmon (myxospore input)
- temperatures above 15˚C (increases rate of disease in fish)

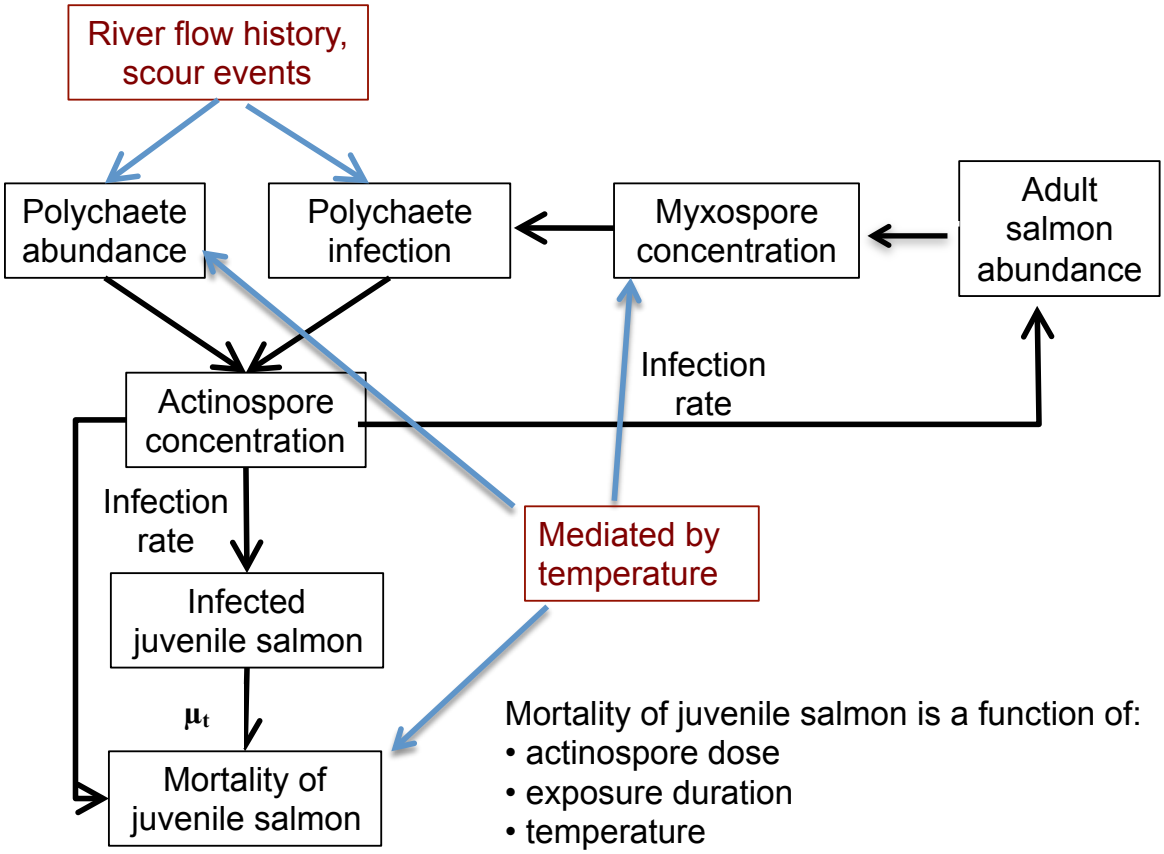

Figure 2. Conceptual model for variables that influence infection and mortality of juvenile fall-run Chinook salmon. μ_t is the mortality rate of infected juvenile salmon, estimated from weekly actinospore concentration.

In this report we present an approach for examining the relationship between water temperature and disease. The remaining variables, flow and adult salmon abundance, will be examined similarly in an expanded model that incorporates data on discharge, measures of flow variability and numbers of adult spawners.

DATA SETS USED FOR PREDICTING DISEASE EFFECTS

The following datasets are being used to interpret disease effects:

Data collected at index sites below Klamath dam from 2004-2010 (OSU studies, funded by the Bureau of Reclamation)

1. Water sampling for actinospore density
    a. From 2004-2010
    b. > 300 samples
2. Sentinel fish mortality and infection prevalence
    a. From 2004-2010
3. Polychaete density (2006-2010)

Data collected in April and May (prior to IGH release) natural origin juvenile salmon collected primarily at kinsman trap (Rm 146) in the Klamath River (USFWS)

1. Outmigrant infection prevalence
    a. From 2004-2010

We estimate disease-related mortality with the following equation:

$$\Pr(\text{Disease Mortality}) = \Pr(\text{Infection}) * \Pr(\text{Mortality} \mid \text{Infection})$$

Pr(Infection) = Prevalence of *C. shasta* infection data from outmigrant studies
Pr(Mortality | Infection) = cumulative *C. shasta* mortality data from sentinel studies

As discussed above, current models with October to March flows and adult carcass numbers do not predict actinospore concentrations and associated Prevalence of infection (POI) estimates.

The following factors mediate the life cycle and will be eventually used to make predictions about disease effects:

1. Water temperature
2. Adult numbers
3. Discharge
4. Mesohabitat types and abundance

RELATIONSHIP BETWEEN ENVIRONMENTAL CONDITIONS AND DISEASE

Disease effects reflect both actinospore release and parasite pathogenesis mechanisms. We examine these effects individually using data from water sample analysis (actinospore concentration), outmigrant infection prevalence, and sentinel fish studies (mortality of infected fish). Generalized linear models (GLM) and linear models were used to examine relationships among environmental conditions and

actinospore concentration, prevalence of infection, and mortality of juvenile Chinook salmon using the software R (R Development Core Team, 2007). More specifically, log-transformed river spore concentration collected at Beaver Cr. was modeled as a quadratic function of water temperature. The model includes year as a categorical variable, maximum discharge the previous year, and spawner abundance upstream of Beaver Creek in the previous year. Mortality of sentinel fish exposed in the infectious zone was analyzed using an exponential model as a function of water temperature and actinospore concentration. To model prevalence of infection through time, we used a three-state irreversible disease model (Heisey et al., 2006) with actinospore concentration as the covariate. Models with different covariates were compared using Akaike's Information Criterion (AIC, Burnham and Anderson, 1998), and the best-fitting model (i.e., the lowest AIC) is shown here.

Factors affecting actinospore concentration

As described in the Compilation Report, section 6.2.3, temperature affects both actinospore release and pathogenesis within the infected fish. Water sample data indicates that actinospore concentration increases as temperatures rise above 10˚C in spring and drops as water temperature reaches 23°C in late July (Section 2.3).

Water temperature had a strong quadratic effect on log-actinospore concentration. Actinospore concentrations rose until temperature reached about 17 C and then decreased at higher temperatures (Figure 3). Spawner abundance and maximum discharge explained little variation in actinospore concentration, but we found significant variability in actinospore concentration among years (Figure 3). For example, 2010 exhibited low actinospore concentrations relative to the temperatures observed in 2010, whereas 2007 had the highest actinospore concentrations. Although temperature explains seasonal variation in actinospore concentrations, quantifying the mechanisms causing the significant year-to-year variation remains a formidable challenge.

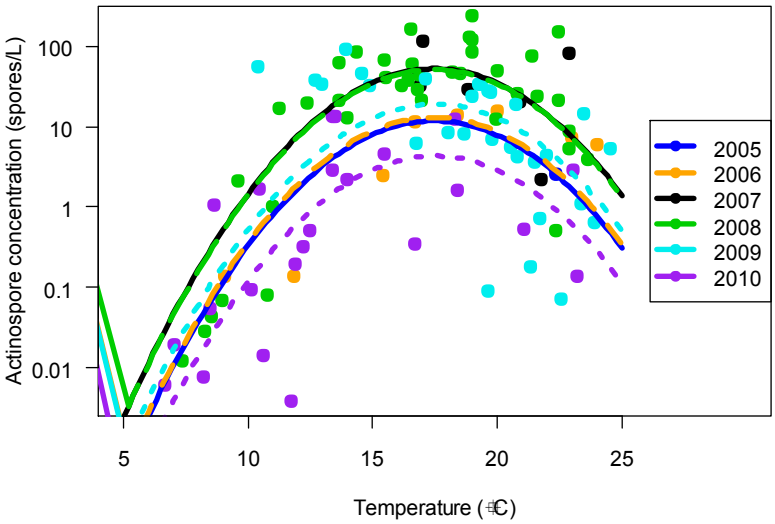

Figure 3 River actinospore concentration in the lower Klamath River at different temperatures, 2005-2010.

Relationship between actinospore concentration and infection prevalence

Over 2,500 juvenile fall-run Chinook were sampled in the lower Klamath River where the prevalence of infection by *Ceratomyxa shasta* was determined. Greater than 1,400 of those were sampled in the infectious zone which was the focus area for modeling infection rates as a function of environmental conditions, especially covariates for spore concentration and water temperature.

Observed weekly infection rates increased during the spring season and peaked during late May into June. Average weekly infection rates were about 3.2 percent in the upper most reach from the confluence of the Shasta River up to Iron Gate Dam, zero percent in the reach below the infectious zone, and 23.7 percent in the infectious zone. Sample sizes were small for reaches other than the infectious zone (N<10).

Using a three-state irreversible disease model that expresses the effects of covariates through time, actinospore concentration, temperature, and fish size were significant in explaining variation of infection rates. However, actinospore concentration explained most of the variation of infection rates (Figure 4). Infection rates increase substantially above 10 actinospores/L.

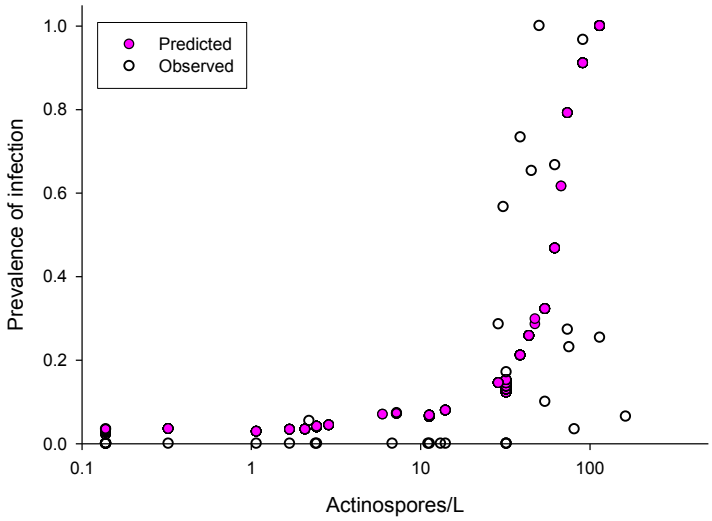

Figure 4. Relationship between infection prevalence in juvenile fall-run Chinook salmon and actinospores/L in the infectious zone.

Effect of temperature and actinospore concentration on mortality

Sentinel mortality experiments were performed from 2002 to 2010, with the exception of 2003. This included exposures of over 3,900 fish for assessing mortality. Mortality was highest during June, consistent with high infection rates and actinospore concentrations. Observed mortality in sentinel trials at Klamathon

Bridge averaged 0.3 percent, 36 percent in the infectious zone at Beaver Creek, 28 percent at Seiad, and 2.5 percent at Orleans. Trials in reaches outside of the infectious zone were limited (12 trials/reach), whereas, many more were performed within the infectious zone at Beaver Creek (42 trials/reach).

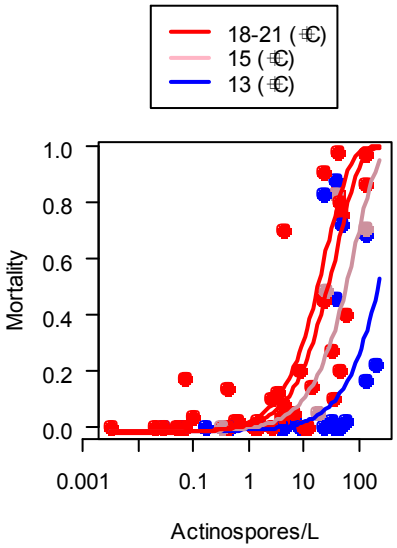

Figure 5. Relationship between actinospore concentration, holding temperature and mortality from *Ceratomyxa shasta* at Beaver Creek.

Actinospore concentration, holding temperature, and their interaction were significant in describing variation of mortality in sentinel trials. Mortality increased sharply at a temperature-dependent threshold in actinospore concentration. As temperatures increase, the threshold concentration decreases below 10 actinospores/L.

RELATIONSHIP BETWEEN WATER FLOW AND DISEASE

As described in the Compilation Report section 5.2, flow is likely to have a strong effect on the stability of polychaete populations and thus on disease.

Flow levels considered to have an effect are those that will result in bed mobilization that would release fine material trapped in gravel and rework channel features. As discussed above, using the model run to assess the relationship with temperature and disease, mean annual and maximum annual discharge were not significantly related to actinospore concentration. The duration of high flows necessary to disrupt high-density polychaete habitat in the infectious zone and the influence of winter flows on transmission of myxospores to polychaetes is unknown. Other factors that are likely to have an influence include microhabitat water velocity and frequency of threshold flows; however, we currently lack data to incorporate these variables in the model and cannot estimate the microenvironments throughout the river. Additionally, the infectious zone may have unique bed movement properties that require higher flows

to mobilize D50 particles than reaches below it (B. Greimann, January 10, 2011 Chinook Tech. Workgroup presentation).

Other aspects of water flow that will inform the model include:

- Water velocity effects on transmission of the actinospore to the salmonid host.
    - At higher velocities the ability of the parasite to successfully attach to the salmonid host's gill may decrease.
- Variability between the minimum and maximum flows in different reaches.
    - Dynamic or variable flows may decrease the ability of the polychaete to establish large high-density colonies, whereas in a reach with low flow variability, polychaetes may inhabit a greater portion of the river channel.

These parameters are currently being explored experimentally and models will be developed to incorporate the effects of variability as a potential covariate of salmon disease and mortality.

RELATIONSHIP BETWEEN ADULT SPAWNERS AND DISEASE

As described in the Compilation Report 7.2.2, we make the following assumptions for the carcass numbers and their disease effects listed below:

- Annual infectivity cycle for polychaete populations in the Klamath River depends on adult Chinook carcasses releasing myxospores (October to May) into the water. Juvenile salmon myxospore input is considered insignificant to the infectious zone, although release of myxospores in the lower river may serve as a focus of infection for adult salmon.

- Carcasses in the mainstem Klamath R. and within 20 rkm upriver of polychaetes have the maximum influence on myxospore transmission. Data on myxospore input from tributary carcasses is insufficient, but it is likely their input is less than main stem carcasses unless within 20 rkm of the mainstem. Approximately 45% of the carcasses that contribute to the current infectious zone occur in the Iron Gate dam to Shasta R. reach, ~ 20 rkm (USFWS carcass survey data). Estimated carcass numbers within this reach between 2004 to 2009 ranged from 3587 to 5523 fish (Arcata FWO annual spawner survey report "http://www.fws.gov/arcata/fisheries/projectUpdates/KRSpawningSurvey/KlamathSpawnSummary2009.pdf"

Data is insufficient to support establishing a threshold number of carcasses that could maintain *C. shasta*. Comparison of annual variability of actinospore concentration with spawners during the previous year above Beaver Creek did not show a strong relationship because of the multiple pathways involved, as discussed in the next section.

CURRENT STATUS AND REMAINING UNCERTAINTIES

Our analysis showed that both prevalence of infection and mortality can be linked closely to actinospore concentration and temperature. This allows juvenile mortality to be predicted if actinospore concentration and water temperature are known. However, given the data currently available, we were unable to empirically link either water flow or spawner abundance to actinospore concentration. As shown in Figure 2, there are many links and feedbacks where actinospore concentration may be influenced. For example:

- Myxospore concentrations depend on spawner abundance, spawner infection prevalence, and rate of myxospore maturation, all of which are affected by spawner migration timing and water temperature.
    - These complex relationships, in turn, affect predictions about the relationship between numbers of adult spawners and actinospore levels the following year.
- Actinospore concentrations are affected by polychaete abundance and infection prevalence, both of which may be affected by river flow.
    - For example, high winter flows the previous year may affect transmission of myxospores to polychaetes, reducing infection prevalence.

These linkages may act to obscure a direct correlation between, for example, spawner abundance and actinospore concentration the next year. For example, spawner abundance may be high, but actinospore concentration low if there is a low infection rate of spawners.

Future challenges are to quantify the mechanisms driving the interannual variation in actinospore concentrations and to determine the factors that influence actinospore abundance outside the current infectious zone. The ability to make these predictions is limited, in part, by the following:

- Consistent data on disease incidence and severity is only available for the period 2005 to present.
- System-wide habitat data is on a larger scale (meso-macro habitat) than what is needed to link polychaete density data to habitat abundance
    - High gradient, boulder topography of the current infectious zone is likely creating microhabitats associated with high density polychaete populations
- Limited data available on infection prevalence in polychaete populations
- Limited data available on transmission rates of myxospores from adult salmon to polychaetes
    - E.g how winter flows in the Klamath River and carcass bearing tributary streams influence transmission

QUALITATIVE DISEASE MORTALITY PREDICTIONS UNDER DAMS OUT

We assume that water temperature and actinospore concentration are primary drivers of juvenile mortality. The effects of temperature on disease in the infectious zone under different temperatures can be qualitatively described as follows:

Disease effect	Temperature range
none	< 10°C or >23˚C
low	10 - 12°C
moderate	13 - 15°C
high	>15°C ≤ 23°C

Qualitative assessments of mortality for the current infectious zone and other river reaches under dams out are considered below and are defined as:

High	Similar to current infectious zone, 40 - 60% disease mortality
Moderate	10- 39% of the infectious zone disease mortality
Low	Similar to below the infectious zone, <10% disease mortality

Williamson River – low to moderate

Currently, both high polychaete abundance and *Ceratomyxa shasta* infection are found in portions of the lower Williamson R. The movement of genotype I with Chinook salmon is likely to establish this Chinook-virulent genotype in this localized reach. Flow patterns are not likely to change in the future. Adult salmon number (myxospore input) and water temperatures during juvenile Chinook rearing periods are judged to be lower than the current infectious zone but sufficient for the moderate risk designation. The rating reflects the possibility of low flow years with warmer temperatures (≥ 15ºC) corresponding with juvenile salmon rearing.

Sprague River – Low

There is little data on polychaete distribution or potential habitat in the Sprague however current spawning habitat is limited and would likely influence adult carcass numbers.

Keno Dam to Iron Gate dam – low to moderate

The river directly below Keno dam is not likely to be a source of adult carcasses. Spawning is unlikely as fish will avoid the impaired water quality in this area during the early fall. Future spawning distribution in the remainder of the designated reach is unknown; however, concentrations of > 3500 adults within 20 rkm areas are considered unlikely. This reach has a higher gradient than the current infectious zone that will influence scour velocities. The fine organic material habitat that supports high polychaete abundance in the current infectious zone is less likely to be maintained in this reach. One exception may be the Copco to Cottonwood creek reach due to its lower gradient, increased sediment input, and historic meander pattern. These attributes could produce high abundance polychaete habitat. The higher frequency of disruptive flows projected for post-dam conditions could limit the stability of polychaete populations in the Copco-Cottonwood creek reach. The rating reflects the possibility of low flow years

with minor polychaete habitat disruption and warmer temperatures (≥ 15ºC).

Infectious zone (Iron Gate to Indian Creek) – low to moderate
Frequency of disruptive flows projected for post-dam conditions should decrease the stability of polychaete populations in the current infectious zone and adult carcass number may decline with removal of migration barriers. The lower threshold for myxospore infection of polychaetes necessary for disease and the associated spawner carcass number is not known. As other elements of the infectious zone will remain, ceratomyxosis is expected to continue under low water year scenarios.

Below Indian Creek – low
Current low parasite densities in this reach are likely a result of reduced polychaete populations in the main stem river and dilution effects of tributaries. Reduction in actinospore production above this reach after dam removal should lower the current low disease risk.

SUMMARY

Data sets (juvenile salmon infection prevalence, *C. shasta* actinospore concentration and infectivity) from the *C. shasta* infectious zone of the lower Klamath River have informed models that describe the effect of temperature and actinospore concentration on fish disease within the infectious zone. In addition, we quantified seasonal and interannual variability in actinospore concentration in the infectious zone, but were unable to link interannual variability to factors thought important to disease (e.g., polychaete and spawner abundance). For SALMOD, these findings allow us to incorporate mortality due to disease as a function of temperature and weekly actinospore concentration when the mean annual actinospore concentration is known. Given the mean annual actinospore concentration, weekly actinospore concentration, prevalence of infection, and mortality can be estimated within the infectious zone. This represents an important advancement over a simple temperature-disease relationships or step functions because it explicitly incorporates the dose-response relationship causing mortality. Furthermore, our analysis allows us to replicate interannual variability in mean actinospore concentrations, which will provide insights into year-to-year variability in disease mortality. These dose-response relationships will be incorporated into SALMOD, and mean annual actinospore concentration can be set by the user or drawn from a distribution of values.

Clearly, the ideal model would link mean annual actinospore concentration to physical (e.g., river flows) or biological (e.g., polychaete abundance) variables so that year-to-year variation in disease mortality evolves dynamically from mechanistic relationships. This remains our central challenge due to the many links in the disease cycle affecting mean annual actinospore concentration. Incomplete understanding of the interaction of environmental factors, such as flow, on the multiple elements of the parasite life cycle requires further research that will continue to inform models of disease dynamics in the Klamath River.

REFERENCES

Bartholomew, J. L. and J. S. Foott. 2010. Compilation of Information Relating to Myxozoan Disease Effects to Inform the Klamath Basin Restoration Agreement. http://klamathrestoration.gov/sites/klamathrestoration.gov/files/Disease%20synthesis_11-1_final.bartholomew.foott.pdf

Burnham, K.P. and D.R. Anderson. 1998. Model selection: a practical information theoretic approach. Springer, New York, NY

Heisey, D. M., Joly, D. O. and F. Messier. 2006. The fitting of general force-of-infection models to wildlife disease prevalence data. Ecology 87:2356–2365.

R Development Core Team, 2010, R: a language and environment for statistical computing: R Foundation for Statistical Computing, Vienna, Austria, ISBN 3-900051-07-0, accessed May 5, 2011 at http://www.r-project.org/.