

## MODELING INFECTION AND MORTALITY OF JUVENILE CHINOOK SALMON DUE TO DISEASE CAUSED BY CERATONOVA SHASTA IN THE KLAMATH RIVER

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Disease can often shape population dynamics but complex host-parasite interactions can be difficult to incorporate into life cycle models. Juvenile Chinook salmon (*Oncorhynchus tshawytscha*) in the Klamath River become infected with the myxozoan parasite *Ceratonova shasta* when the polychaete worm *Manayunkia speciosa* releases actinospores into the water column. In the Klamath River, disease prevalence and actinospore concentrations have been routinely monitored since 2005, providing information about population-level disease prevalence. Concurrently, sentinel experiments with fish held in-river for a known duration have revealed that mortality increases with spore concentration and temperature. We developed statistical models to relate rates of infection and mortality to spore concentrations and temperature. We then incorporated these models into a dynamic life-cycle simulation model to understand how migration and exposure of juvenile Chinook salmon influences the magnitude and location of their mortality in the Klamath River. This model provides an estimate of disease-related mortality at the population-level, which can then be incorporated as a life-stage transition probability in a broader life-cycle model.

### 1 MODELING FRAMEWORK OF THE STREAM SALMONID SIMULATOR

The Stream Salmonid Simulator (S3) is a spatially explicit model that is currently being developed to track abundance of juvenile Chinook salmon (*Oncorhynchus tshawytscha*) as they migrate from natal areas to the ocean in the Klamath River. The heart of the S3 model is the dynamic simulation of movement and survival of juvenile salmon during freshwater rearing in a river environment. The S3 model tracks the number of individuals by habitat unit, date, life stage, and source population. Formally, we define this quantity as  $n_{p,s}(h,t)$  where:

$n$  = number of individuals

$p$  = source population,  $p = 1, \dots, P$

$s$  = life stage,  $s = \text{fry, parr, or smolt, for example}$

$h$  = habitat unit,  $h = 1, \dots, H$

$t$  = day (or date),  $t = 1, \dots, T$ .

As this notation implies, S3 is a discrete-space, discrete-time model that runs on a daily time step. Spatially, the continuous longitudinal axis of the river is divided into a series of discrete spatial units of length  $\Delta x_h = x_{h,\text{up}} -$

$x_{h,down}$ , where  $x$  is the distance from the downstream terminus of the model (the river's mouth) and  $x_{h,up}$  and  $x_{h,down}$  mark the upstream and downstream boundaries of habitat unit  $h$ . Source populations represent individuals entering the model's domain from different tributaries or from emerging fry that are progeny of spawning adults within the mainstem river.

The dynamics of the model operate much like a stage-structured matrix population model that is governed by a series of transition equations. Transition equations consist of daily survival, movement probabilities, and a growth function that determines when fish grow large enough to transition from one life stage to the next. In terms of order of operations, fish in habitat  $h$  on day  $t$  survive, grow, and then move. In this paper, we focus on modeling infection and mortality owing to *C. shasta* in the Klamath River. We model mortality due to disease by first simulating infection of juvenile fish with *C. shasta*, and then modeling mortality of infected fish.

## 2 MODELING INFECTION RATES

Infection rates were modeled using a standard "force of infection" model (Heisey et al. [1]):

$$\Pr(\text{Infection}) = 1 - \exp(-\lambda t) \quad (1)$$

where  $\Pr(\text{Infection})$  is the probability of infection,  $\lambda$  is the rate of infection, and  $t$  is exposure time. Infection rate ( $\lambda$ ) is thought to vary as a function of temperature ( $T$ ) and the concentration of spores in the water column ( $S$ ), as is evidenced by their effect on disease-caused mortality (Hallett et al. [2], Ray et al. [3]). Therefore, we developed a model that allowed  $\lambda$  to vary as a function of these variables:

$$\lambda = \exp(\beta_0 + \beta_1 S + \beta_2 T) \quad (2)$$

where  $\beta_0$ ,  $\beta_1$ , and  $\beta_2$  are the intercept and slope coefficients for spore concentration and temperature, respectively. Coefficients for the model were estimated using the methods of Heisey et al. [1] from observations of 252 coded-wire tagged fish captured at a trap between 2007 and 2012 and then assayed for disease presence (1 = infected, 0 = not infected). Information from coded tag fish included their exposure time from release to recapture, as well as their daily exposure history to observed temperatures and spore concentrations. The resulting model showed that infection rates increased with both spore concentration and temperature (Figure 1).

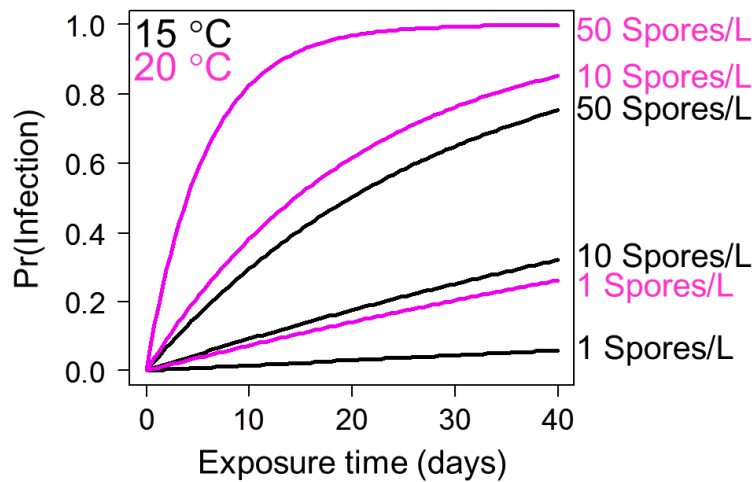


Figure 1. Probability of infection of juvenile Chinook salmon with *C. shasta* as a function of exposure time for different combinations water temperature and spore concentrations of *C. shasta*.

## 3 MODELING SURVIVAL OF INFECTED FISH

We used the survival model of Ray et al. [4] to simulate disease-related mortality of juvenile Chinook salmon:

$$S(t) = (1 - \pi) + \pi S(t | \text{death}) \quad (3)$$

where  $S(t)$  is the probability of surviving  $t$  days after infection,  $1 - \pi$  is the probability of recovering and surviving from infection, and  $S(t | \text{death})$  is the probability of surviving to time  $t$  conditional of fish that died due to disease. Ray et al. [4] used a Weibull distribution for  $S(t | \text{death})$  and fit covariates to both  $\pi$  and the rate parameter of

$S(t|\text{death})$  to a multiyear data set of sentinel exposure experiments. Fish were held at known spore concentrations and mortality rate was modeled over time. Their analysis found that both  $\pi$  and  $S(t|\text{death})$  were affected by spore concentration and water temperature (Figure 2).

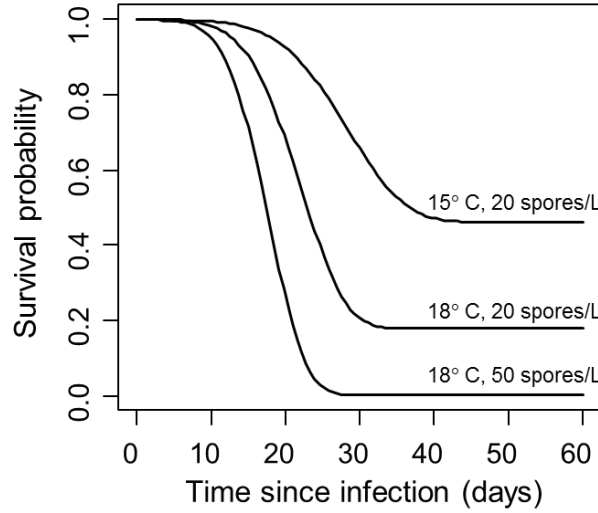


Figure 2. Survival probability of juvenile Chinook salmon as a function of time since infection for different combinations of water temperature and spore concentrations of *C. shasta* based on the model of Ray et al. [4].

#### 4 SIMULATING DAILY INFECTION AND MORTALITY IN THE KLAMATH RIVER

To simulate daily infection and mortality in the S3 population model, fish are transitioned each day from non-infected to infected groups:

$$I_{p,s}(h,t+1) = I_{p,s}(h,t) + \Pr(\text{Infection},t)(n_{p,s}(h,t) - I_{p,s}(h,t)) \quad (4)$$

where  $I_{p,s}(h,t)$  is the number of infected fish in source population  $p$  and life stage  $s$  in habitat unit  $h$  at time  $t$ , and the second term in the sum is the number of non-infected fish that become infected at time  $t$ . Once fish become infected, we keep track of the average time since infection, and then the probability of infected fish surviving disease from time  $t$  to  $t+1$  is calculated as  $S(t+1)/S(t)$ .

We simulated infection and mortality of juvenile Chinook salmon in 2008, a particularly “bad” disease year where spore concentration exceeded 10 spores/L for much of the juvenile salmon rearing period (Figure 3). We assessed simulated infection and mortality of fish migrating past a trap site and assessed mortality over the entire river. Overall, our simulation showed that only 12.8% of the wild juvenile salmon population passing the trap site became infected because much of the population migrated downstream prior to the increase in spore concentrations (Figure 3). In contrast, our simulation showed that over 54% of the hatchery-origin juvenile salmon became infected by the time they passed the trap site because they were released in late May and June when spore concentrations were high. We found that simulated mortality due to *C. shasta* ranged from 9% to 35% depending on source population, but mortality of hatchery origin fish was 73%. Empirically estimating these mortality rates from field studies would be very difficult. Our simulation sheds light on how disease dynamics interact with population-specific migration timing to differentially affect infection and mortality rates of juvenile Chinook salmon in the Klamath River.

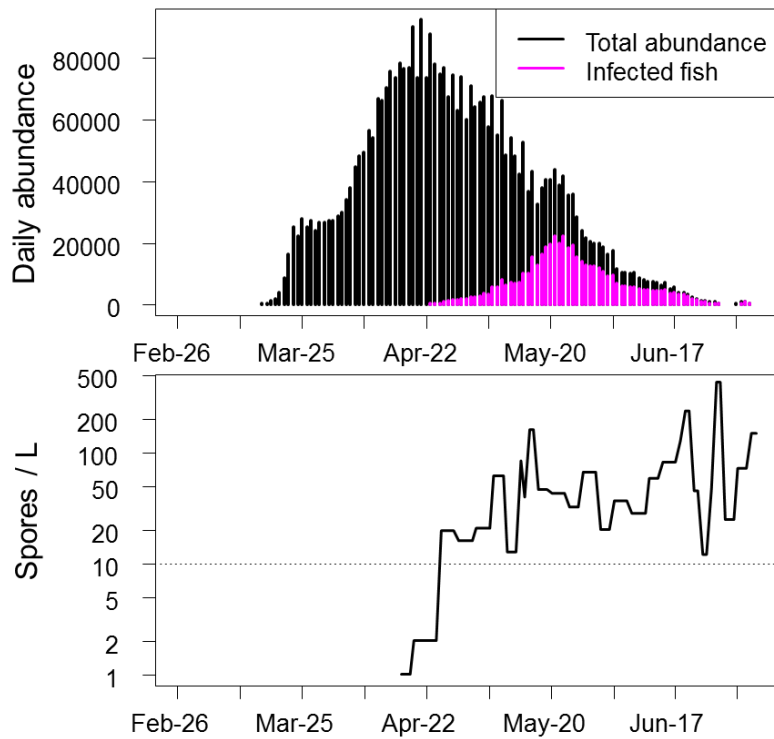


Figure 3. Simulated daily abundance of naturally produced juvenile Chinook salmon passing a trap site on the Klamath River (top) and time series of measured spore concentrations of *C. shasta* in the Klamath River in 2008. The dashed line in the bottom panel at 10 spores/L marks a threshold above which infection rates begin to increase sharply.

## REFERENCES

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